

SIGNS AND SYMPTOMS OF CARDIOVASCULAR SYSTEM DISEASES (syndromes of acquired heart defects)

LECTURE IN INTERNAL MEDICINE PROPAEDEUTICS

M. Yabluchansky, L. Bogun, L.Martymianova, O. Bychkova, N. Lysenko
V.N. Karazin National University Medical School' Internal Medicine Dept.

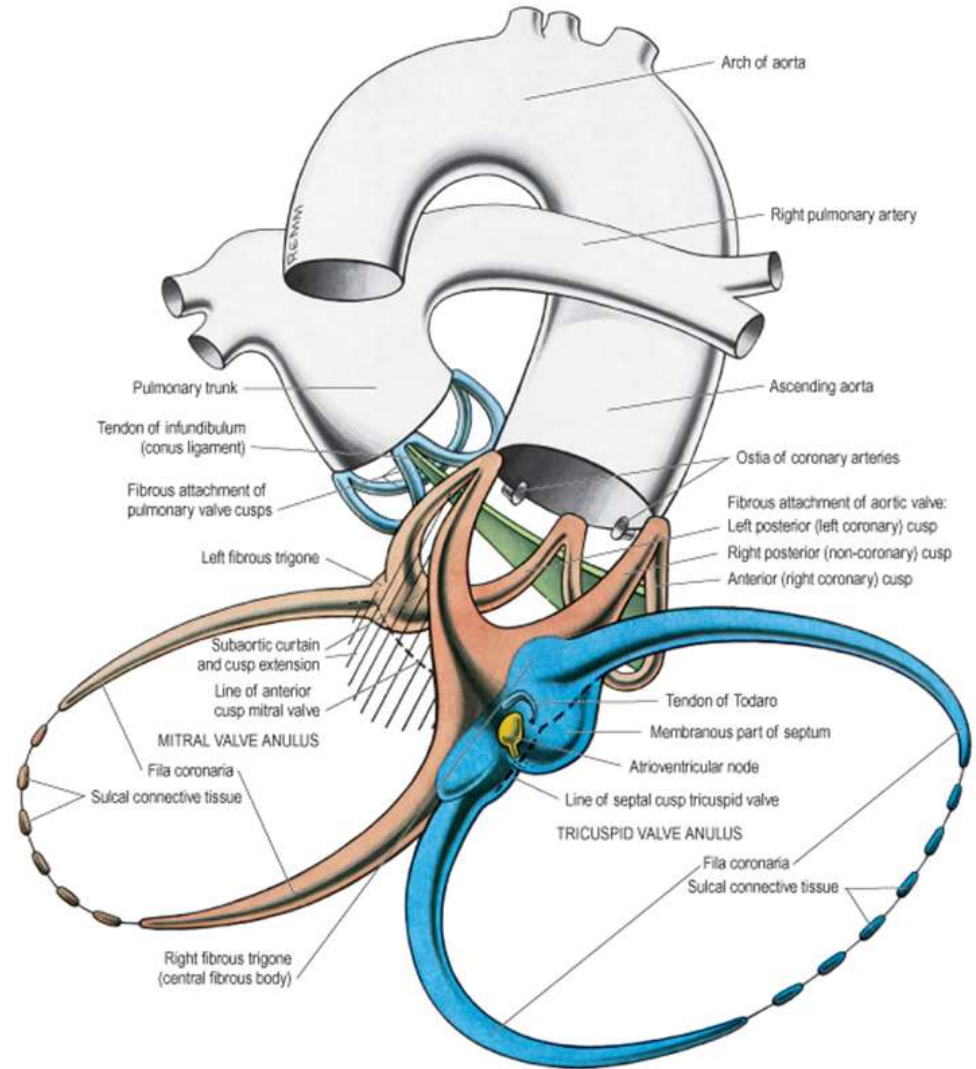
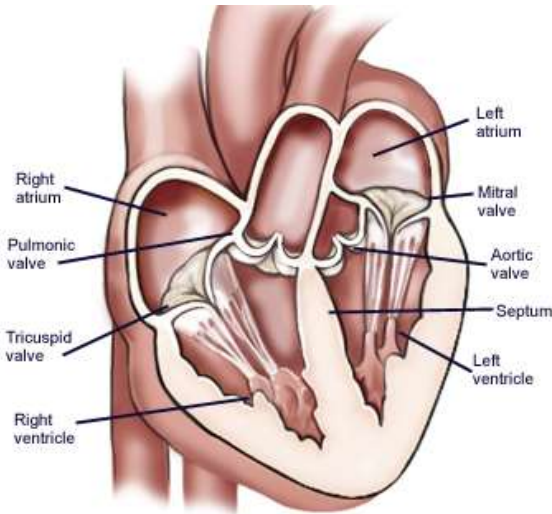
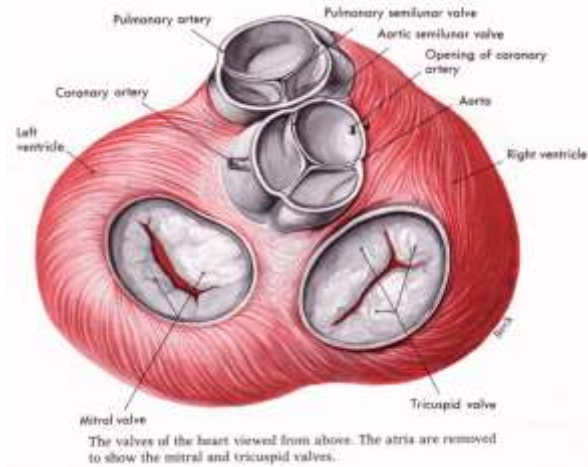
Plan of the lecture



Acquired valvular heart defects

- Common data
- Aortic valve stenosis
- Aortic valve insufficiency/regurgitation
- Mitral valve stenosis
- Mitral valve insufficiency/regurgitation

Heart valves



Definition

- The heart valves are part of the dense connective tissue makeup of the heart known as the heart skeleton
- Valves that are formed properly at birth can still develop problems in form of acquired valvular heart defects that may involve one or more of the four valves of the heart (the aortic and mitral valves on the left and the pulmonary and tricuspid valves on the right)

Classification

- The two major types defects of everyone heart valve are:
 - Insufficiency/regurgitation (the valve's tissue flaps (leaflets), which control the flow and direction of the blood, do not fully close or the edges do not fully meet, which causes blood to leak back into the heart)
 - Stenosis (the leaflets cannot open fully to allow enough blood to flow through)

Causes and risk factors

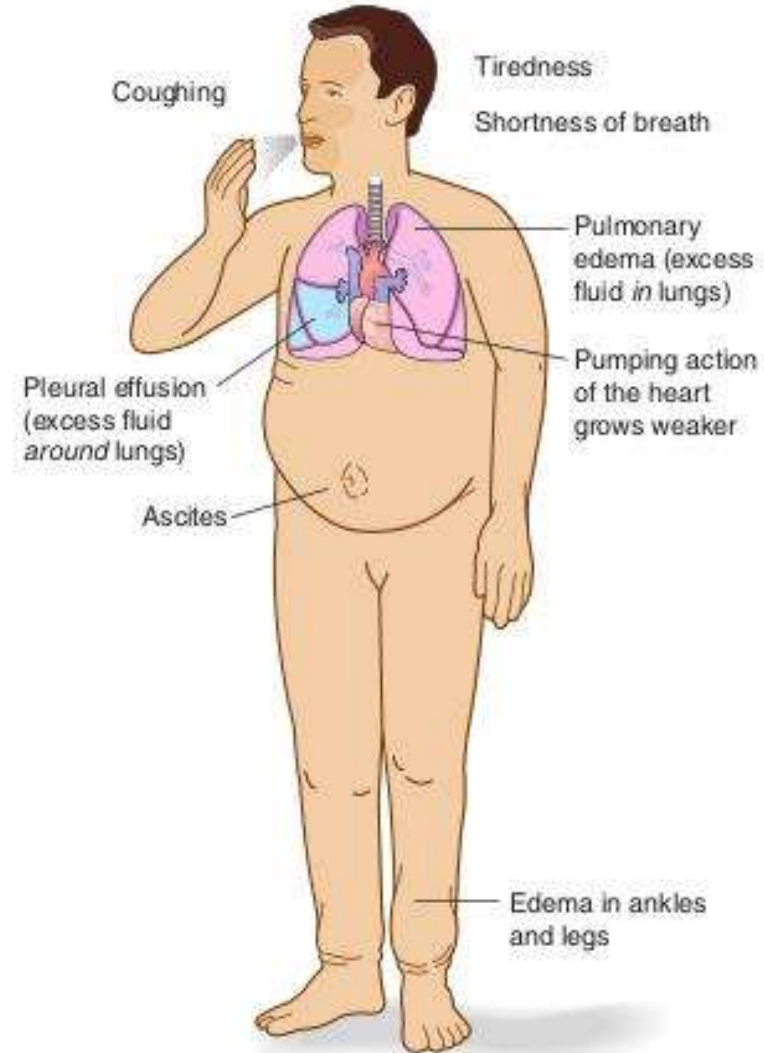
- Age
- Heart attack
- Heredity
- Calcium deposits
- Endocarditis
- Rheumatic fever
- High blood pressure
- Cardiomyopathy
- Connective tissue diseases



Calcified aortic valves

Common symptoms

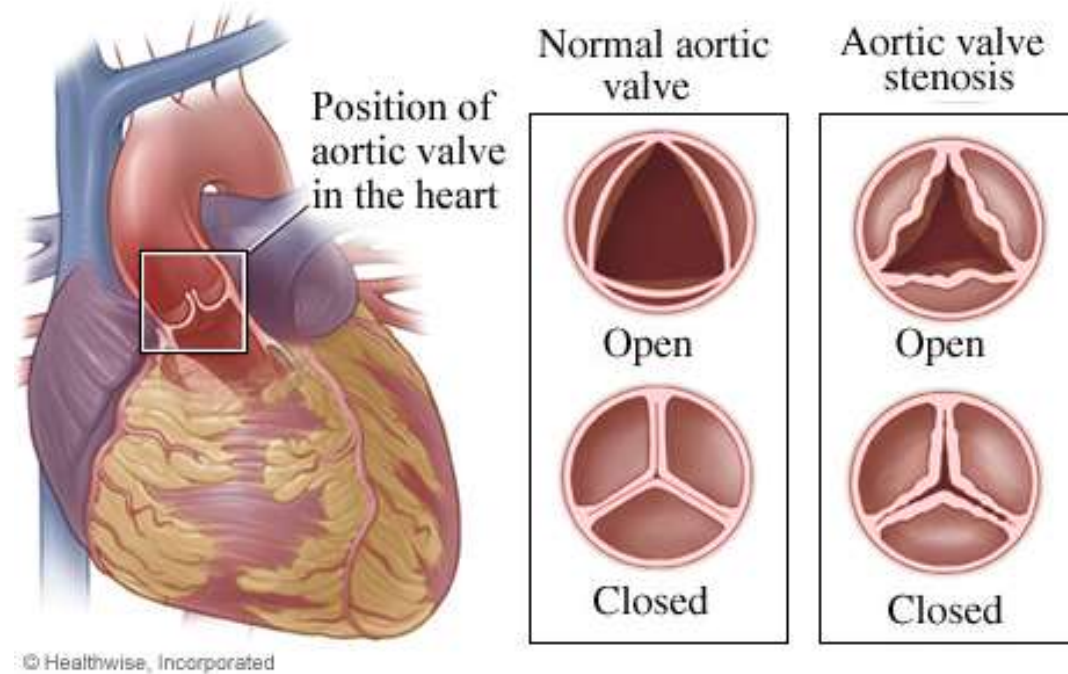
- Shortness of breath
- Weakness
- Dizziness
- Discomfort in chest
- Palpitations
- Edema



Aortic valve stenosis

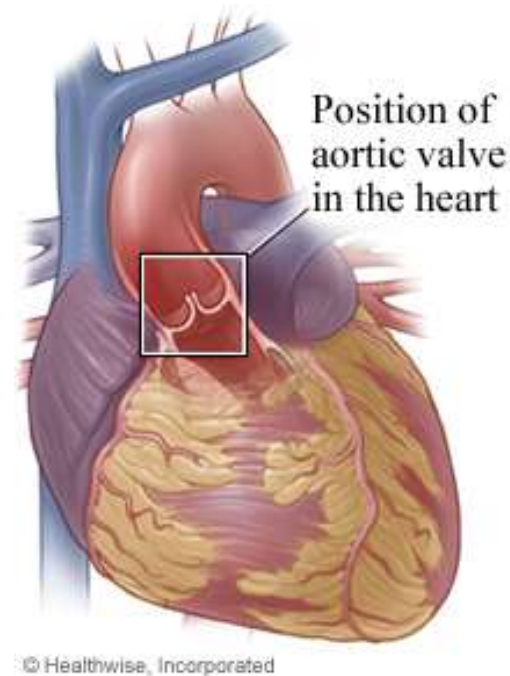
Definition

Aortic stenosis is the obstruction of blood flow across the aortic valve



Classification

Aortic valve stenosis can be classified according to the anatomical location: supravalvular, valvular and subvalvular



Normal aortic valve



Open



Closed

Aortic valve stenosis



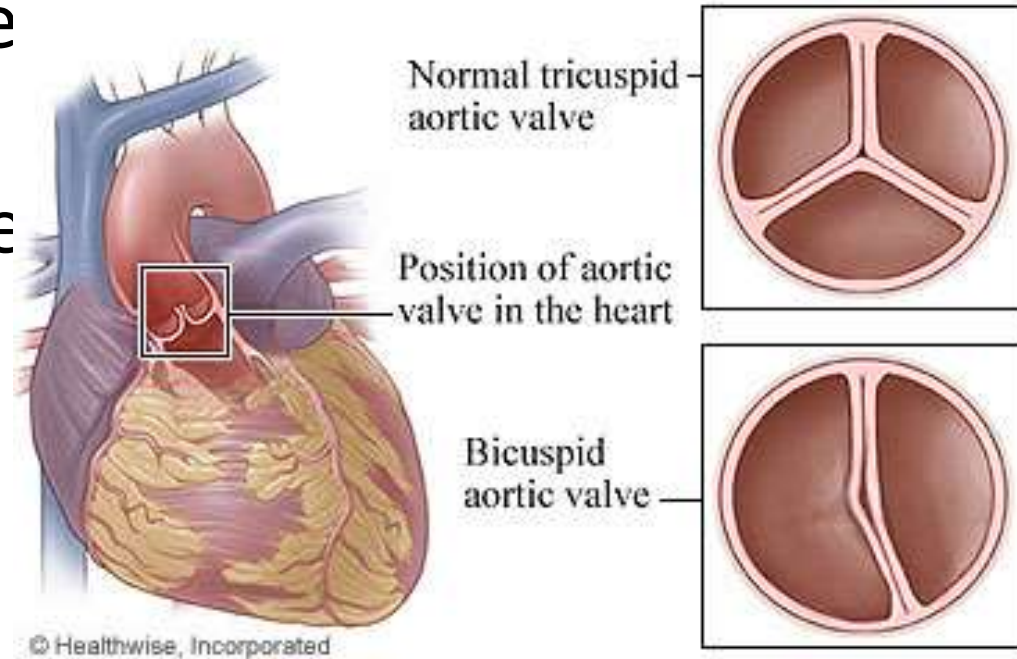
Open



Closed

Causes

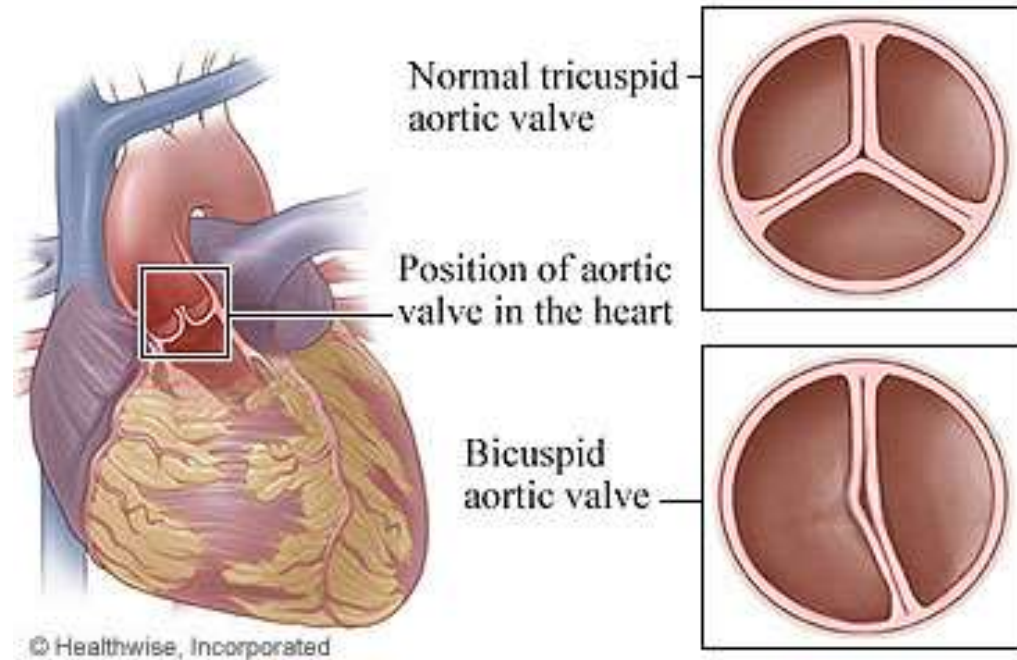
The causes of aortic valve disease include bicuspid aortic valve, degenerative aortic valve disease, rheumatic heart disease, atherosclerotic aortic disease, etc.



Bicuspid aortic valve

Risk factors

Risk factors are closely associated with atherosclerosis including diabetes, smoking, hypertension and dyslipidemia



Bicuspid aortic valve

Pathophysiology 1

- When the aortic valve becomes stenotic, resistance to systolic ejection occurs and a systolic pressure gradient develops between the left ventricle and the aorta
- This outflow obstruction leads to an increase in left ventricular (LV) systolic pressure
- As a compensatory mechanism to normalize LV wall stress, LV wall thickness increases by parallel replication of sarcomeres, producing concentric hypertrophy

Pathophysiology 2

- At this stage, the chamber is not dilated and ventricular function is preserved, although diastolic compliance is reduced
- Eventually, LV end-diastolic pressure (LVEDP) rises, which causes an increase in pulmonary capillary arterial pressures and a decrease in cardiac output due to diastolic dysfunction
- The contractility of the myocardium may also diminish, which leads to a decrease in cardiac output due to systolic dysfunction
- Ultimately, heart failure develops

Pathophysiology 3

- In most patients, LV systolic function is preserved and cardiac output is maintained for many years despite an elevated LV systolic pressure
- Although cardiac output is normal at rest, it often fails to increase appropriately during exercise, which may result in exercise-induced symptoms
- Diastolic dysfunction may occur as a consequence of impaired LV relaxation and/or decreased LV compliance, as a result of increased afterload, LV hypertrophy, or myocardial ischemia

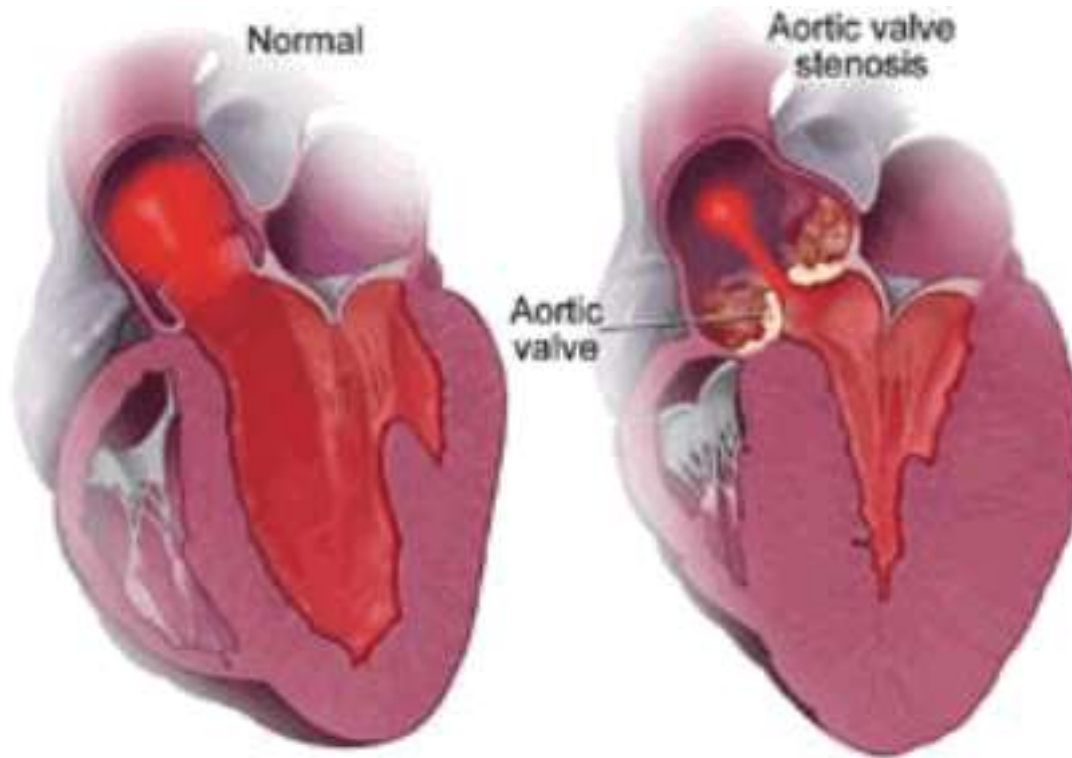
Pathophysiology 4

- LV hypertrophy often regresses following relief of valvular obstruction
- Some individuals develop extensive myocardial fibrosis, which may not resolve despite regression of hypertrophy
- In patients with severe stenosis, atrial contraction plays a particularly important role in diastolic filling of the LV with development of atrial fibrillation often leads to heart failure due to an inability to maintain cardiac output

Pathophysiology 5

- Increased LV mass, increased LV systolic pressure, and prolongation of the systolic ejection phase all elevate the myocardial oxygen requirement, especially in the subendocardial region
- Although coronary blood flow may be normal when corrected for LV mass, coronary flow reserve is often reduced, which results in myocardial ischemia
- Angina results from a concomitant increased oxygen requirement by the hypertrophic myocardium and diminished oxygen delivery secondary to diminished coronary flow reserve, etc.

Heart changes



Normal and aortic stenosis' hearts:
find difference

Signs and symptoms: classic triad

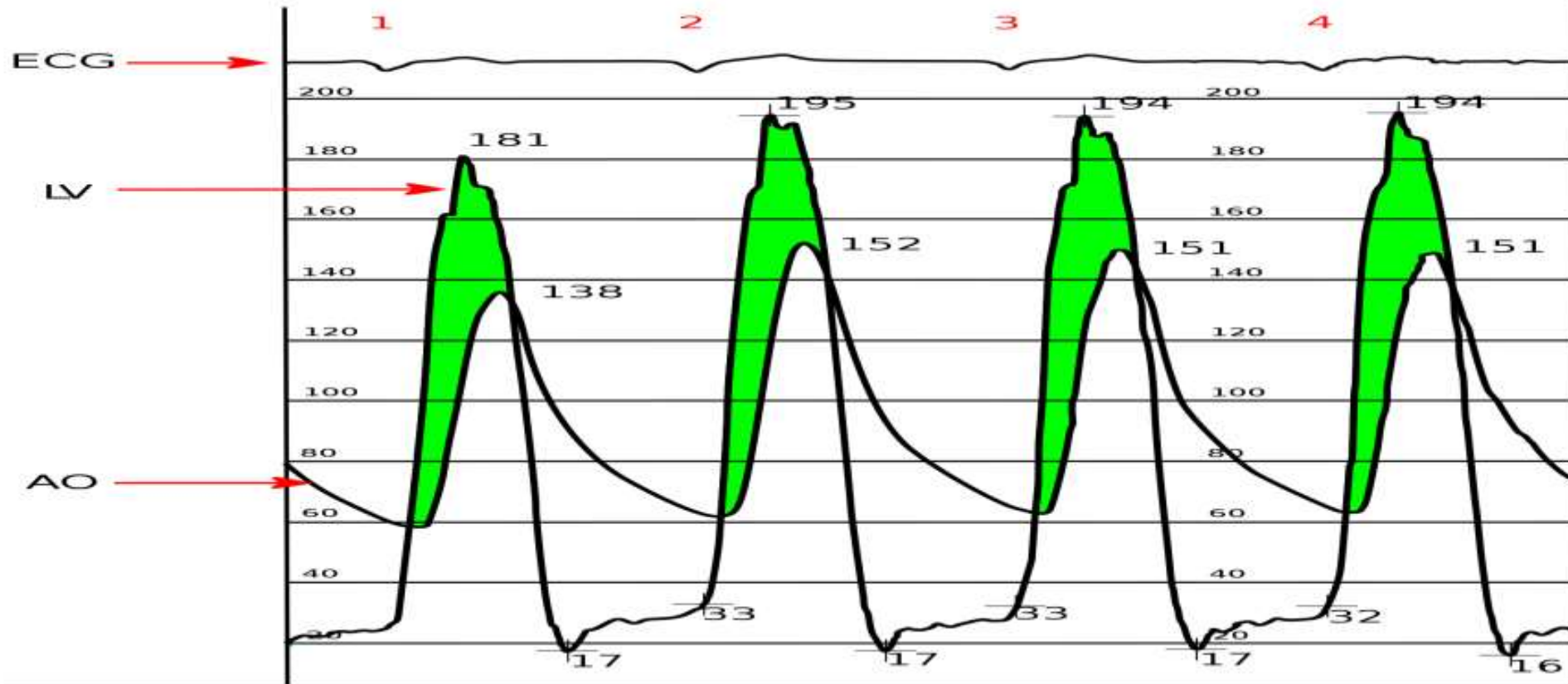


- Chest pain: angina pectoris is typically precipitated by exertion and relieved by rest
- Heart failure: symptoms include paroxysmal nocturnal dyspnea, orthopnea, dyspnea on exertion, and shortness of breath
- Syncope: often occurs upon exertion when systemic vasodilatation in the presence of a fixed forward stroke volume causes the arterial systolic blood pressure to decline

Signs and symptoms: other 1

- Low values of systolic and pulse blood pressure
- Pulsus alternans
- Hyperdynamic left ventricle
- Soft or normal S1, diminished or absent A2, paradoxical splitting of the S2, accentuated P2, ejection click, prominent S4, the classic crescendo-decrescendo systolic murmur

Signs and symptoms: other 2



Pressure gradient between the left ventricle and aorta, suggesting aortic stenosis

Signs and symptoms: other 3

The murmur of aortic regurgitation

Mild aortic regurgitation



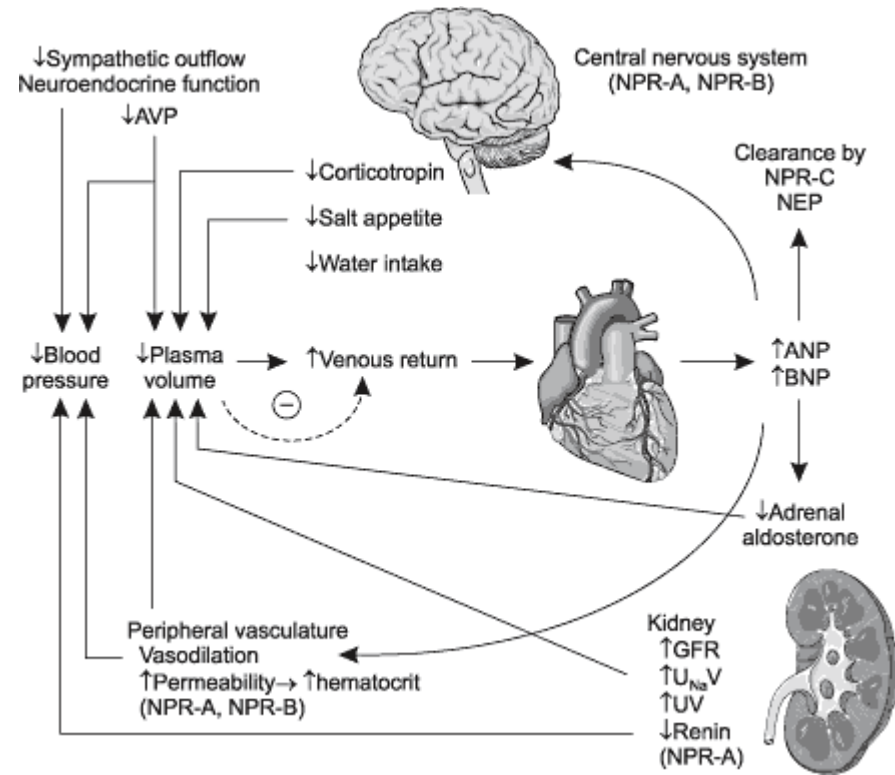
Severe aortic regurgitation



Soft or normal S₁, paradoxical splitting of the S₂, the classic crescendo-decrescendo systolic murmur

Selected laboratory studies

- Serum electrolyte levels
- Cardiac biomarkers
- Complete blood count
- B-type natriuretic peptide: may provide incremental prognostic information for predicting symptom onset in asymptomatic patients with severe aortic stenosis



The effects of B-type natriuretic peptide (BNP) on target organs

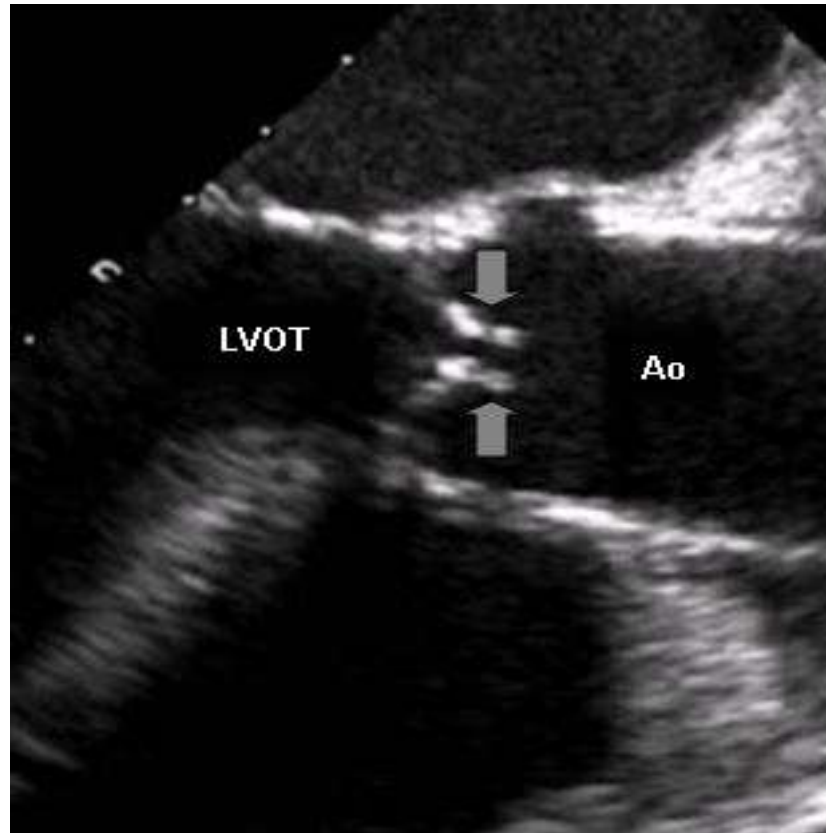
Instrumental studies

- Chest X-ray
- Electrocardiogram
- Echocardiogram
- Exercise electrocardiogram
- Cardiac MRI
- Cardiac catheterization
- Radionuclide ventriculography

Parameters	Slight	Moderate	Severe
PG max	15-40 mmHg	40-70 mmHg	70-100 mmHg (> 100 mmHg severe AoS)
PG mean	<20mmHg	20-40mmHg>	40mmHg
AVA>	1.5cm ²	1.5-1cm ²	<1cm ²
Dimensionless index>	0.50	0.25 to 0.50	<0.25
Vmax	<3m/s	3-4m/s	>4m/s
AVA BSAindex	>0.85cm ² /m ²	0.85-0.60cm ² /m ²	<0.60cm ² /m ²

Aortic stenosis quantification

B-mode echocardiography 1



Transoesophageal echocardiogram of a severely

B-mode echocardiography 2



Severe Aortic Stenosis

B-mode echocardiography 3



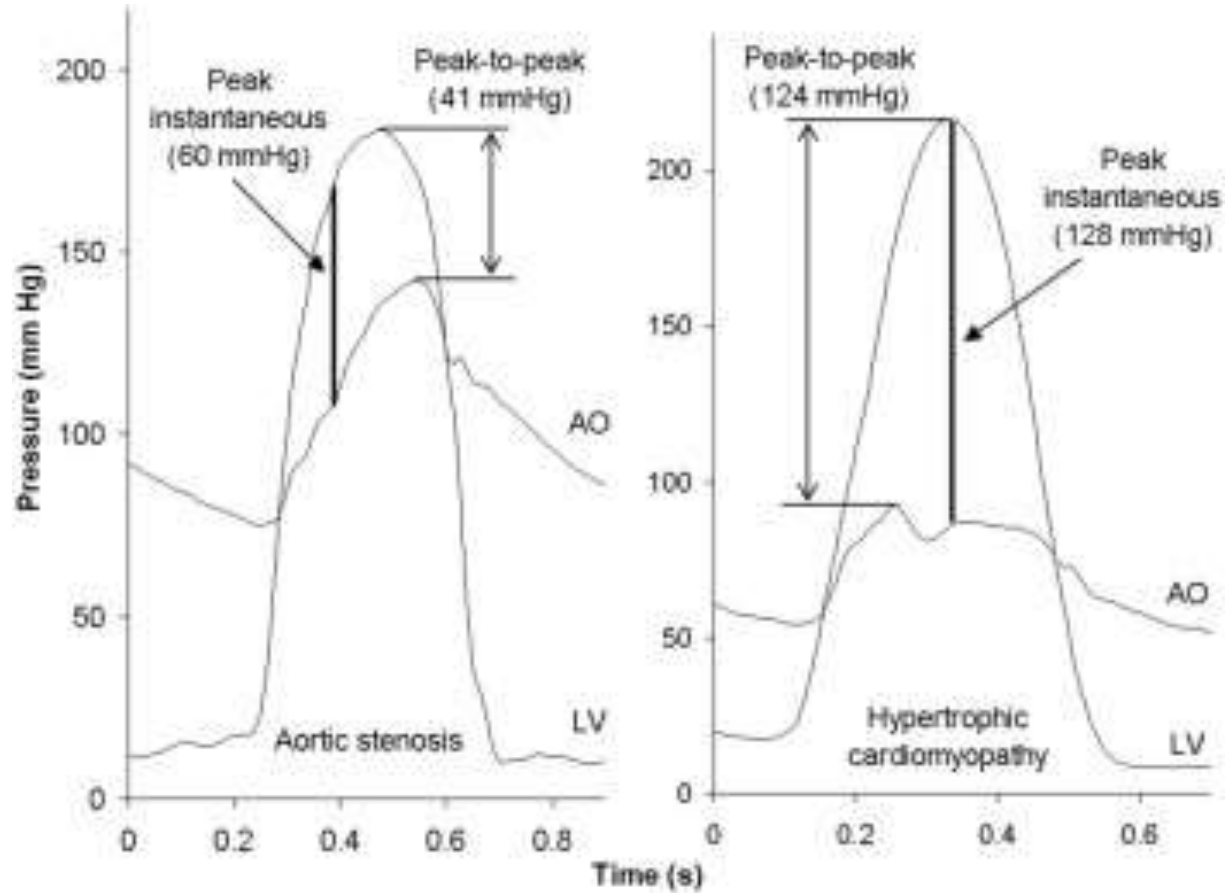
Echocardiography for transcatheter aortic valve implantation

Computer tomography



The effects of B-type natriuretic peptide (BNP) on target organs

Cardiac catheterization

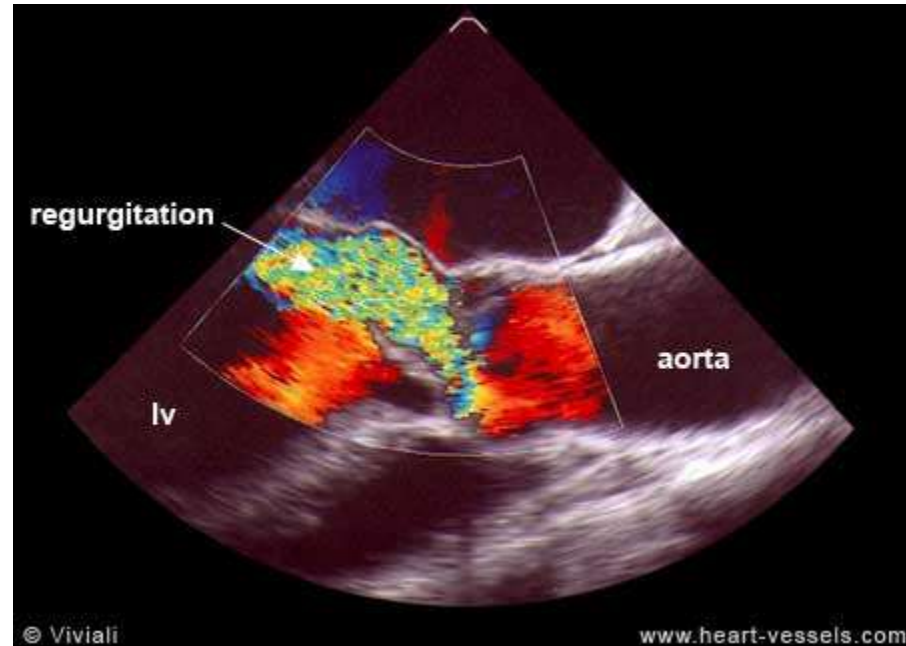


Assessment of Left Ventricular Outflow Gradient

Aortic valve insufficiency/regurgitation

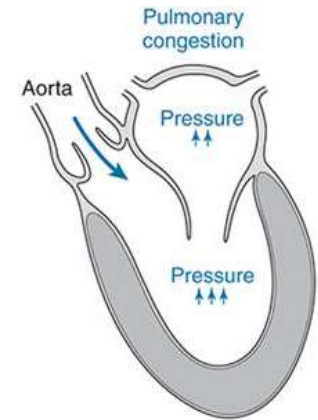
Definition

Aortic insufficiency/
regurgitation (AI) is the
leaking of the aortic valve of
the heart that causes blood
to flow in the reverse
direction during
ventricular diastole, from
the aorta into the left
ventricle

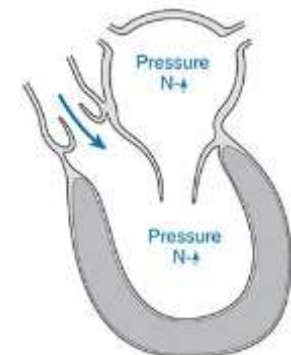


Types

- Acute: the acute onset of aortic regurgitation is usually a medical emergency due to the inability of the left ventricle to quickly adapt to the rapid increase in end-diastolic volume caused by regurgitant blood
- Chronic: the chronic aortic valve regurgitation develops over decades



ACUTE AORTIC REGURGITATION



CHRONIC AORTIC REGURGITATION

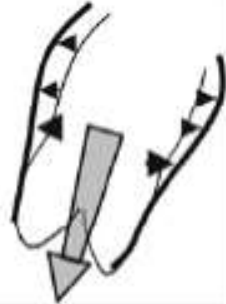
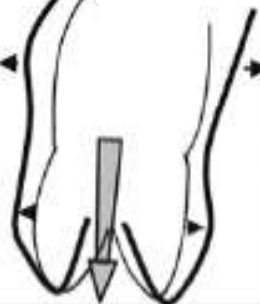
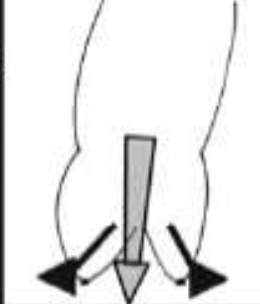

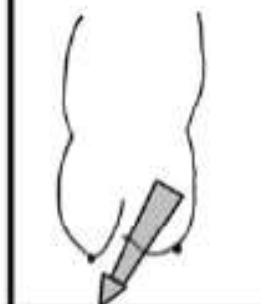
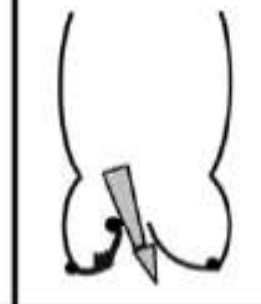
Causes 1

- Half of the cases of AI are due to the aortic root dilation, which is idiopathic in 80% of cases, but otherwise may result from aging, syphilitic aortitis, osteogenesis imperfecta, aortic dissection, Behçet's disease, reactive arthritis and systemic hypertension
- In 15% the cause is innate bicuspidal aortic valve, while another 15% cases are due to retraction of the cusps as part of postinflammatory processes of endocarditis in rheumatic fever/infective endocarditis and various collagen vascular diseases

Causes 2

- AI has been linked to the use of some medications (fenfluramine, dexfenfluramine isomers, dopamine agonists, etc.)
- Other potential causes include Marfan's syndrome, Ehlers–Danlos syndrome, ankylosing spondylitis, etc.

Types of causes

AI Class	Type I Normal cusp motion with FAA dilatation or cusp perforation				Type II Cusp Prolapse	Type III Cusp Restriction
	Ia	Ib	Ic	Id		
Mechanism						

Functional classification of aortic insufficiency (AI)
with description of disease mechanisms

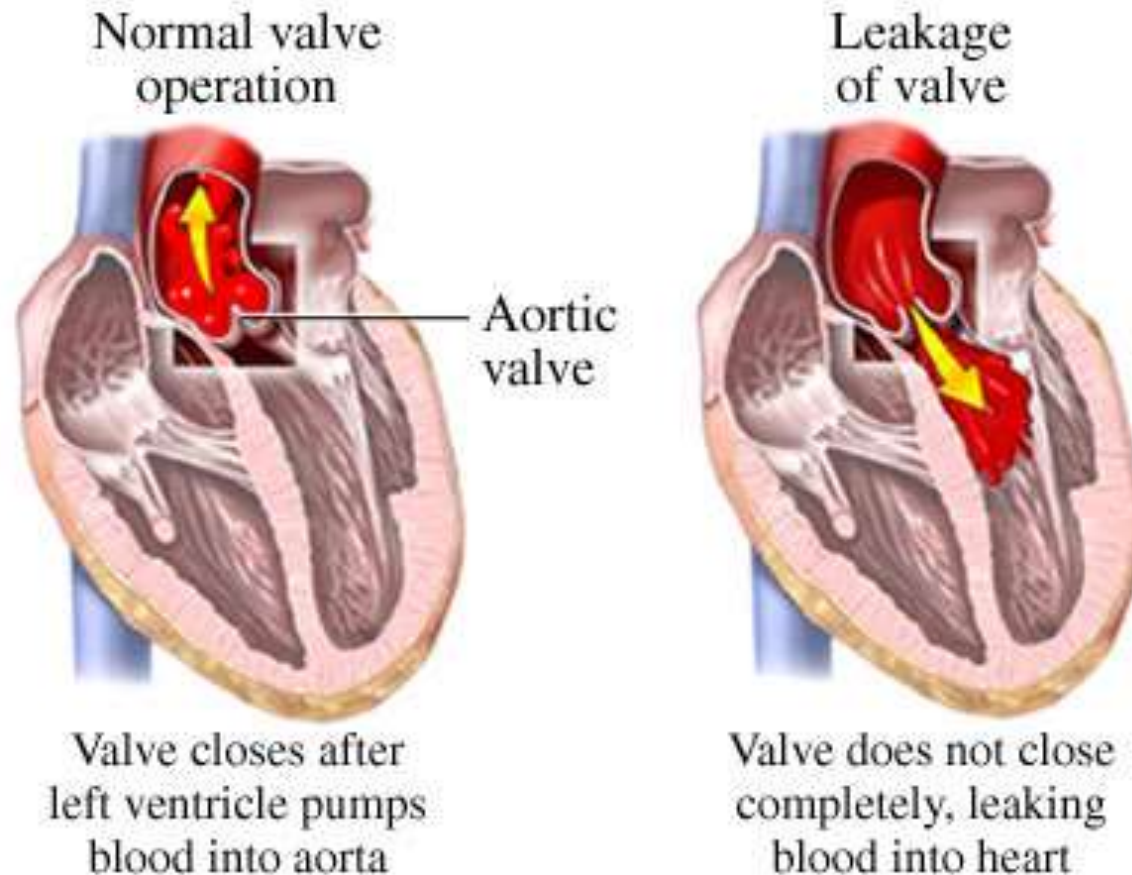
Pathophysiology 1

- In aortic insufficiency (AI), when the pressure in the left ventricle falls below the pressure in the aorta, the aortic valve is not able to completely close, and this causes a leaking of blood from the aorta back into the left ventricle (*regurgitating*)
- The percentage of blood that regurgitates back through the aortic valve due to AI is known as the regurgitant fraction

Pathophysiology 2

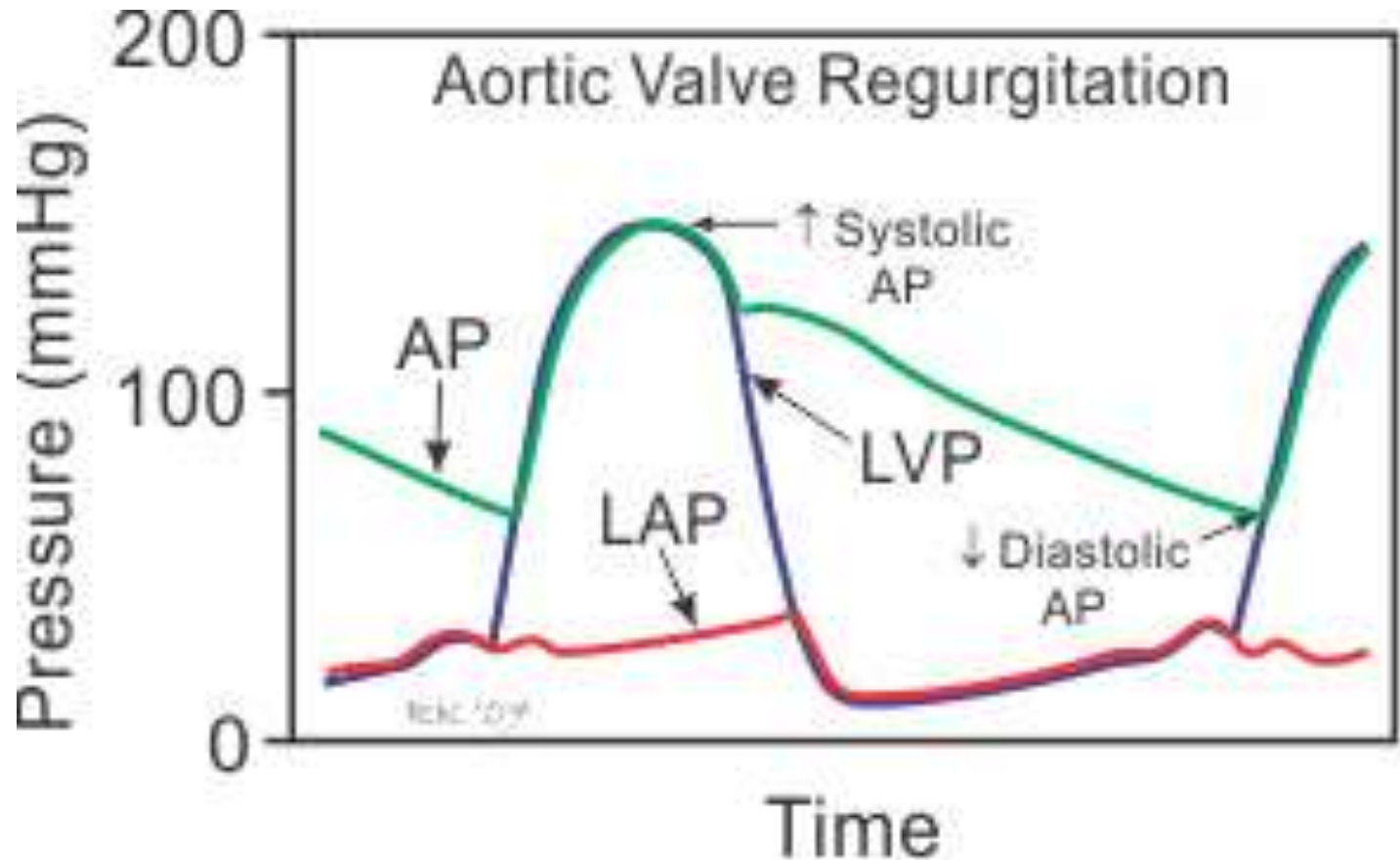
- The regurgitant flow causes a decrease in the diastolic blood pressure in the aorta, and therefore an increase in the pulse pressure
- While diastolic blood pressure is diminished and the pulse pressure widens, systolic blood pressure generally remains normal or can even be slightly elevated
- The volume overload causes left ventricular hypertrophy (LVH) and dilation.

Heart changes



Normal and aortic insufficiency/regurgitation' hearts:
find difference

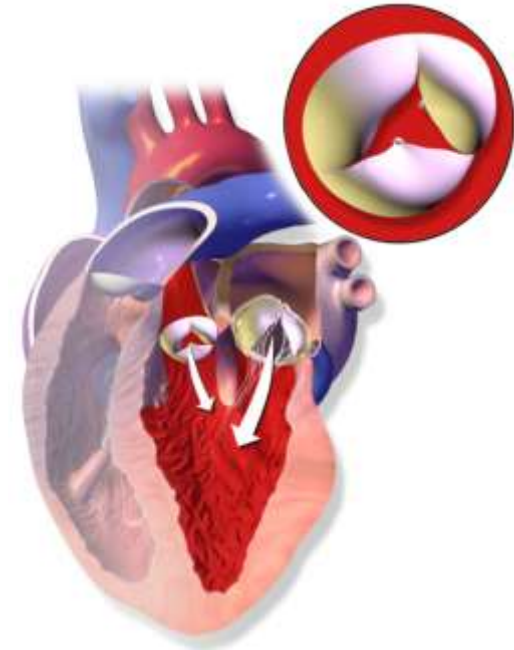
Main hemodynamics disturbances



The changes in aortic pressure (AP), left ventricular pressure (LVP) and left atrial pressure (LAP)

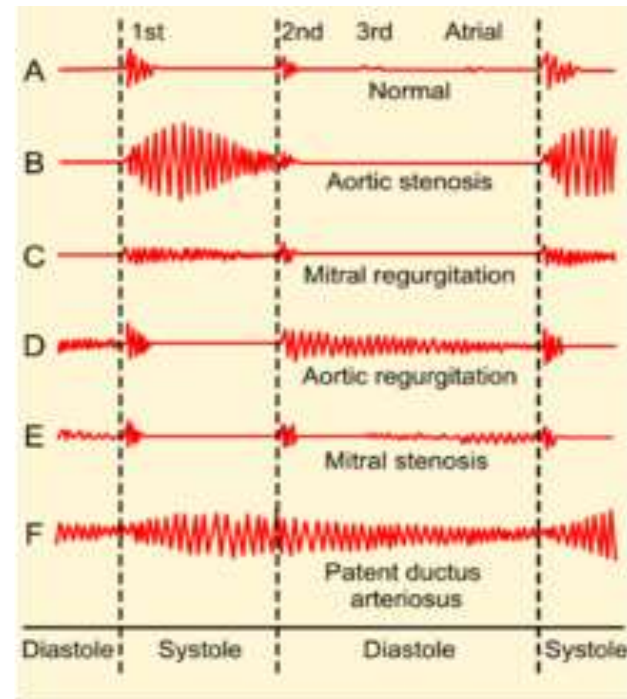
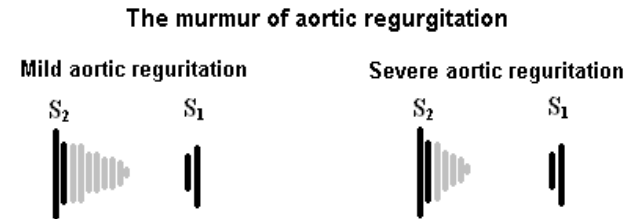
Symptoms

- Dyspnea on exertion, orthopnea
- Paroxysmal nocturnal dyspnea
- Palpitations
- Heart murmur
- Angina pectoris
- Fatigue, weakness
- Swollen ankles and feet (edema)
- Lightheadedness or fainting
- Cyanosis
- Circulatory shock



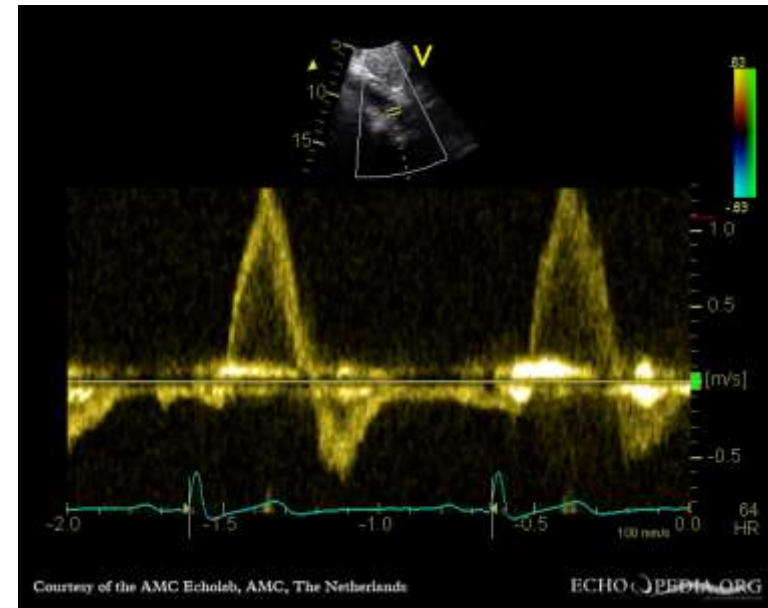
Physical examination

- Heart murmur that can be heard through a stethoscope
- Very forceful beating of the heart
- Bobbing of the head in time with the heartbeat
- Hard pulses in the arms and legs
- Low diastolic blood pressure
- Signs of fluid in the lungs



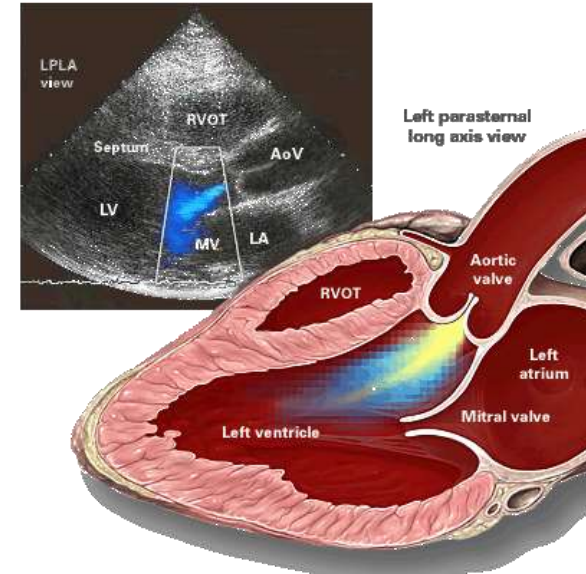
Diagnostic tests

- Echocardiography
- Stress tests
- Cardiac magnetic resonance imaging
- Electrocardiography (ECG)
- Chest X-ray
- Cardiac catheterization



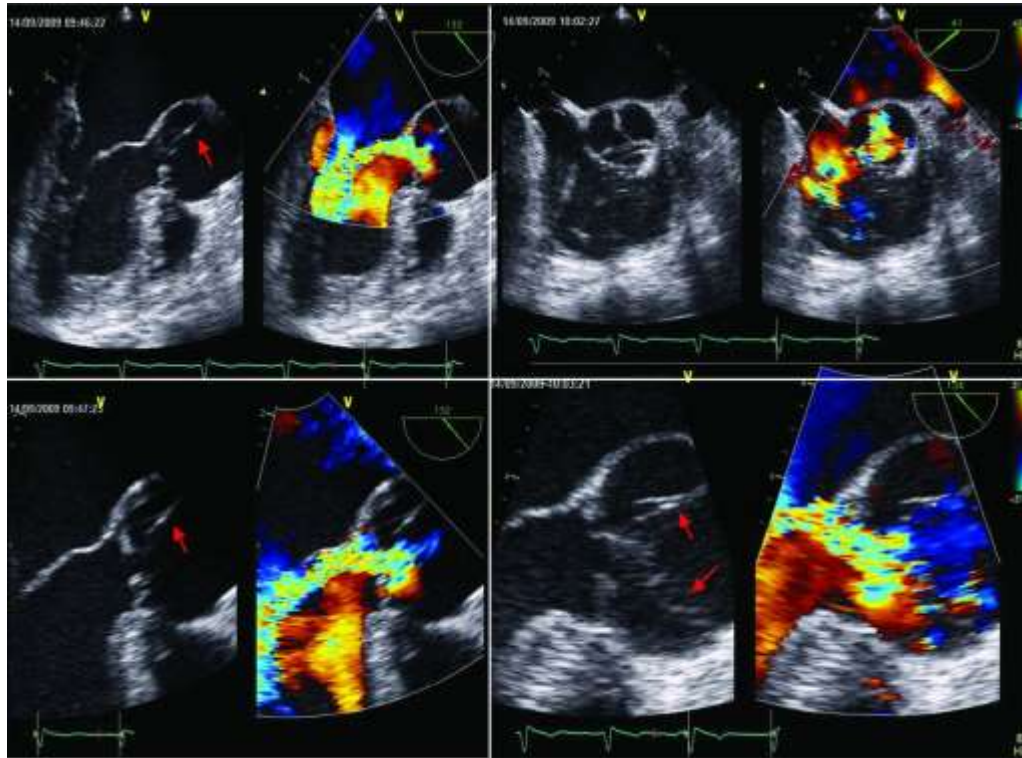
Echocardiography 1

- The severity of aortic regurgitation is estimated using three parameters on echocardiography:
- Regurgitant jet size
- Pressure half-time
- Regurgitant fraction



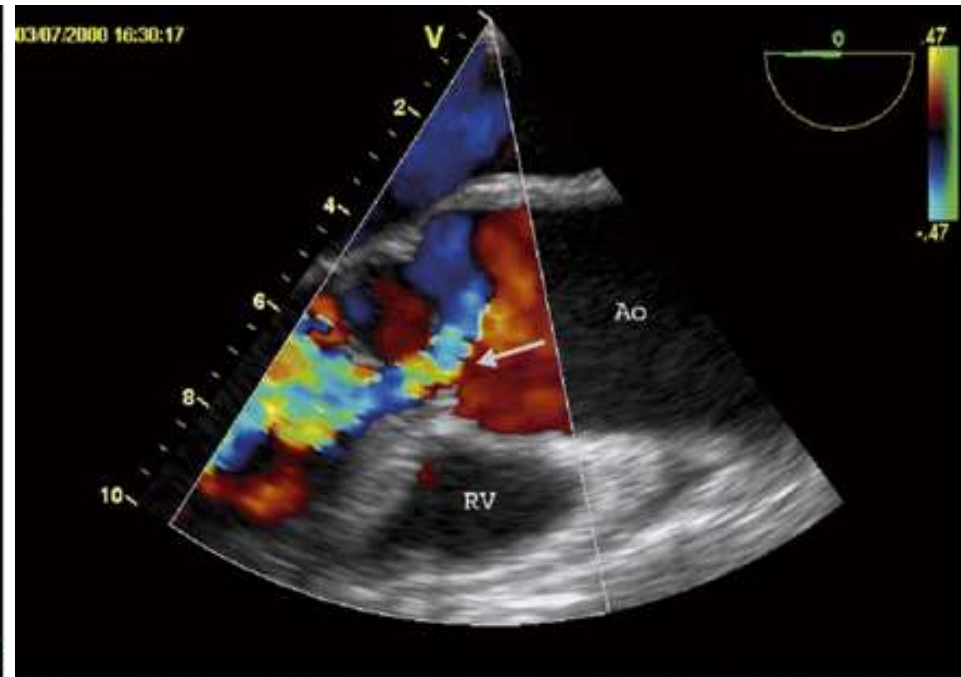
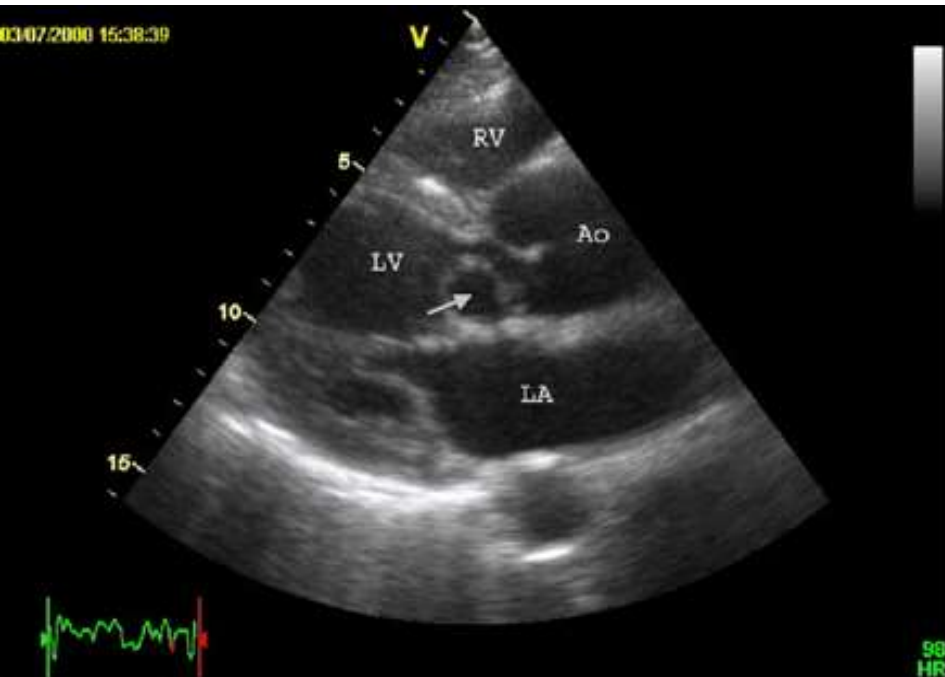
Severity	Jet Size Ratio	Pressure Half-Time	Regurgitant Fraction (%)
Mild	<24	>500	<20
Moderate	25-45	500-349	20-35
Moderate-severe	46-64	349-200	56-50
Severe	> 65	< 200	> 50

Echocardiography 2



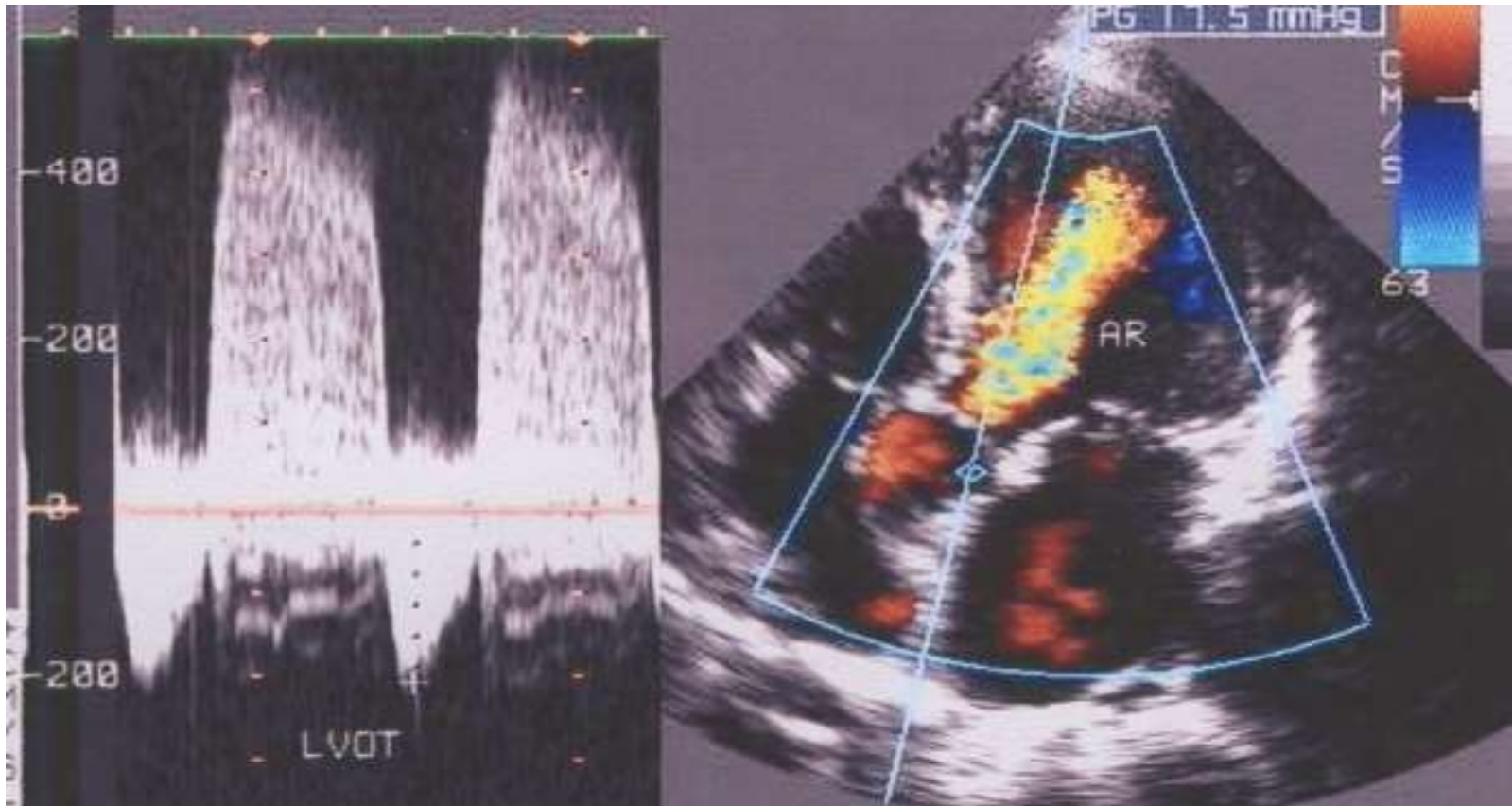
Transesophageal echo showing the chordae tendineae strands (arrows) connecting a mildly dilated aortic root to sigmoid cusps that present tenting and restriction with a severe aortic regurgitation

Echocardiography 3



Aortic valve regurgitation due to cusp aneurysm

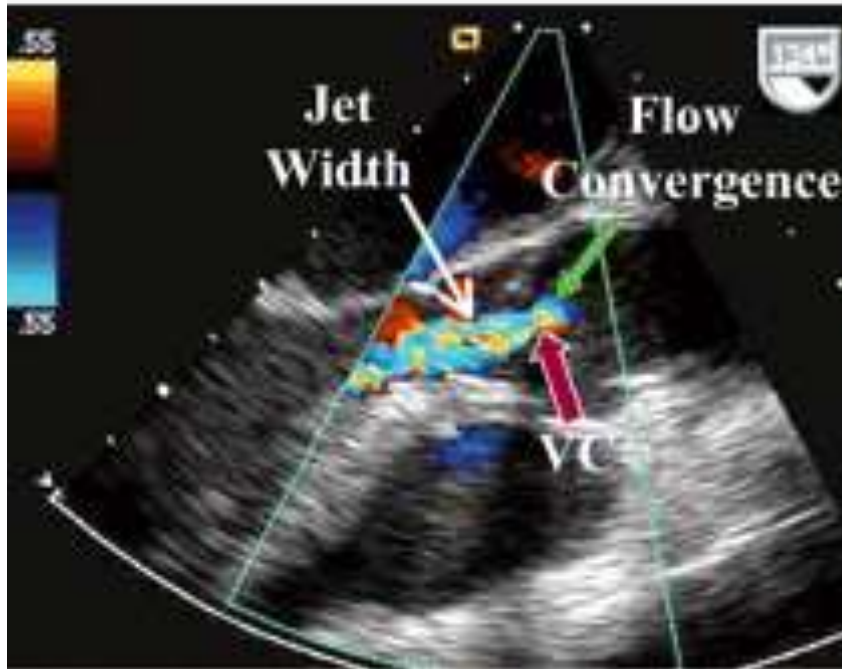
Echocardiography 4



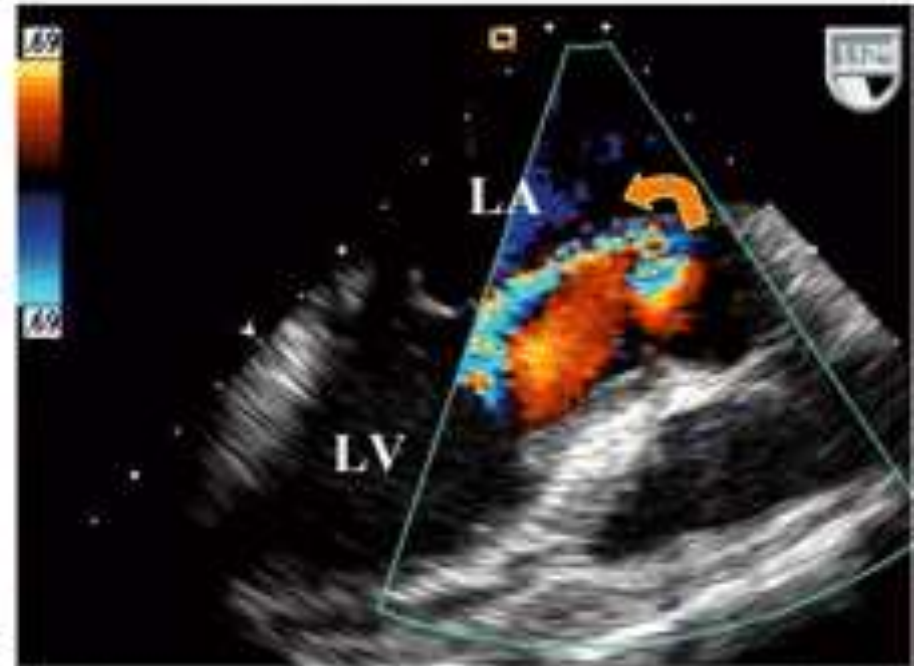
Aortic regurgitation (multicoloured mosaic jet seen in left ventricle)

Echocardiography 5

Central AR Jet

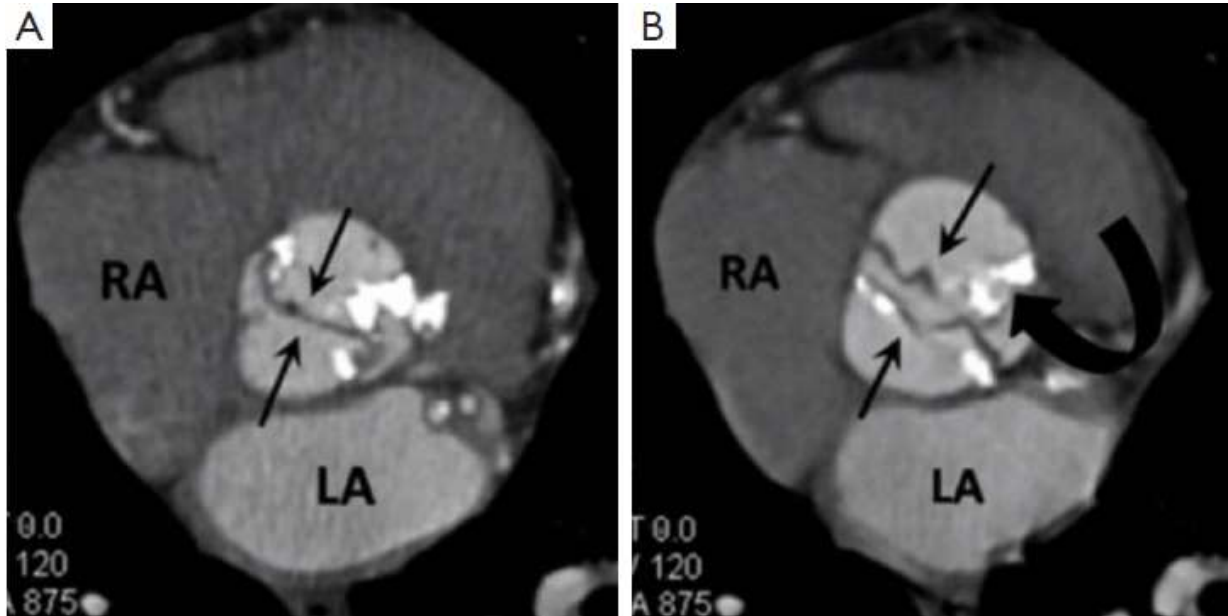


Eccentric AR Jet



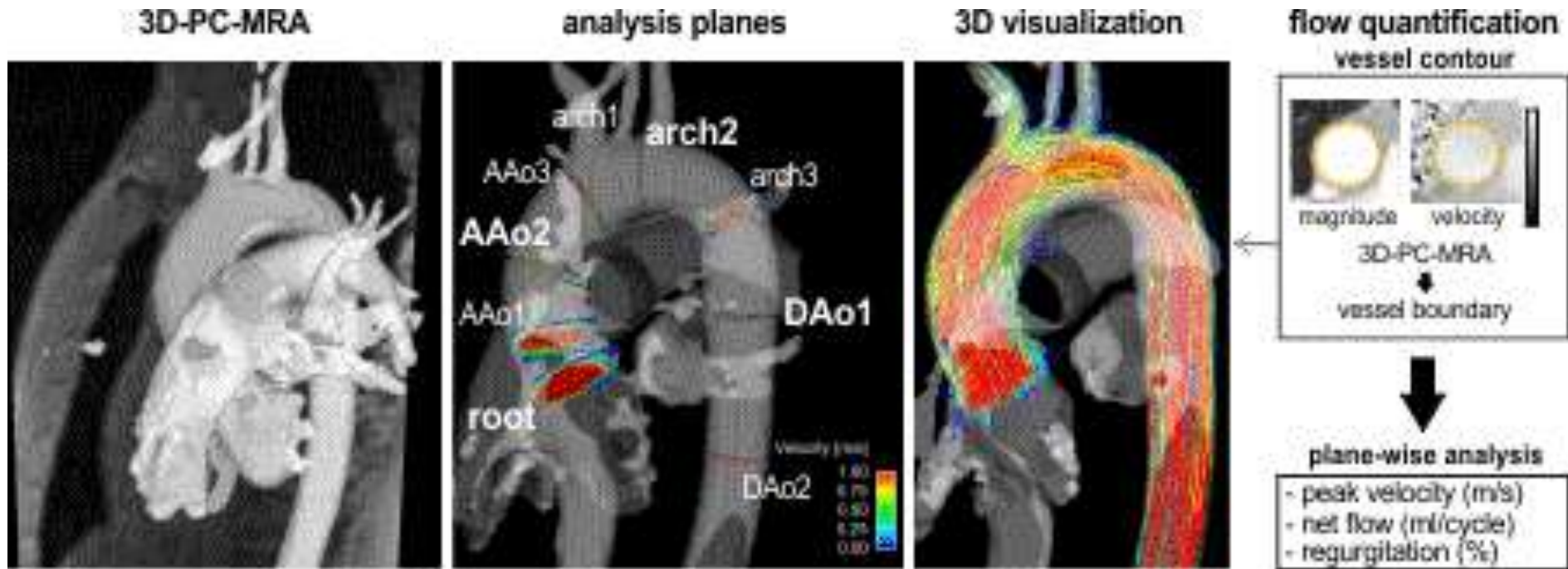
Examples of central and eccentric aortic regurgitation (AR) jets

Cardiac magnetic resonance imaging 1



(A) CT reconstruction in diastole showing an apparent single line of valve fusion (arrows) raising the possibility of a BAV. (B) CT reconstruction in systole showing an “fishmouth” opening pattern in keeping with a BAV. BAV, bicuspid aortic valve

Cardiac magnetic resonance imaging 2



4D flow MRI-based analysis of aortic hemodynamics can be performed with good reproducibility and accuracy using a new faster and semi-automated workflow

Computed tomography



Arrow points to vegetation below non-coronary cusp (NCC). Vegetation measures 10 x 6mm in this view.

Stress tests

Parameters	Baseline	20 μ g/kg/min	P
HR (bpm)	74.5 \pm 12.2	94.2 \pm 18.0	<0.001
EF (%)	62.3 \pm 7.9	71.5 \pm 10.5	<0.001
mG (mmHg)	6.8 \pm 2.5	14.7 \pm 9.3	<0.001

Bpm - beats per minute, HR - heart rate; EF - ejection fraction, mG - mean aortic gradient; min - minutes.

Dobutamine-stress echocardiography

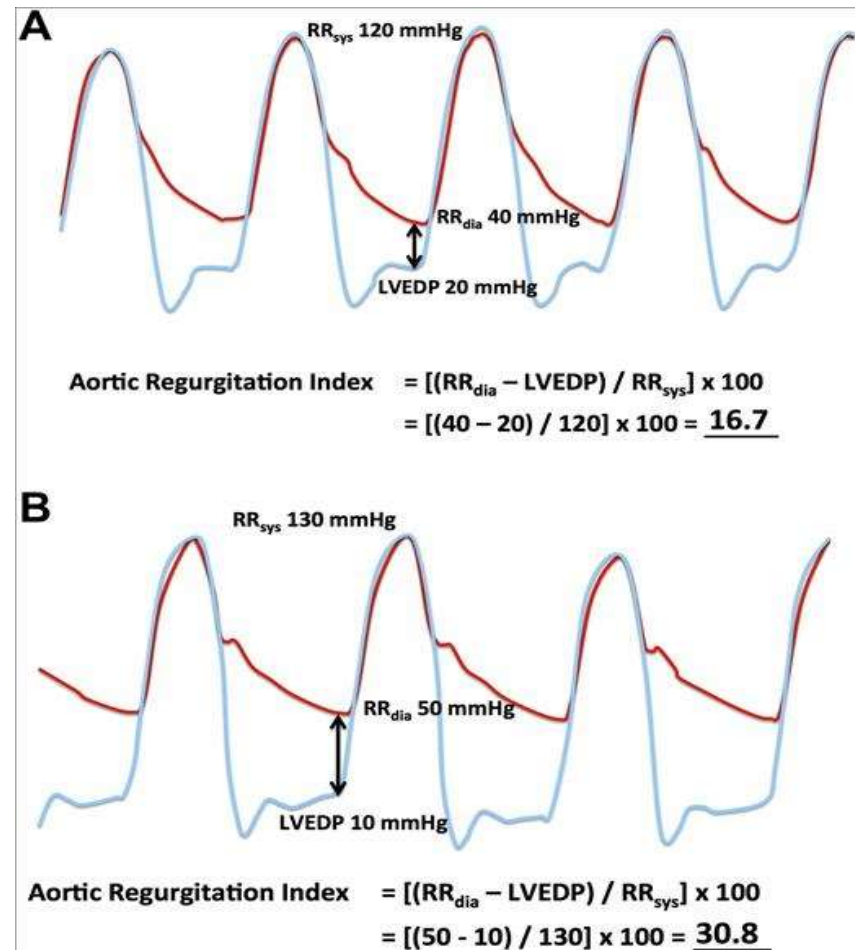
Chest X-ray



A case of severe aortic valve regurgitation caused by an ascending aortic aneurysm in a young patient

Cardiac catheterisation

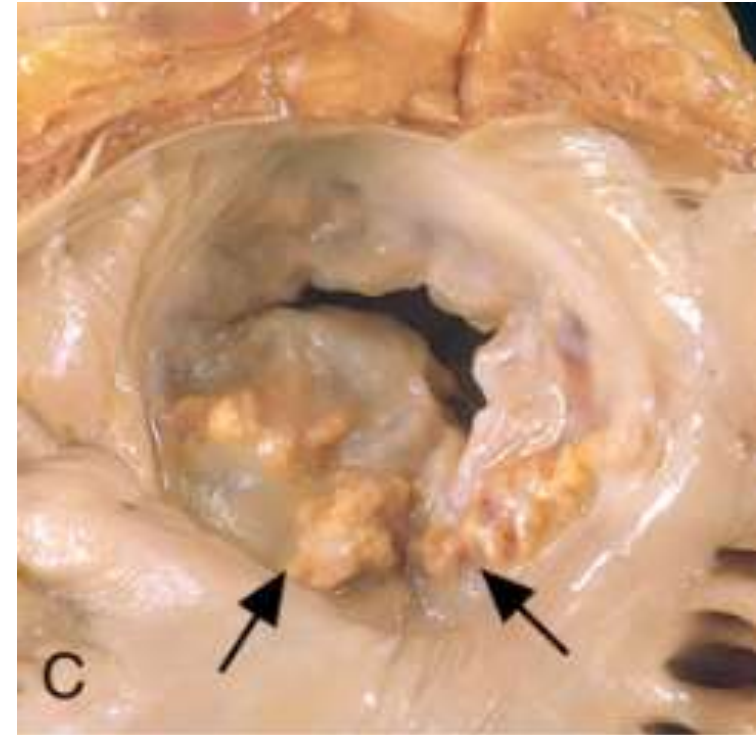
After adjustment for the systolic blood pressure level, the transvalvular gradient results in an ARI of 16.7 in a patient with moderate paravalvular aortic regurgitation PAR (A) and an ARI of 30.8 in a patient with trivial PAR(B).



Mitral valve stenosis

Definition

Mitral stenosis (MS) is characterized by obstruction to left ventricular inflow at the level of mitral valve due to structural abnormality of the mitral valve apparatus



Causes

- The most common cause is rheumatic fever
- Other causes: malignant carcinoid disease, systemic lupus erythematosus, rheumatoid arthritis, mucopolysaccharidoses of the Hunter-Hurler phenotype, Fabry disease, Whipple disease, methysergide therapy, nonrheumatic mitral annular calcification, etc.

Pathophysiology 1

- The normal mitral valve orifice area is approximately 4-6 cm²; as the orifice size decreases, the pressure gradient across the mitral valve increases to maintain adequate flow
- Patients will not experience valve-related symptoms until the valve area is 2-2.5 cm² or less, at which point moderate exercise or tachycardia may result in exertional dyspnea from the increased transmitral gradient and left atrial pressure

Pathophysiology 2

- Severe MS occurs with a valve area of less than 1 cm²; as the valve progressively narrows, the resting diastolic mitral valve gradient, and hence left atrial pressure, increases; this leads to transudation of fluid into the lung interstitium and dyspnea at rest or with minimal exertion
- Hemoptysis may occur if the bronchial veins rupture and left atrial dilatation increases the risk for atrial fibrillation and subsequent thromboembolism

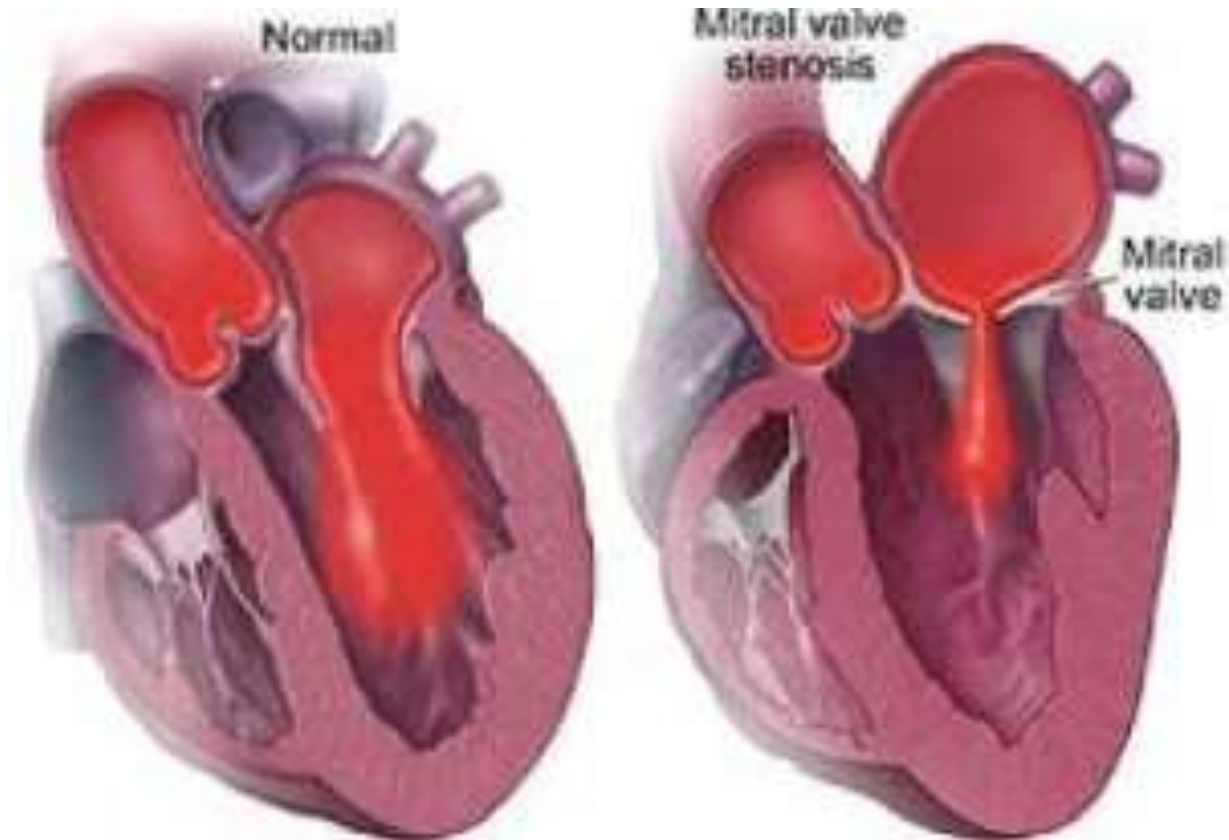
Pathophysiology 3

- Pulmonary hypertension may develop as a result of (1) retrograde transmission of left atrial pressure, (2) pulmonary arteriolar constriction, (3) interstitial edema, or (4) obliterative changes in the pulmonary vascular bed
- As pulmonary arterial pressure increases, right ventricular dilation and tricuspid regurgitation may develop, leading to elevated jugular venous pressure, liver congestion, ascites, and pedal edema

Pathophysiology 4

- LVEDP and cardiac output are usually normal in the person with isolated mitral stenosis
- As the severity of MS increases, the cardiac output becomes subnormal at rest and fails to increase during exercise
- 1/3 of patients with rheumatic MS have depressed LV systolic function as a result of chronic rheumatic myocarditis

Heart changes



Normal and mitral valve stenosis' hearts:
find difference

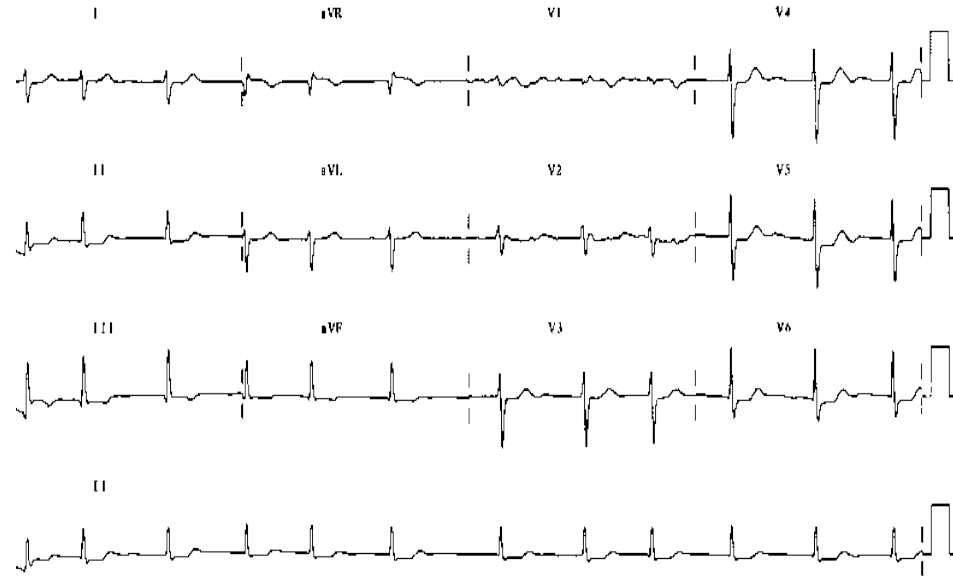
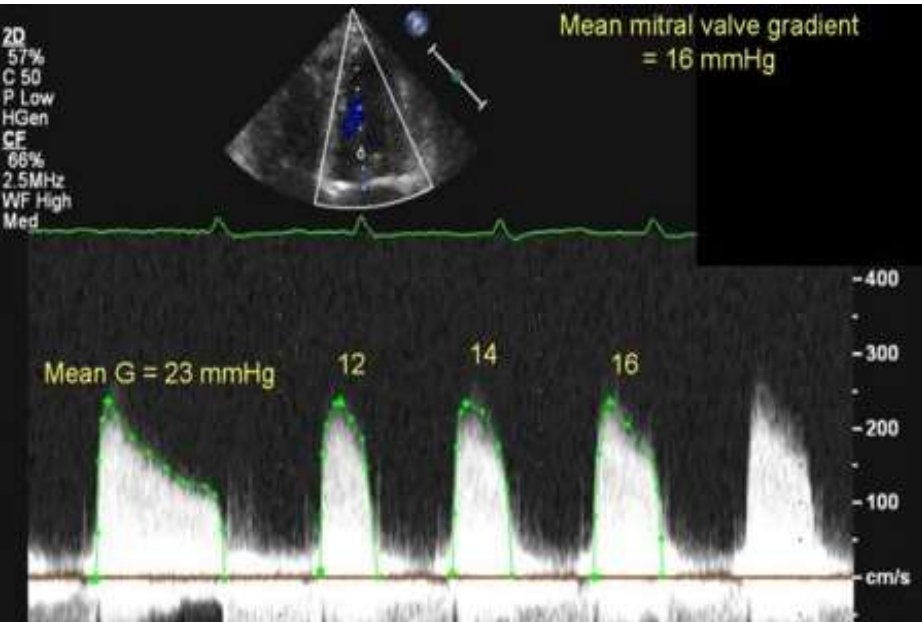
Signs and symptoms 1

- Irregular pulse of atrial fibrillation
- Rise in jugular venous pressure
- Parasternal heave with right ventricular hypertrophy
- Loud first heart sound
- Opening snap, which disappears as the leaflets become rigid

Signs and symptoms 2

- Classic late diastolic murmur with presystolic accentuation
- Shortness of breath
- Fatigue or weakness
- Paroxysmal nocturnal dyspnea (cardiac asthma)

Signs and symptoms 3



Atrial fibrillation

Less common signs and symptoms

- Hoarseness and vocal cord paralysis
- Trouble swallowing
- Chest pain
- Skin color changes, such as pink to purple shades on the cheeks or a dark blue color on the body from reduced blood flow in the end stages of the disease
- Coughing up blood (Hemoptysis)
- Thromboembolism in later stages
- Ascites and edema and hepatomegaly (right-side heart failure)



Physical examination 1

- Jugular venous distension due to rise in jugular venous pressure (with coexistent tricuspid regurgitation)
- Irregular pulse of atrial fibrillation
- Parasternal heave with right ventricular hypertrophy
- Tapping apex beat (manifestation of a loud first heart sound)

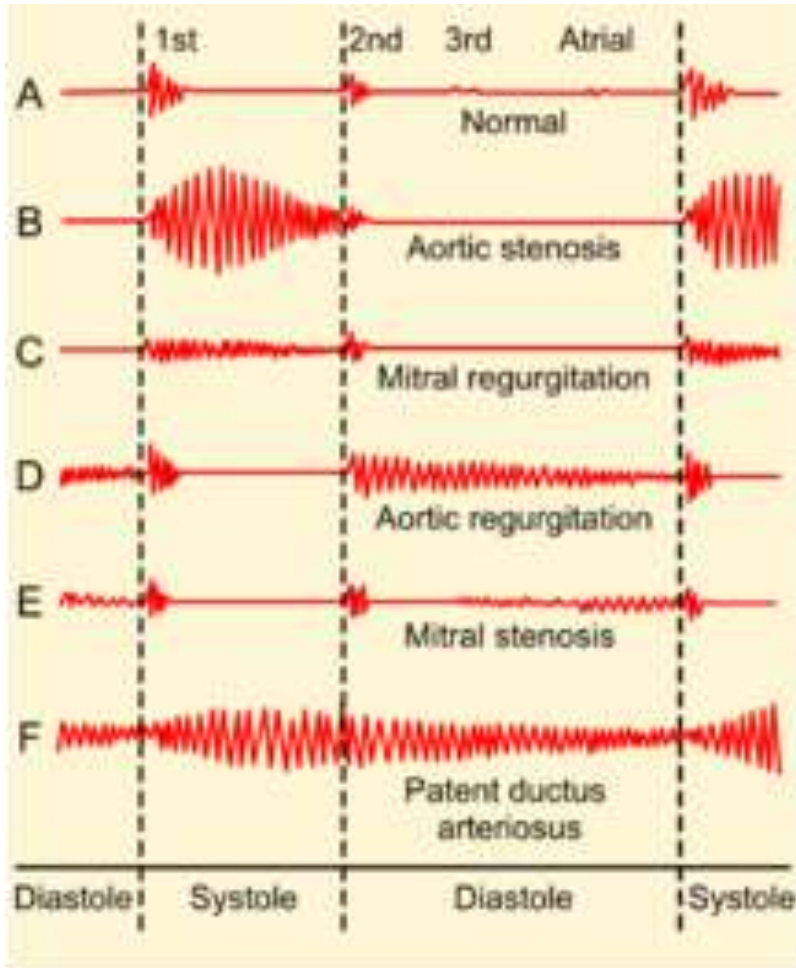
Physical examination 2

- Shift of heart dullness upward (left atrium) and to the right (right ventricle)
- The first heart sound is usually loud and may be palpable (tapping apex beat) because of increased force in closing the mitral valve
- If pulmonary hypertension secondary to mitral stenosis is severe, the pulmonic component of the second heart sound will become loud

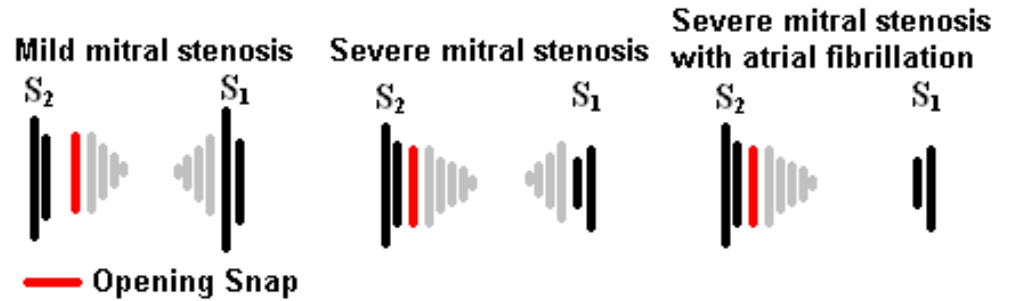
Physical examination 3

- A mid-diastolic rumbling murmur with presystolic accentuation will be heard after the opening snap
- Signs of pulmonary hypertension
- Hepatomegaly
- Ascites

Physical examination



The murmur of mitral stenosis



Auscultation

Selected laboratory studies 1

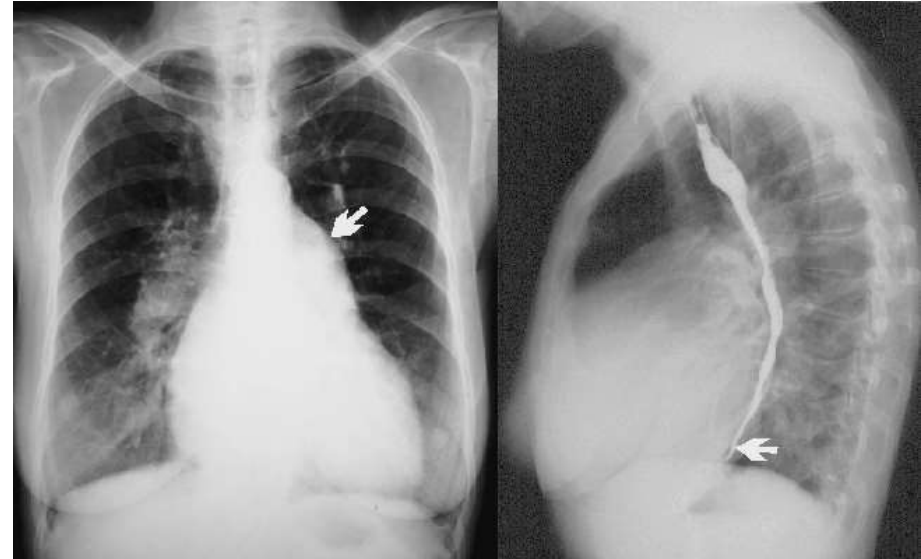
- Serum electrolyte levels
- Cardiac biomarkers
- Complete blood count
- Antistreptolysin O (ASLO) antibodies
- Renal and liver function tests
- Acute phase reactions: ESR / CRP / Leukocytosis

Selected laboratory studies 2

- Evidence of antecedent Strep. infection: ASO / Strep antibodies / Strep group A throat culture / Recent scarlet fever / anti-deoxyribonuclease B / anti-hyaluronidase
- Levels of antinuclear antibodies, etc.
- Assess for amyloid deposits in affected tissues

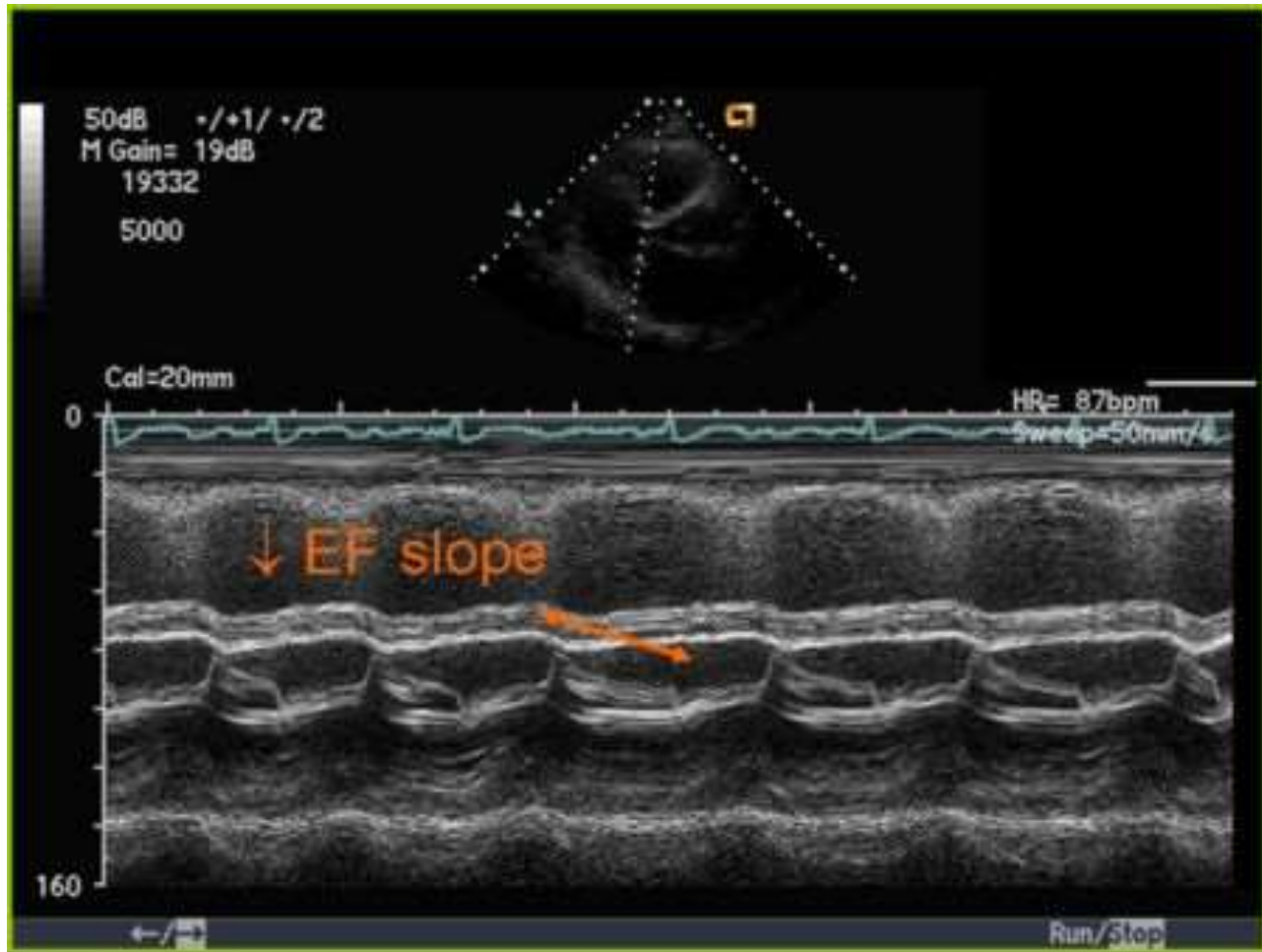
Instrumental studies

- Chest X-ray
- Electrocardiogram
- Echocardiogram
- Cardiac MRI
- Cardiac catheterization
- Radionuclide ventriculography



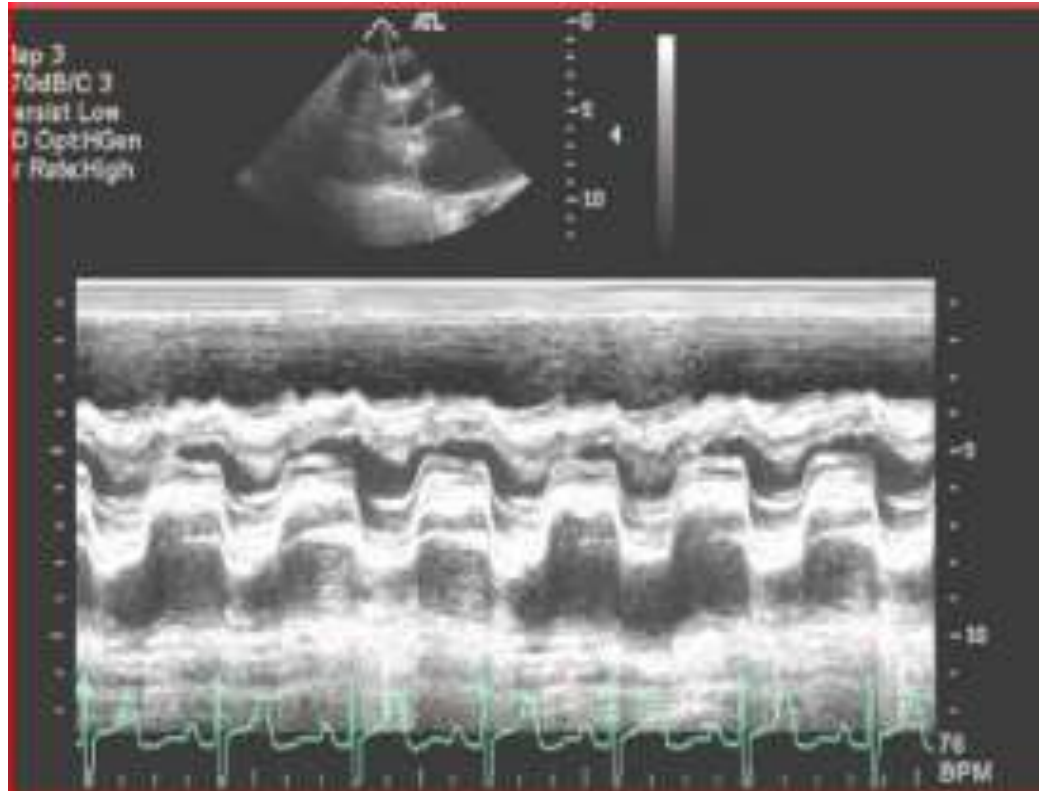
Mitral stenosis

M -mode echocardiography



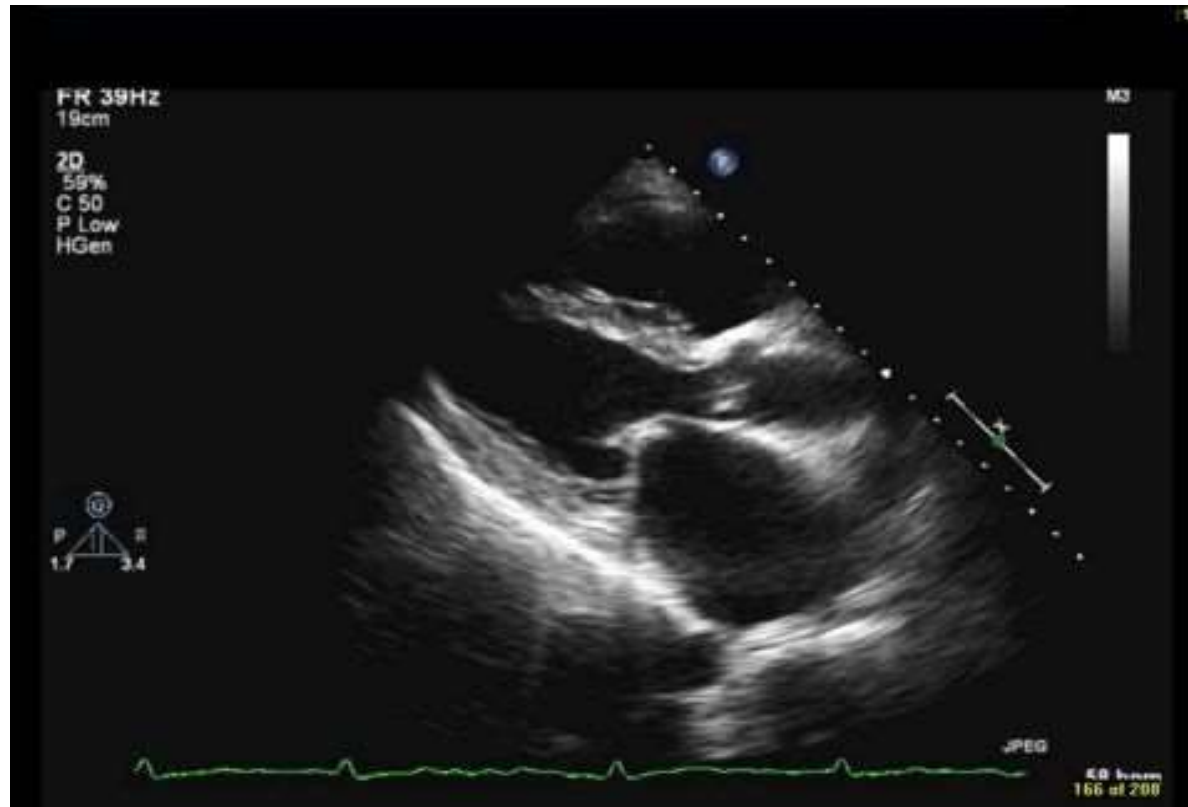
Mitral Stenosis M Mode

M and B -mode echocardiography



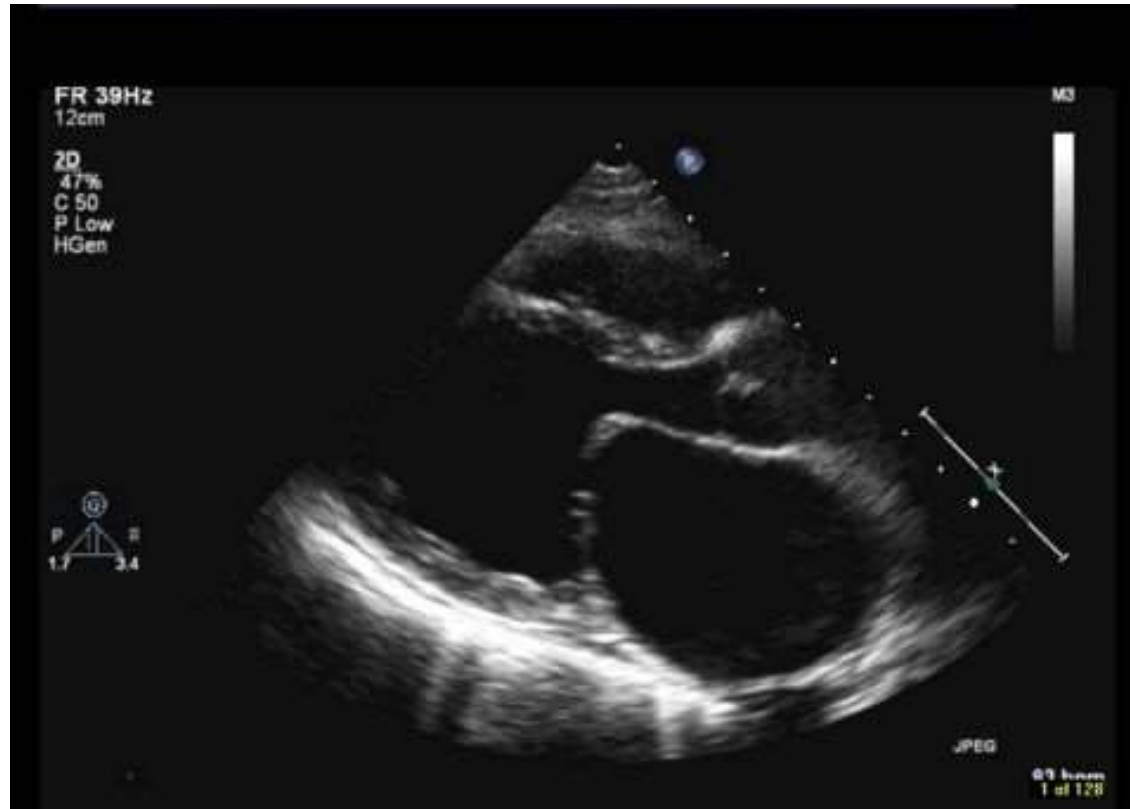
Hyperechogenicity of valvular leaflets and annulus together with calcification of supporting valvular structures

B-mode echocardiography 1



There is thickening and fusion of the mitral valve commissural edges and chordae, which will result in a "doming" appearance of the mitral valve opening

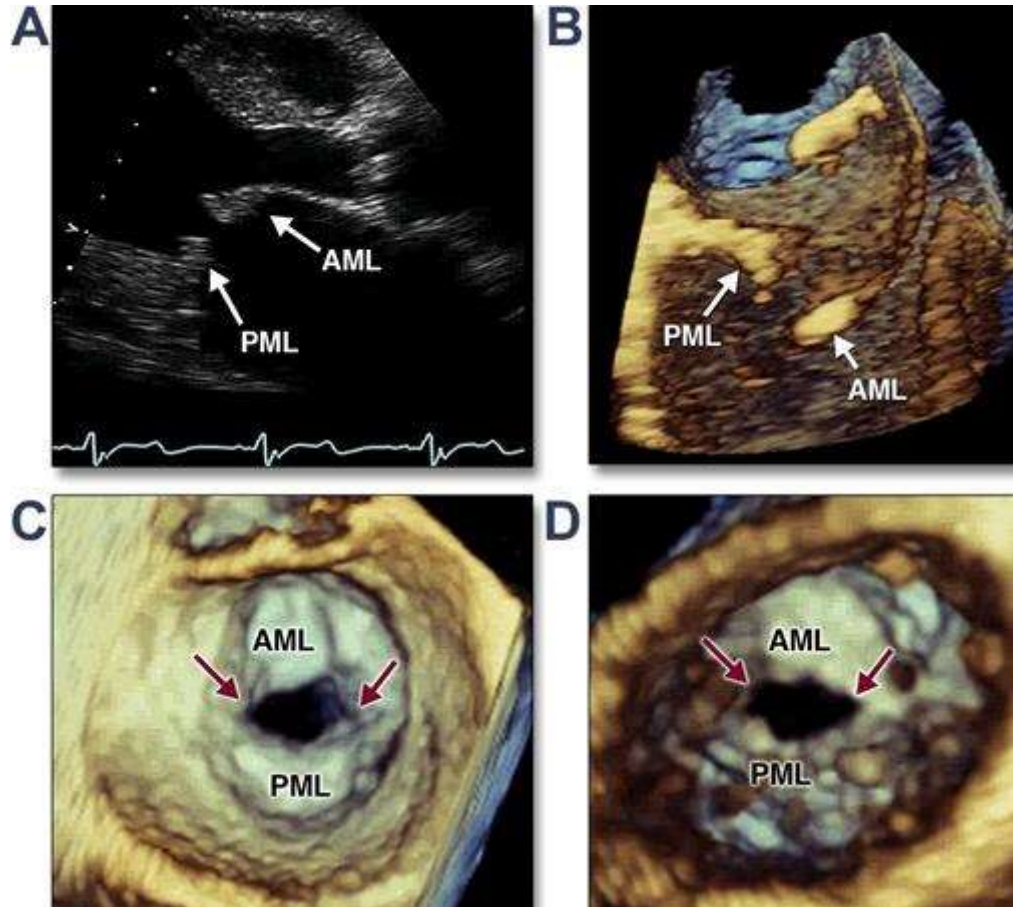
B-mode echocardiography 2



The anterior leaflet has been described as opening in a "hockey stick" appearance in parasternal long axis view

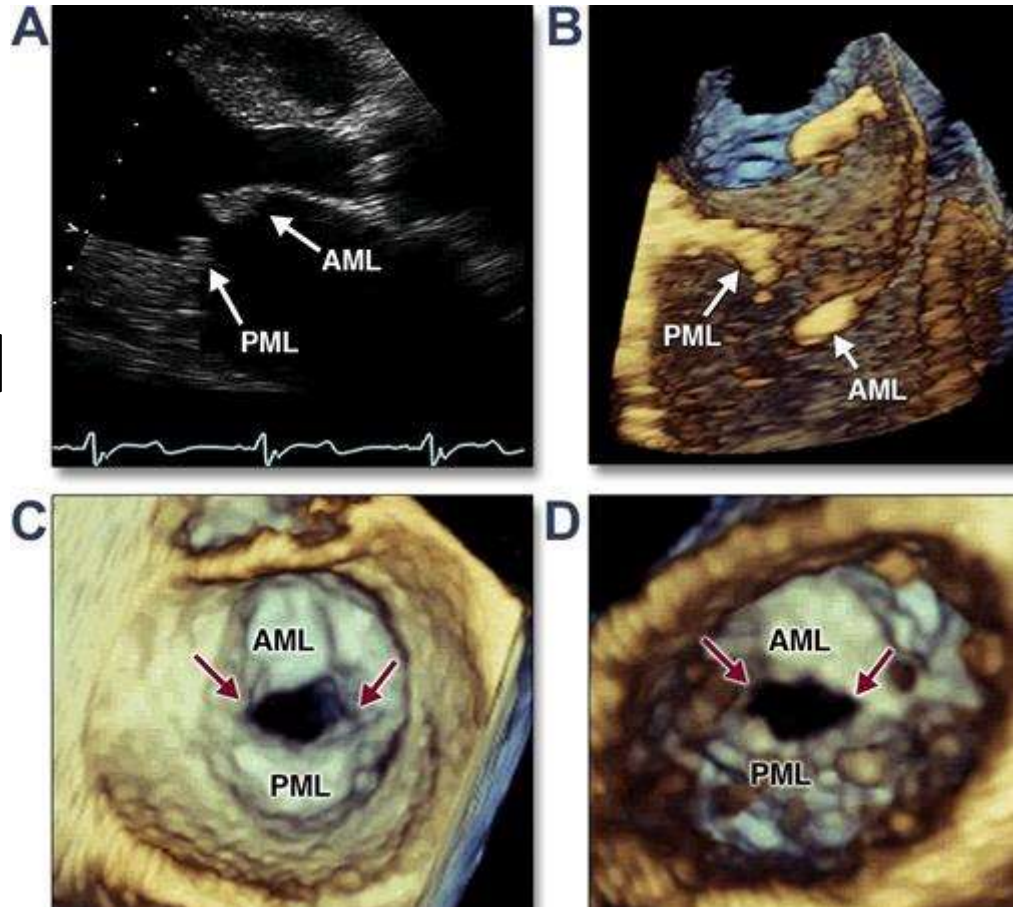
B-mode echocardiography 3a

(A) Leaflet thickening at the edges is shown in a parasternal long axis transthoracic view



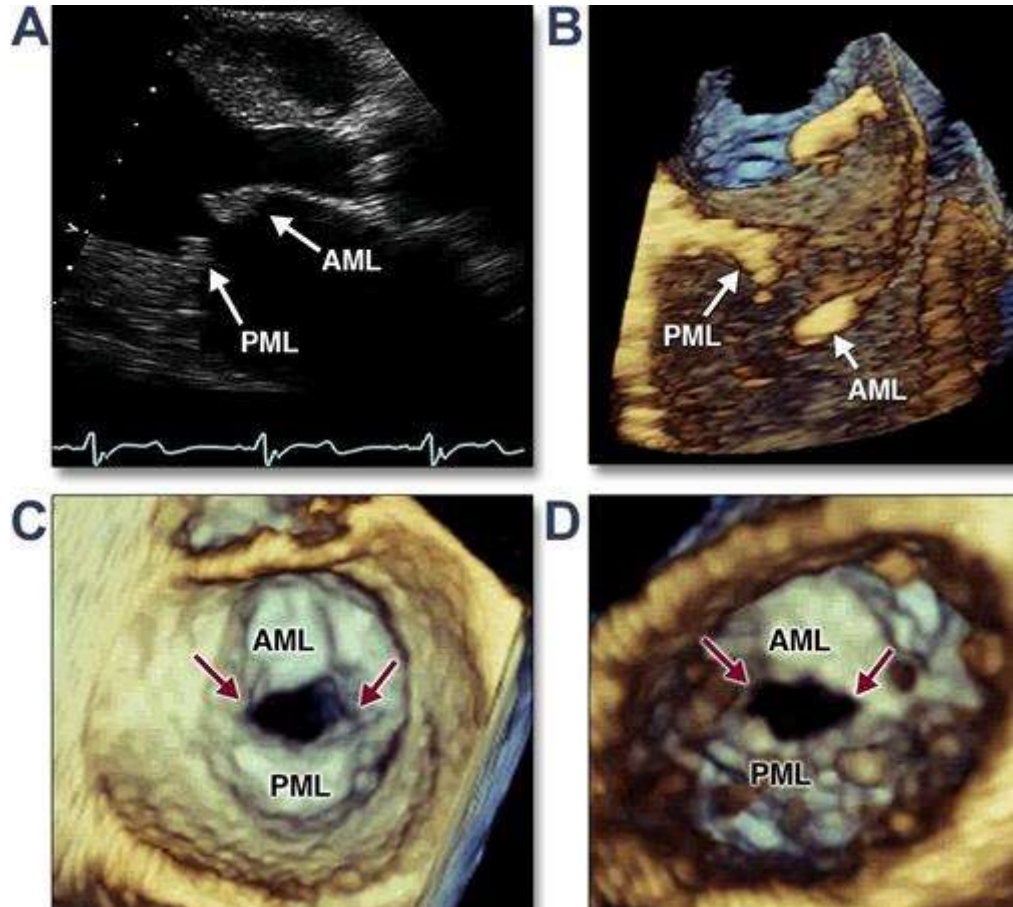
B-mode echocardiography 3b

(B) The immobility of the posterior leaflet and the doming of the anterior leaflet as typical morphological characteristics of rheumatic mitral valve disease are shown in a 3-dimensional transesophageal image

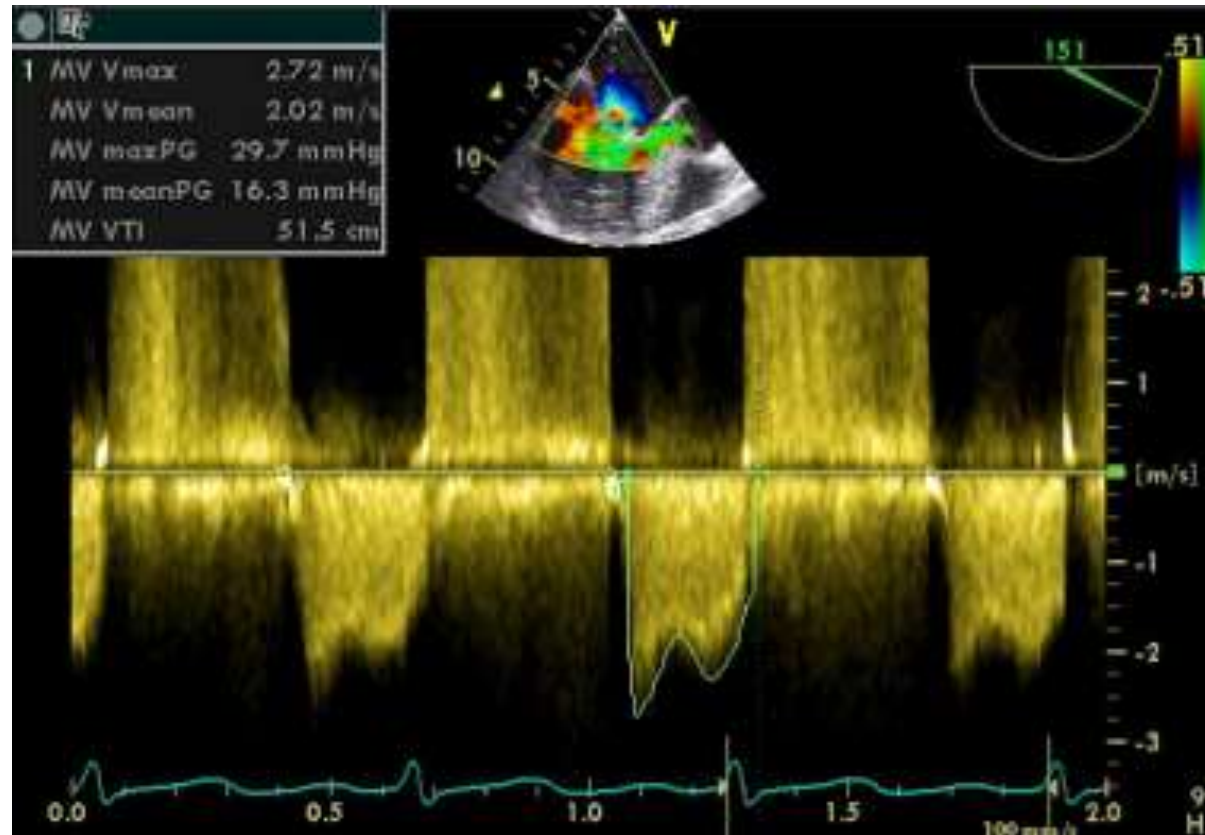


B-mode echocardiography 3c

[C] and (left ventricular aspect [D]) show the fusion of both commissures (**red arrows**). AML = anterior mitral leaflet; PML = posterior mitral leaflet.

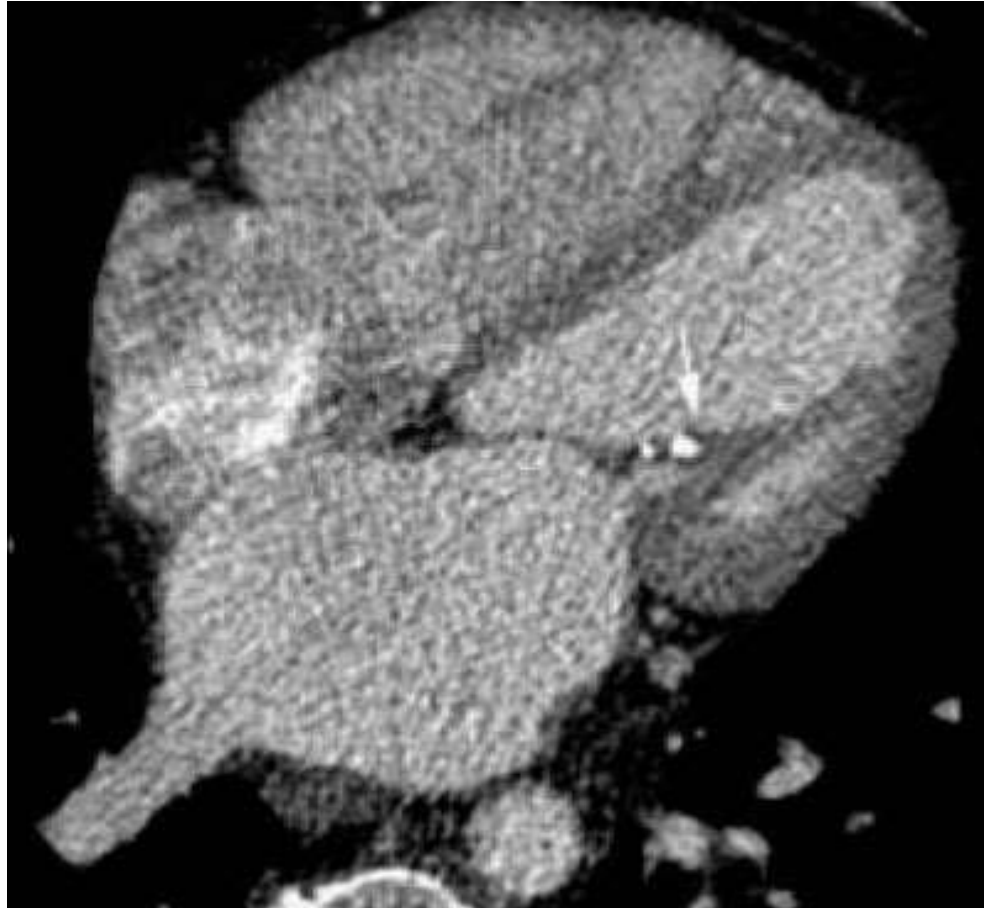


Doppler echocardiography

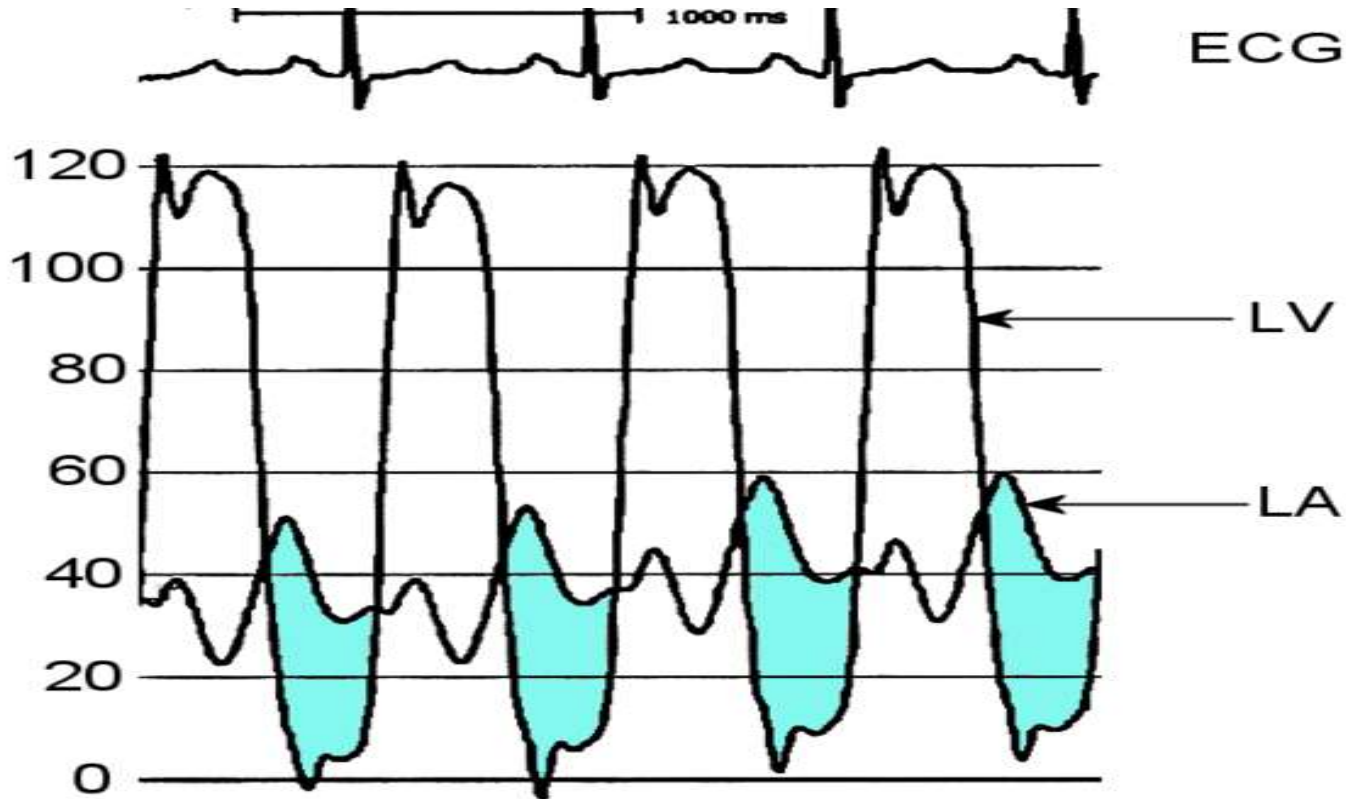


Transesophageal echocardiogram with continuous wave

Computed tomography



Cardiac catheterization



Pressure tracings in the LA and the LV in an individual with severe mitral stenosis. Blue areas represent the diastolic pressure gradient due to the stenotic valve.

Mitral valve
insufficiency/regurgitation

Definition

Mitral insufficiency/regurgitation (MI) or mitral incompetence is a disorder of the heart in which the mitral valve does not close properly when the heart pumps out blood; it is the abnormal leaking of blood backwards from the left ventricle, through the mitral valve, into the left atrium, when the left ventricle contracts, i.e. there is regurgitation of blood back into the left atrium

Types, causes

- Types: acute, chronic
- Causes:
 - the most common cause is mitral valve prolapse (MVP)
 - Other causes: ischemic heart disease, Rheumatic fever, Marfan's syndrome, Ehlers Danlos Syndrome, the dilatation of the left ventricle that causes stretching of the mitral valve annulus and displacement of the papillary muscles (dilated cardiomyopathy, etc.)

Pathophysiology 1

- The pathophysiology of MI can be broken into three phases of the disease process:
 - the acute phase
 - the chronic compensated phase
 - the chronic decompensated phase

Pathophysiology 2

The acute phase:

- Acute MI (as may occur due to the sudden rupture of a chorda tendineae or papillary muscle) causes a sudden volume overload of both the LA and the LV
- The regurgitant volume causes a volume overload and a pressure overload of the LA and the LV.
- The increased pressures in the LA may inhibit drainage of blood from the lungs and lead to pulmonary congestion.

Pathophysiology 3

The chronic compensated phase

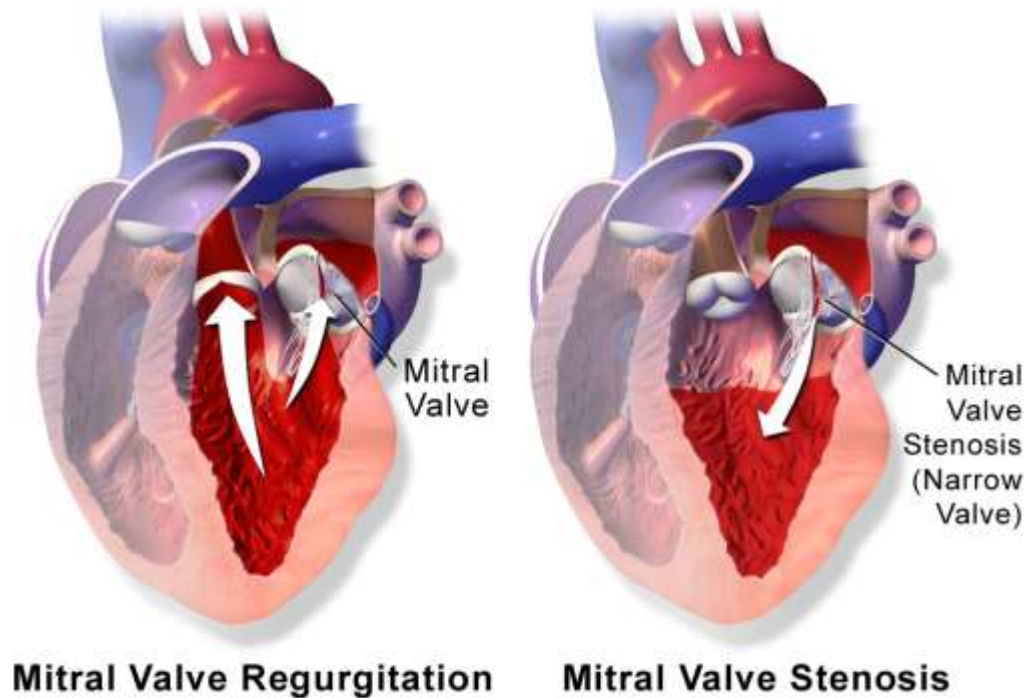
- If the MI develops slowly over months to years or if the acute phase cannot be managed with medical therapy, the individual will enter the chronic compensated phase of the disease
- In this phase, the LV eccentric hypertrophy and the enlargement of the LA improve the drainage from the pulmonary veins, and patients may be asymptomatic and have normal exercise tolerances

Pathophysiology 4

The chronic decompensated phase

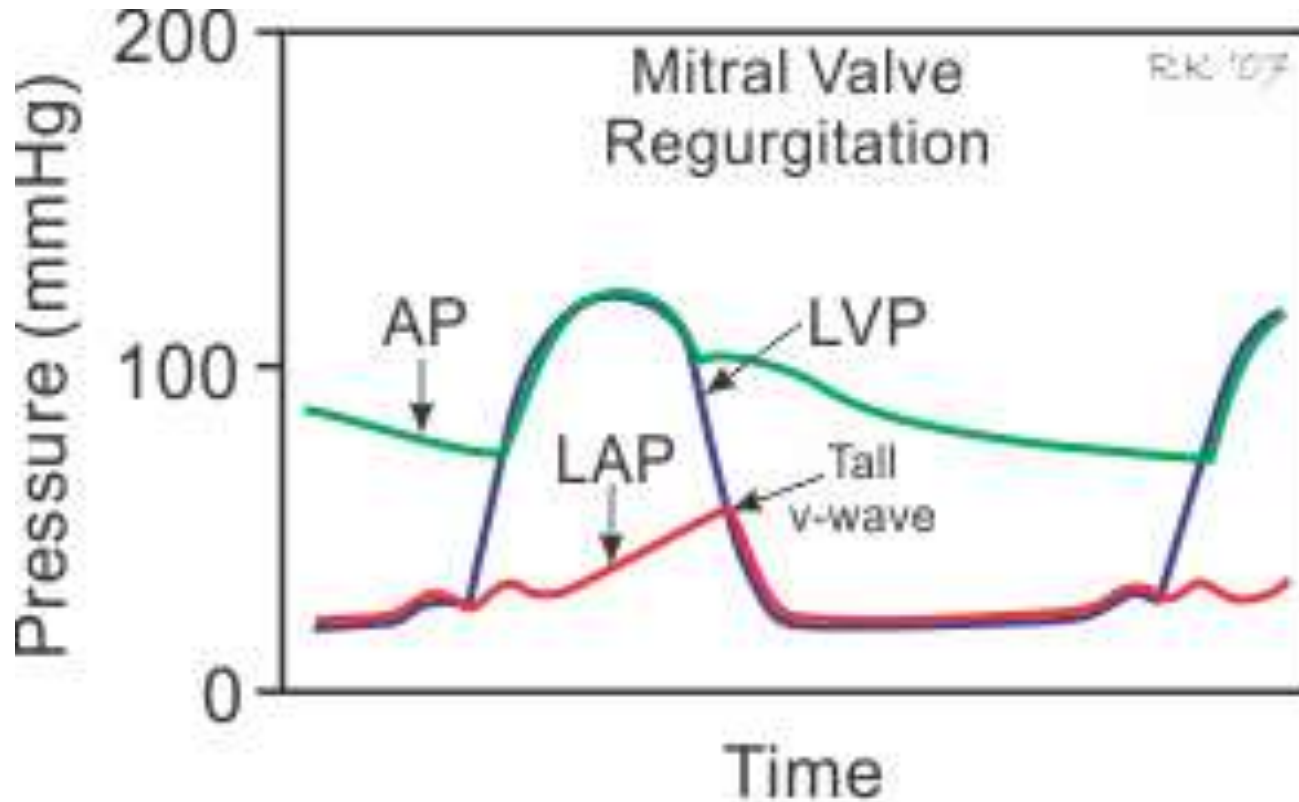
- The ventricular myocardium is no longer able to contract adequately to compensate for the volume overload of MR, and the SV of the LV will decrease with decreased forward cardiac output
- The LV begins to dilate with a dilatation of the mitral valve annulus, which may worsen the degree of MI
- A decreased SV and no other cardiac abnormality should alert the physician that the MI may be in its decompensated phase

Heart changes



Mitral valve insufficiency/regurgitation' and stenosis hearts: find difference

Main hemodynamics disturbances



The changes in aortic pressure (AP), left ventricular pressure (LVP) and left atrial pressure (LAP)

Symptoms

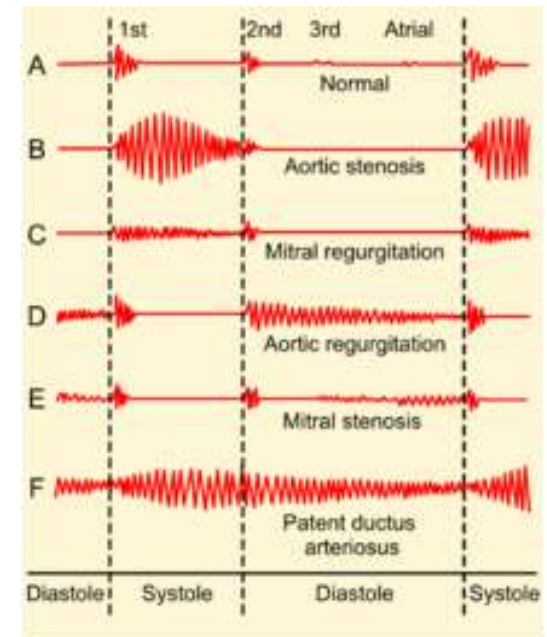
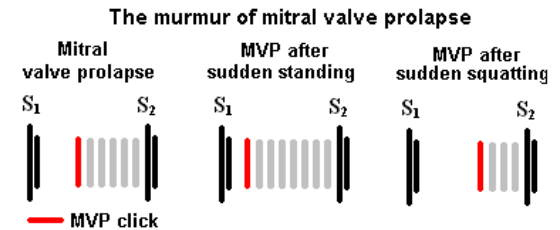
- The larger the LV, the more advanced is the MR
- Symptoms include:
 - Shortness of breath, which may later develop into shortness of breath at rest and at night
 - Fatigue and weakness
 - Edema (fluid buildup in the legs and feet, orthopnea, paroxysmal nocturnal dyspnea)
 - Heart palpitations (atrial fibrillation, which can lead to blood clots forming in the atrium)

Physical examination 1

- Asthenic body habitus
- Low body weight or body mass index (BMI)
- Straight-back syndrome
- Scoliosis or kyphosis
- Pectus excavatum
- Hypermobility of the joints (e.g., Marfan syndrome)

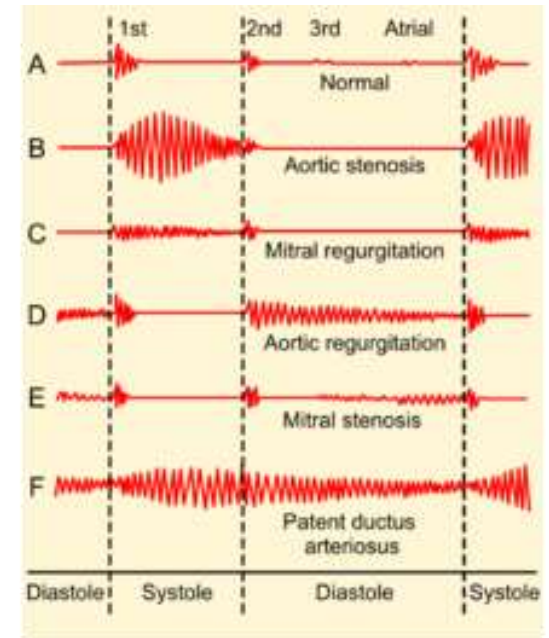
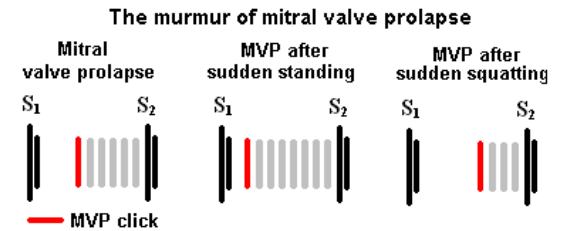
Physical examination 2

The 1st heart sound (S_1) may be soft (or occasionally loud); a 3rd heart sound (S_3) at the apex reflects a dilated LV and important MR; an S_3 that accompanies mitral regurgitation suggests a dilated left ventricle and progression to heart failure



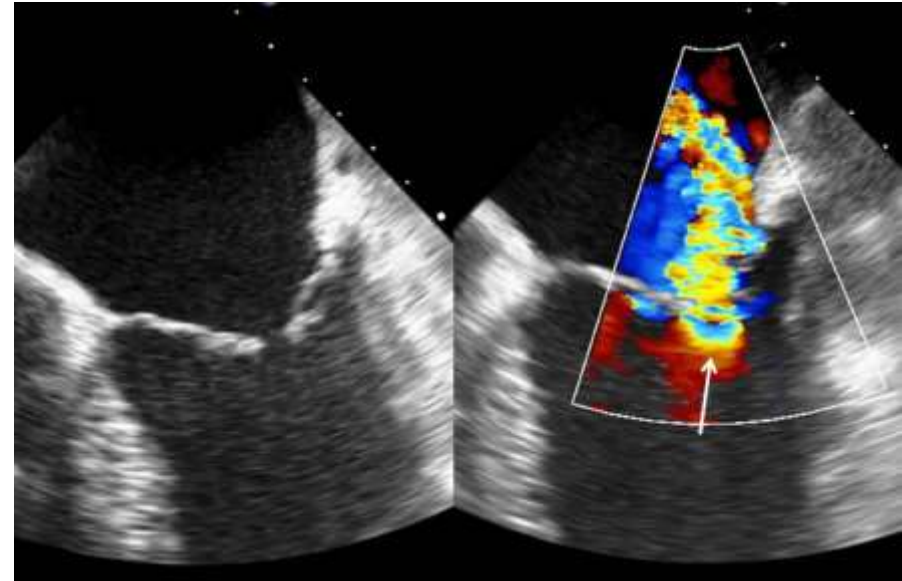
Physical examination 3

The cardinal sign of MR is a holosystolic (pansystolic) murmur, heard best at the apex with the diaphragm of the stethoscope when the patient is in the left lateral decubitus position

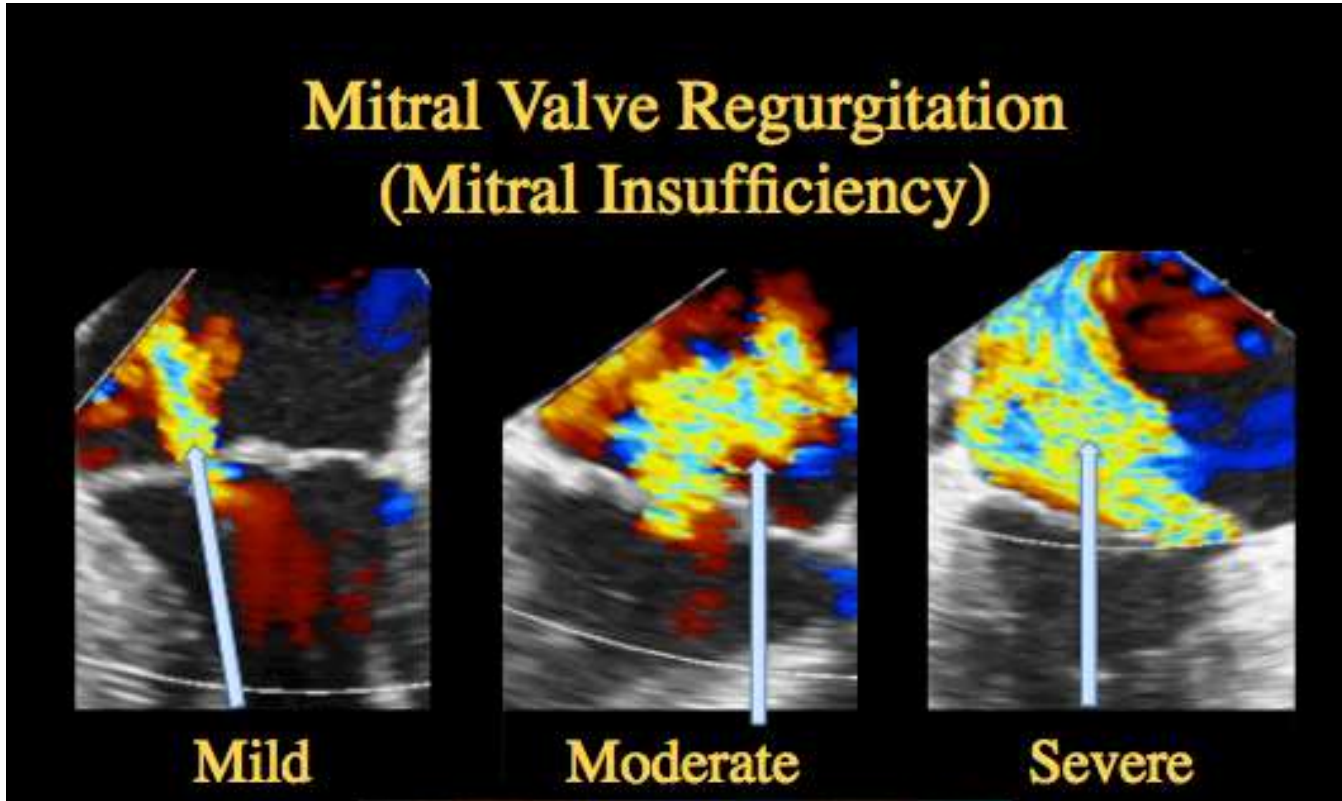


Diagnostic tests

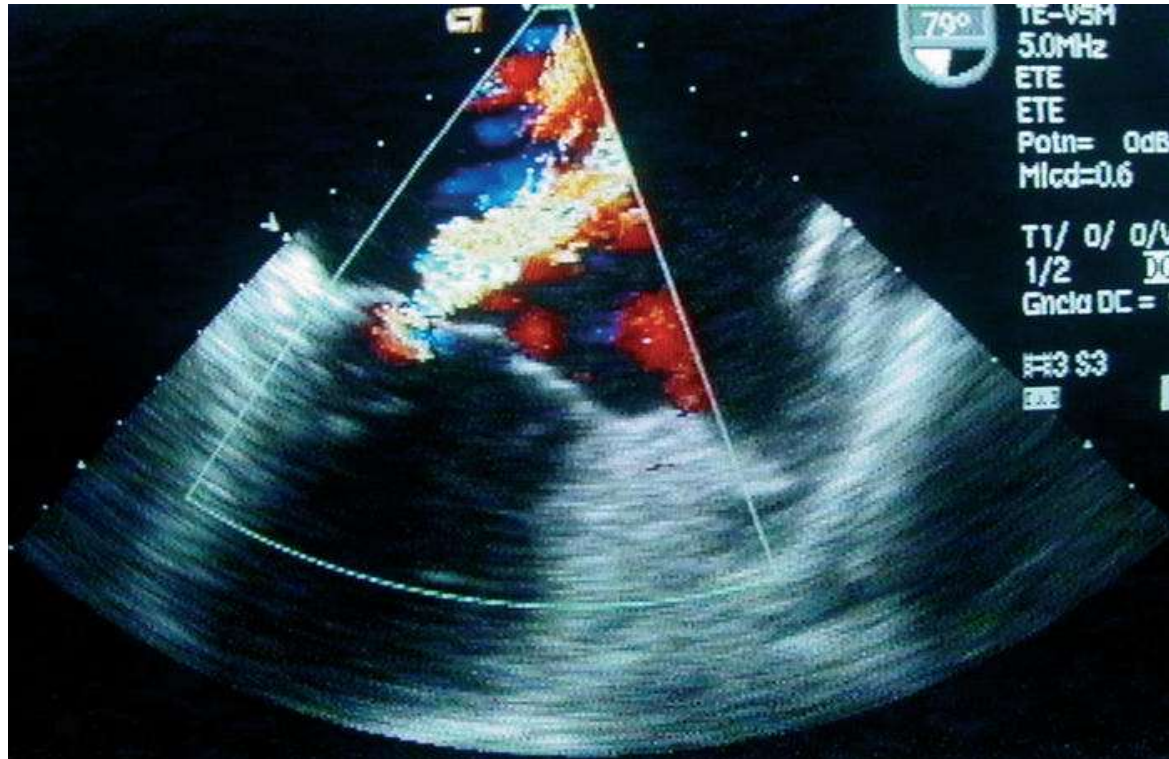
- Echocardiography
- Quantification of mitral insufficiency
- Cardiac magnetic resonance imaging
- Electrocardiography (ECG)
- Chest X-ray
- Cardiac catheterization



Echocardiography 1

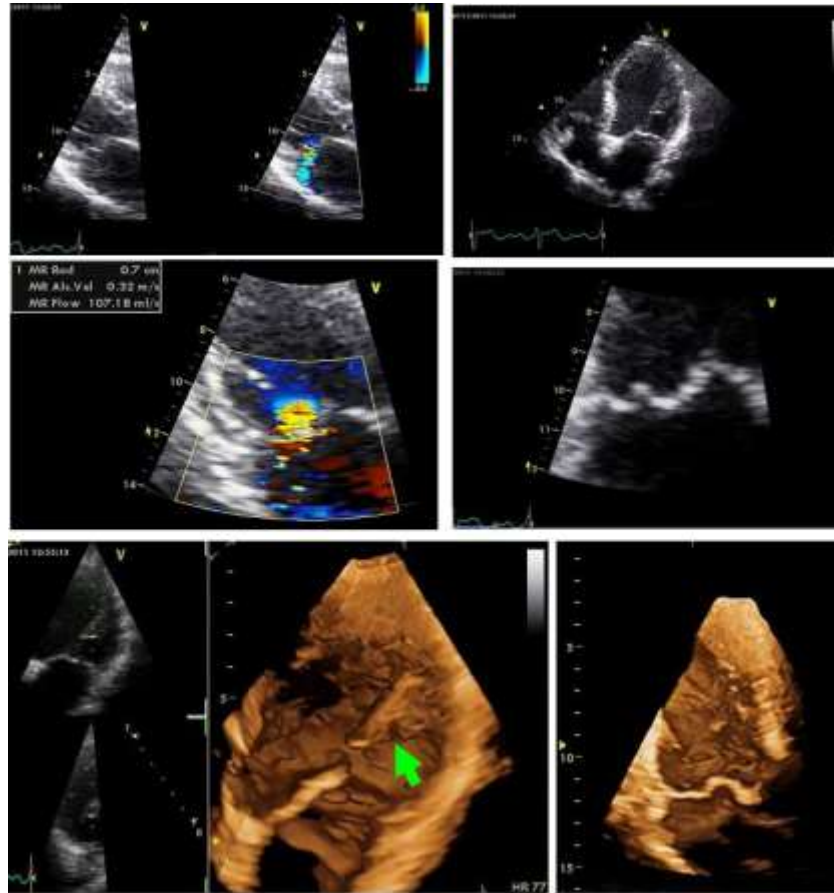


Echocardiography 2



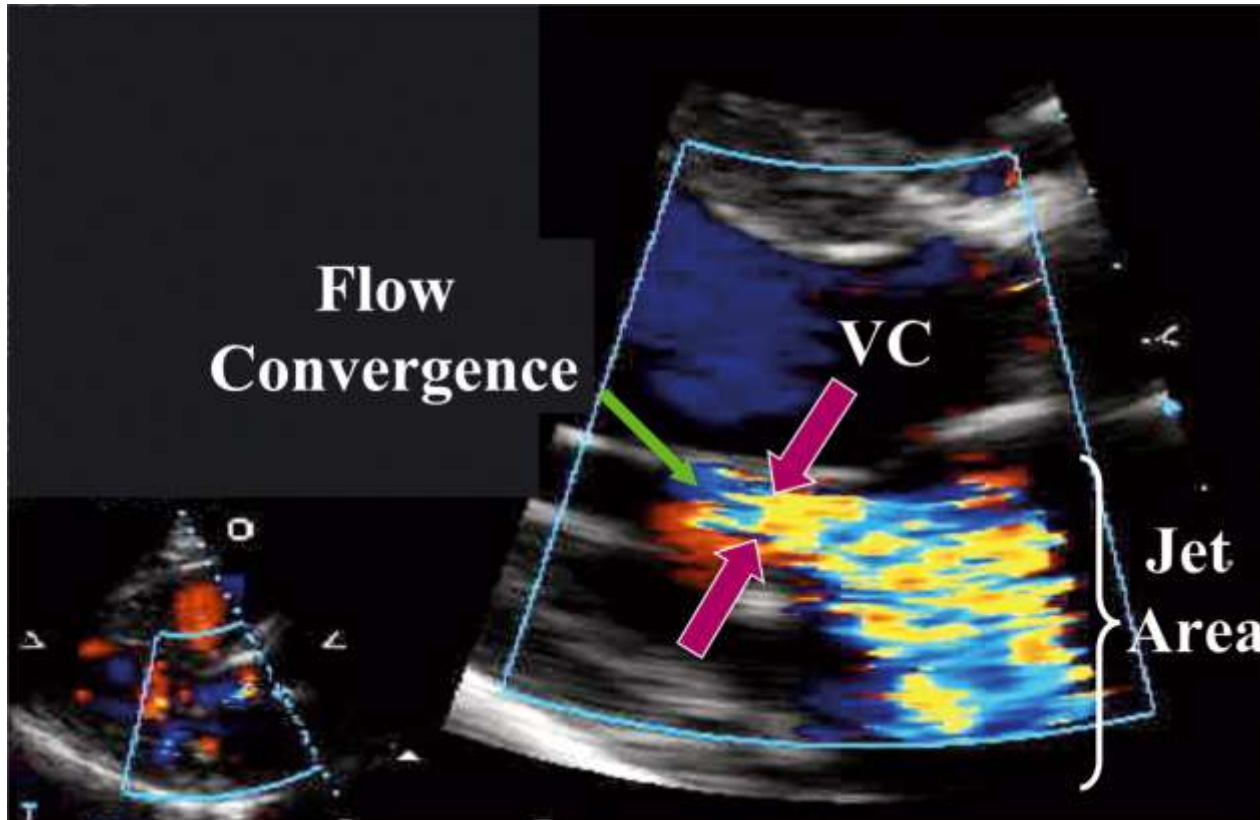
Transesophageal echocardiogram: apical 4-chamber projection where small vegetations can be observed on both mitral valves (arrows); severe MR

Echocardiography 3



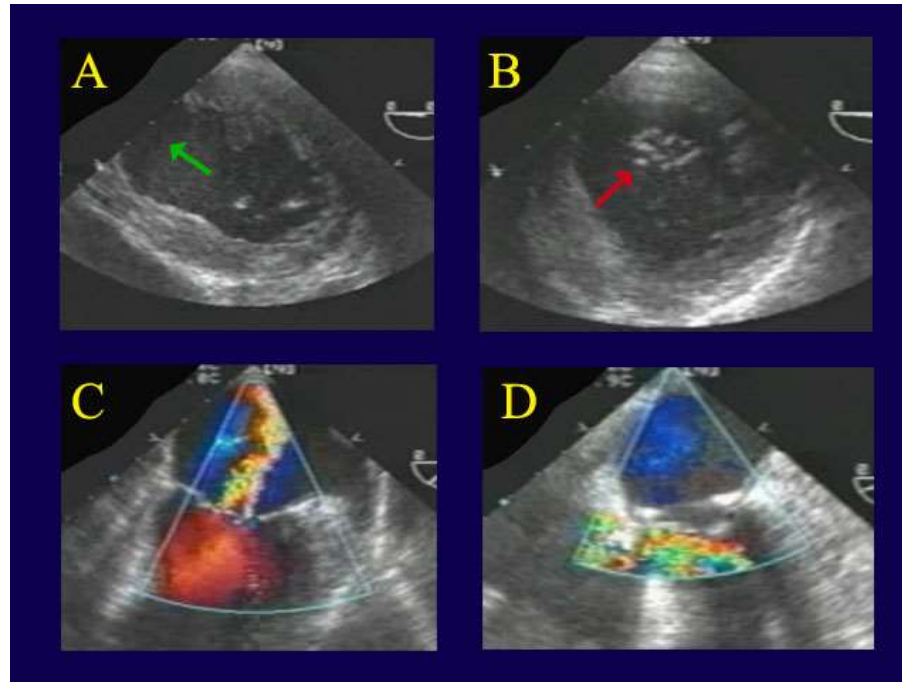
Ischemic mitral regurgitation due to acute elongation of papillary muscle, appearing as mitral valve prolapse

Echocardiography 4



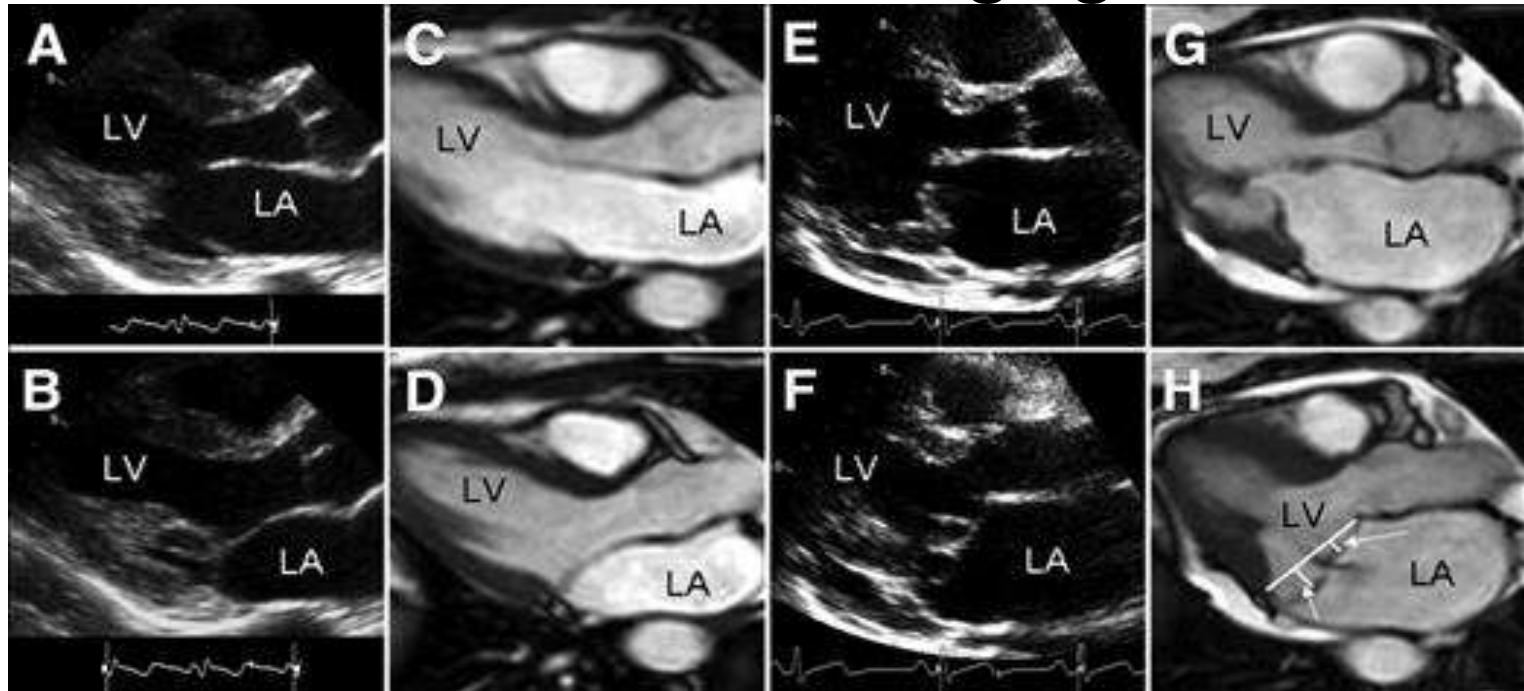
Color flow recording of a MR jet: flow convergence, vena contracta (VC), and jet area in the left atrium.

Echocardiography 5



Panel A showing an expanded posterior-lateral wall infarct (green arrow). Panel B shows a gap in mitral coaptation. (Panel C), but the leaflets are at the annular plane. In panel D, the valve is entirely competent after full ring annuloplasty alone.

Echocardiography combine with cardiac magnetic resonance imaging



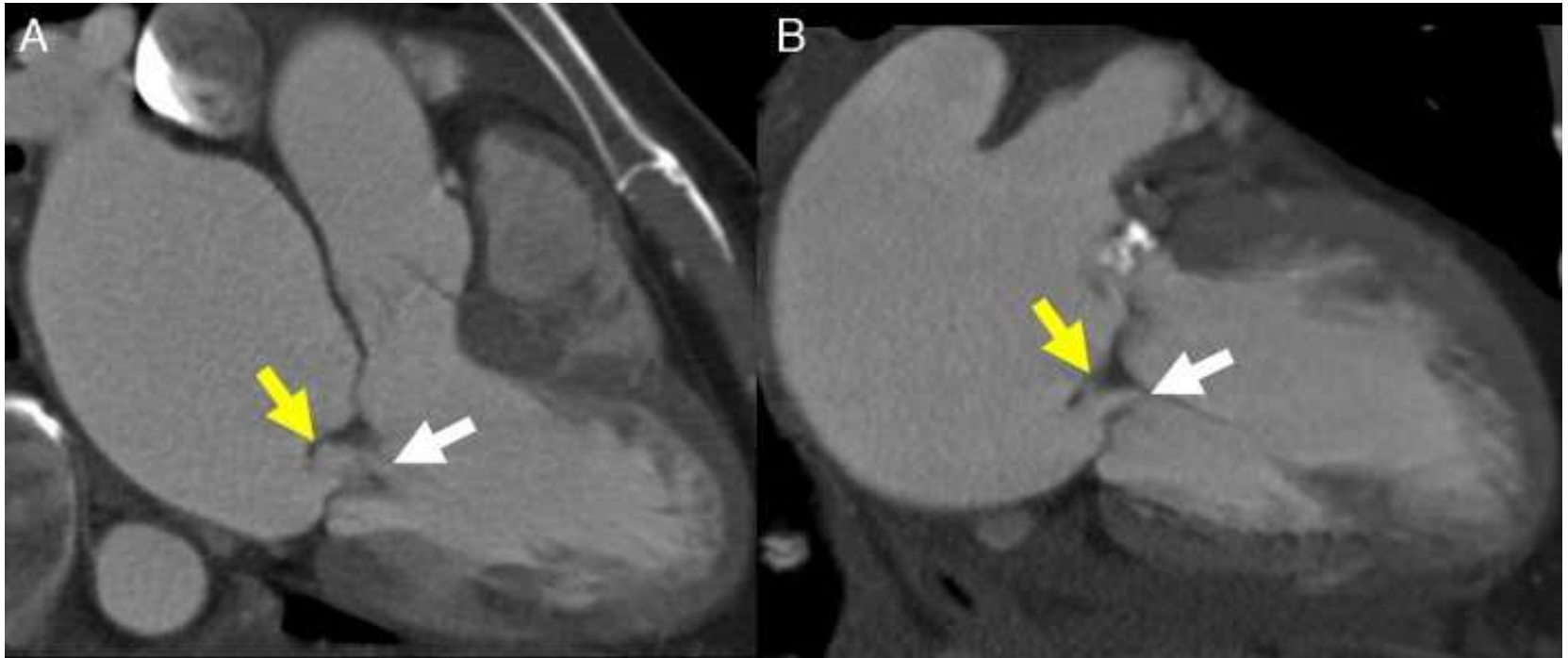
Control (A to D) and mitral valve prolapse (MVP) (E to H) patients. The TTE parasternal long-axis views (A, B, E, F) and CMR LV outflow tract views (C, D, G, H). (A, C, E, G) Diastole; (B, D, F, H) systole

Quantification of mitral insufficiency

Degree of mitral regurgitation	Regurgitant fraction	Regurgitant Orifice area
Mild	< 20 percent	
Moderate	20 - 40 percent	
Moderate to severe	40 - 60 percent	
Severe	> 60 percent	> 0.4 cm ²

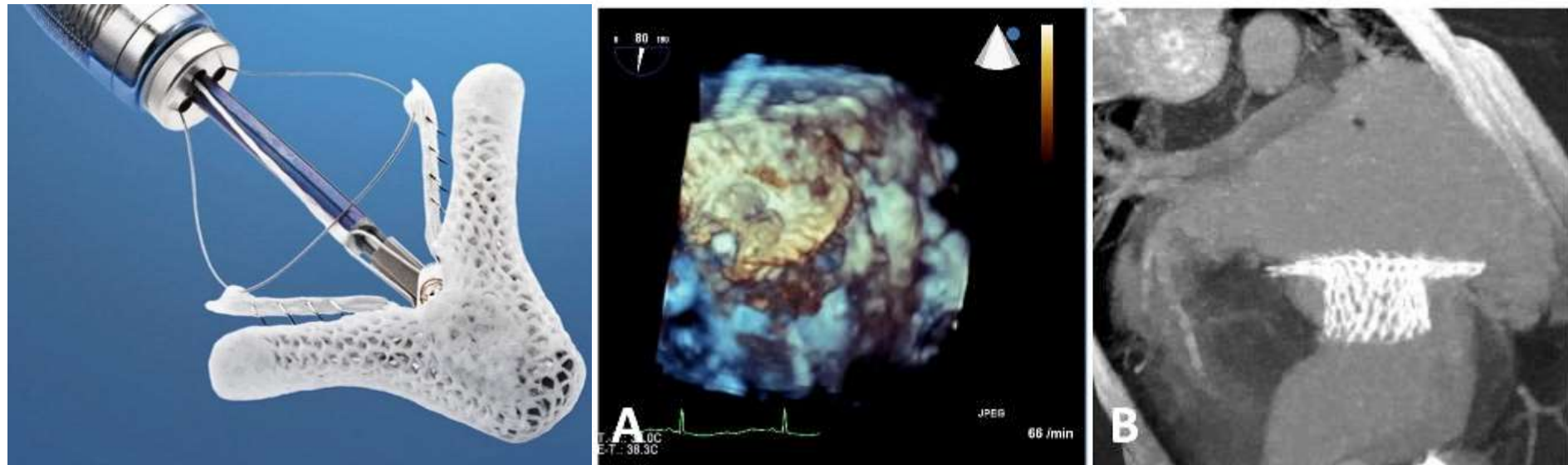
The degree of severity of MI can be quantified by the regurgitant fraction, which is the percentage of the left ventricular stroke volume that regurgitates into the left atrium

Cardiac magnetic resonance imaging



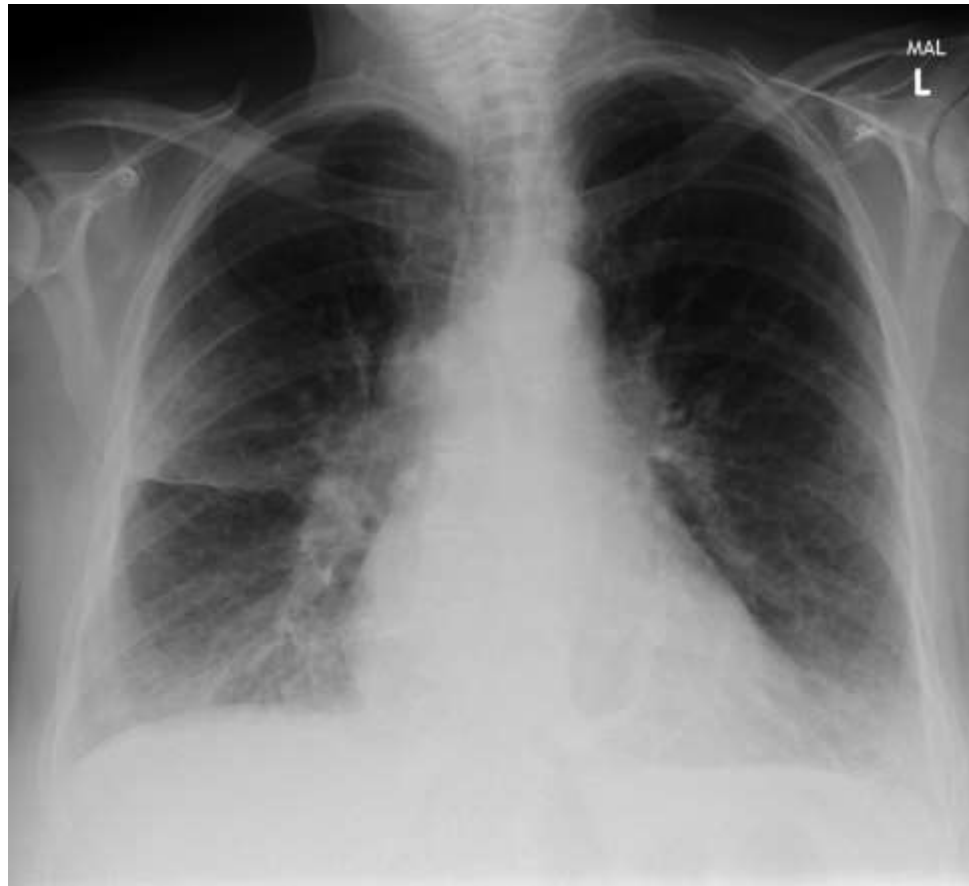
Mitral insufficiency in three-chamber (A) and two-chamber (B) views in diastole show a closure defect of the mitral valve (white arrow)

Computer tomography



3D transesophageal echocardiographic image showing the valved stent after successful deployment in the native mitral annulus during systole

Mitral valve insufficiency/regurgitation: chest X-ray



Unilateral pulmonary oedema in severe mitral regurgitation from posterior leaflet prolapse.

Cardiac catheterisation

