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Essentials of Internal Medicine



4th Edition

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Chapter 4

Valvular Heart Disease

MITRAL STENOSIS

Mitral stenosis (MS) is the most common complication of acute rheumatic fever. Female : Male ratio 2:1.

- **Mitral valve apparatus is composed of**
  - Valve leaflets.
  - Valve annulus.
  - Chordae tendinae.
  - Papillary muscles.
  - Myocardium to which the papillary muscles are attached.

All components of mitral valve may be involved in rheumatic process.

ETIOLOGY OF MITRAL STENOSIS

Major Causes

- **Rheumatic fever** (predominant cause 98%)
  40% of all RHD have pure MS
  40% of all RHD have MS with MR
  Rest are associated with other valvular abnormalities.
- **Congenital** (mitral valve stenosis and cor triatriatum).
- **Rare causes**
  - Senile calcification of valve annulus.
  - SLE.
  - Rheumatoid arthritis.
  - Infective endocarditis—Ball-valve thrombus.
  - Associated with ASD in **Lutembacher syndrome**.
  - Malignant carcinoid.
  - Mucopolysaccharidosis of Hunter-Hurler phenotype.
  - Amyloid, Fabry disease and Whipple's disease.
  - Methyserzide therapy.
  - Congenital diaphargm.
  - Cor triatriatum.
  - Left atrial myxoma.

PATHOLOGY OF MITRAL STENOSIS

Fibrosis resulting from acute rheumatic fever causes fusion of mitral valve apparatus over 2–12 years time:

- **Commissural fusion** (30%)
- **Cuspal fusion** (15%)
- **Chordal fusion** (10%)
- **Rest are combined**.
  - *Commissural*—Valve cusp fuse at their edges.
  - *Cuspal*—Fibrous obliteration, revascularization, thickening and rigidity of cusp.
  - *Chordal*—Fusion, thickening and shortening of the structures.

All these changes lead to typical funnel-shaped appearance. Shaped like fish mouth with calcium deposition over the leaflets and ring.

Progressive thickening, fibrosis, and calcification may be due to—

- Smouldering rheumatic activity or
- Constant trauma by turbulant flow initiated by deformed valve leads to progressive thickening fibrosis and calcification of valve apparatus.

PATHOPHYSIOLOGIC COURSE OF MITRAL STENOSIS

Latent period is at least a decade or more from acute rheumatic fever to develop severe MS. Symptoms commonly commence at 3rd and 4th decade.

In tropics and underdeveloped area, disease progresses very rapidly and present in early adolescent.

Once a patient of MS become symptomatic seriously the disease progresses continuously to death within 2–5 years.

GRADING OF MITRAL STENOSIS

Normal cross-sectional area of valve orifice 4–6 cm^2

- **Mild MS** have valve orifice = <2 cm^2
- **Moderately severe MS** have valve orifice ≅ 1.0–1.5 cm^2
- **Critical MS** valve orifice – <1 cm^2.

PATHOPHYSIOLOGY OF MITRAL STENOSIS

- In case of mild MS, the blood can flow from LA to LV only if propelled by abnormally elevated LA to LV pressure gradient (hemodynamic hallmark of MS).
- In critical MS, the LA pressure is approximately 25 mmHg to maintain normal COP (cardiac output).
- Elevated LA pressure leads to increased pulmonary venous pressure resulting in increased pulmonary

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capillary wedge pressure which ultimately results in decreased compliance of lung and exertional dyspnea.

Combination of mitral valve disease and atrial inflammation secondary to RHD leads to:

- LA dilation.
- Fibrosis of LA wall.
- Disorganization of LA muscle bundle.
- Entry of atrial muscle sleeve into the opening of pulmonary vein.

All these factors lead to:

- Disparity in conduction velocity.
- In homogenous refractory period which ultimately leads to **atrial fibrillation** resulting either from ectopic focus or from reentry.

CAUSES OF PULMONARY HYPERTENSION IN MITRAL STENOSIS

- Passive backward transmisston of LA pressure.
- Reflex pulmonary arterial constriction (reactive pulmonary hypertension).
- Interstitial edema surrounding pulmonary vascular bed.
- Organic obliterative changes in pulmonary vasculature. Severe pulmonary hypertension results in pulmonic regurgitation, RV enlargement secondary TR and right heart failure.

CLINICAL FEATURES OF MITRAL STENOSIS

History

Most patients remain asymptomatic when valvular obstruction remains mild or in case of moderate obstruction with slight increase of LA–LV pressure gradient.

Principal Symptoms

- Dyspnea—
  - It is due to reduced lung compliance.
  - Patients with critical obstruction to left atrial emptying are NYHA class-III dyspneic and generally have orthopnea and at risk of developing Frank pulmonary edema precipitated by any condition which will increase the flow across the AV valve (exercise, emotional stress, respiratory infection, atrial fibrillation, tachyarrhythmia, pregnancy).
- **Cough and wheeze** —It accompanies dyspnea.
- **Hemoptysis**—It is due to
  - *Rupture of pulmonary* **bronchial venous connection** secondary to pulmonary venous hypertension.
  - *Blood-stained sputum* complicating chronic bronchitis, bronchopneumonia, and lobar pneumonia.
  - *Pinkish frothy sputum* characteristic of pulmonary edema due to rupture of alveolar capillary when left atrial failure develop.
  - *Pulmonary infarction* from pulmonary embolism is a late complication of MS associated with leg veins thrombosis.
  - *Anticoagulation overdose.*
- **Chest pain**—
  - 15% of MS patients have chest pain indistinguishable from angina.
  - Causes of chest pain—
    - Right ventricular hypertension
    - Coincidental coronary atherosclerosis
    - Coronary arterial embolization.
- **Thromboembolism**—Thrombus formation occurs in left atrial appendage
  - 10–20% of MS patients suffer from systemic thromboembolism with atrial fibrillation.
  - 10–15% of thromboembolism patients die and responsible for 25% of all fatalities in MS.

  Precipitating factor of thromboembolisms
  - Atrial fibrillation
  - Decrease COP
  - Increase age
  - Underlying infective endocarditis.

  Site of embolism—
  - Cerebral.
  - Coronary.
  - Sudden death may be caused by obstruction of mitral orifice outlet by large pedunculated thrombus.
- **Infective endocarditis**—Rare in isolated MS.
  - More common in milder form of MS and MR than in severe MS.
- **Other symptoms**
  - *Compression of recurrent laryngeal nerve* causing hoarseness of voice by dialated LA, pulmonary artery and tracheobroncheal lymph node known as Ortner's syndrome.
  - *Features of RVF—*
    - Pulsatile neck veins
    - Hepatomegaly
    - Edema
    - Ascites
    - Hydrothorax (in extreme case).

Signs

- **Mitral facies**
  - Pinkish pimple patches on the cheeks.
  - Differential diagnosis—Cushing, thyrotoxicosis, SLE and alcoholism.
- **Arterial pulse**
  - Low/normal volume (low volume pulse indicates severe MS) may be irregular due to associated atrial fibrillation.
- **JVP**
  - Prominent a-wave in patient with sinus rhythm.

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- In atrial fibrilation → a wave disappears only one crest, i.e. prominent V or CV pattern when associated with TR.

- **Apex**—
  - Inconspicuous LV.
  - Tapping S_1 suggestive of still pliable anterior leaflets.
  - Apex may be of RV type in severe RVH.
  - Diastolic thrill at the apex (better palpable with the patient in left lateral recumbant posture).
- **Palpable P_2/diastolic shock over left 2nd ICS**.
- **RV lift on left parasternal region** also suggestive of RVH/left atrial enlargement.
- **Auscultation**—
  - *S_1*—Generally short, sharp, accentuated due to fibrosis of cusp and incomplete filling of LV.
  - *Loud P_2* — Loud P_2 with closely split S_2 due to reduced compliance of pulmonary vascular bed which shortens the hangout time interval of pulmonary valve.

  Finally S_2 becomes single and accentuated.
  - *Other signs of pulmonary hypertension*—
    - Nonvalvular pulmonary ejection sound (click)
    - Systolic murmur of TR
    - Graham Steell murmur of PR
    - S4 originating from RV.
  - *Diastolic murmur*—
    - ***It is a low pitch middiastolic rumbling murmur with a presystolic accentuation, commensing immediately after OS. Best heard at the apex with the bell of stethoscope, patient in left lateral position and the breath held at the end of expiration provided the patient is in sinus rhythm.*** If atrial fibrillation is present with MS, the auscultatory finding of MS is ***middiastolic rumbling murmur with variable intensity 1st heart sound. Presystolic accentuation of the murmur will be absent***.
    - Murmur is accentuated by mild exercise or in any condition which increases the transmitral valve pressure gradient.
    - ***The duration, and not the intensity of the murmur is a guide to the severity of MS***.
    - In mild MS middiastolic and late diastolic/ presystolic component of murmur are not continuous. There may be a gap in between mid and late diastolic murmur.
    - In severe MS, the murmur is hollow diastolic.
  - *Opening snap (OS)*
    - Mechanism of production of OS—It is due to sudden tensing of mitral valve leaflets after the valve cusp has completes its opening excursion. During downward depression of mitral cusp from upward position to downward position at the beginning of ventricular diastole.
    - Most readily audible at expiration at or medial to cardiac apex following A_2 with the diaphragm of the stethoscope.
  - **OS is indicative of**
    - MS is organic
    - Significant MS.
  - **Loud OS signifies**
    - High LA–LV pressure gradient.
    - Valve cusp are still pliable and not calcified.
    - Severe AR, MR, SABE, AF and atrial failure are absent.
    - MS is readily amenable to surgery.
  - **Differential diagnosis of middiastolic murmur of MS**
    - Carey Coombs murmur of acute rheumatic fever.
    - Austin Flint murmur of AR.
    - Flow murmur of severe MR, VSD and PDA.
    - Left atrial myxema.
    - Diastolic flow murmur through tricuspid valve in ASD.

Severity of MS is indicated by
a. Short A_2–OS gap
b. Long duration of delayed diastolic murmur

  - Dynamic auscultation—Sudden standing and resultant reduction of venous return lower LA pressure and widen the A_2–OS interval. This is useful in distinguishing an A_2–OS combination from a split S_2 which narrows on standing.

Investigations

- **ECG**—Changes in MS are
  - **Evidence of LA enlargement in lead-II, as wide notched P-wave (P. mitrale)**.
  - In lead-V_1 → **Biphasic** P-wave with >1 small square duration and deapth.
  - Features of RVH in V_1.
- **X-ray**—Evidence of MS.
  - **LA enlargement** as a double contour at the right border of heart (specially if associated with MR) and prominent LA appandages at the left border.
  - **Enlargement of pulmonary artery** appears as fullness of plumonary bay at the left border in PA view.
  - **LA enlargement** is also evident by C-shaped indentation over barium swallow esophagus in left lateral view.
  - **RVH** is demonstrated by
    - Apex is much above the diaphragm.
    - Diminution of retrosternal space in left lateral view with slight increase in transverse diameter of heart which is boot-shaped due to upturn apex.

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Peasant's wooden boot with upturn toes—Coeren-saboot appearance of cardiac silhouette.
- **Karley's B-line**
  - Dense short horizontal line commensing from periphery at the costophrenic angle due to thickened intralobular septa.
- **Karley's A line—**Straight dense line about 4 cm in length running towards the hilum from periphery (in severe long-standing mitral stenosis).
- Evidence of **pulmonary hemosiderosis**.
- Evidence of **pulmonary edema (rarely)**.
- Bat's wing-shaped opacity spreading from hilum.
- Evidence of **parenchymal calcification**.
- Evidence of **mitral valve calcification** (retrosternal location at the level of 4th ICS).
- Deviation of left main and upper lobe bronchus.
- Prominent upper lobe pulmonary vein.
- Hilar arterial prominence.
- Peripheral prooning of blood vessel.

- **Echocardiography**
  - *M-Mode echo shows*
    - Thickened, calcified and stenotic mitral valve
    - LA enlargement
    - Restricted valve casp movement.
  - *2D echo shows*
    - LA thrombus.
    - Restricted motion and doming of valve leaflets.
    - Valve orifice size measurement.
    - Detection of valve—calcification and thickening of subvalvular apparatus with fusion and retraction of chordae.
    - LV contractility.
  - *TEE* gives superior image of mitral valve and LA thrombus.
  - *Doppler* study shows—Functional status of LA, LV, RV can be assessed and quantification of the severity of MS.
  - Estimate pulmonary artery wedge pressure.
  - Assessment of any other valve anomaly.

CARDIAC CATHETER

To rule out CAD in patient with chest pain.

Management

Medical therapy

1. **Penicillin prophylaxis** for acute rheumatic fever and infective endocarditis.
2. **Restriction of fluid and Na^+ intake**.
3. **Oral loop diuretics** (frusemide with potassium-sparing diuretic).
4. **Digitalis in patient with AF**—To slow the ventricular rate and also in patient with right ventricular failure. β-blockers or non-DHP CCB can also be used.
5. **Warfarin** for 1 year in patient with systemic/pulmonary embolization.
6. **Medical/electrical cardioversion** of AF should be attempted if AF is of recent origin and LA is not husely dialated.

Surgical management

Indications of surgery in MS

1. **Symptomatic patient** with moderate to severe MS (valve size < 1.0 cm^2/m^2 body surface or 1.5 cm^2).
2. **Mild MS** *but symptomatic* with ordinary activity.
3. **Complication**—Development of embolism.
4. **Patient's symptom cannot be controlled** only by medical therapy.
5. **Pregnancy with MS** whose previous pregnancy was symptomatic.
6. **PHTN**—Pulmonary arterial SBP >60 mm after exercise and >50 mm at rest.
   Mean pulmonary capillary wedge presure >25 mm. **Valvotomy is not recommended for entirely asymptomatic patient or when mitral valve orifice is >1.5 cm^2**.

Operative procedure

- **Mitral valvotomy**—Unless contraindicated should be done in all symptomatic patients with isolated MS of orifice size is <1.5 cm^2.
  Two techniques
  - **Percutaneous mitral balloon valvotomy (PMBV)**—Short and long-term results are similar to those of surgical valvotomy.
  - **Surgical valvotomy**—Opening of valve commissure, loosening of any subvalvular fusion of papillary muscles and chordae with removal of deposition of calcium and left atrial thrombus.
    Mortality is about 2%.
    About half of the patients after surgical mitrals valvotomy require reoperation by 10 years.
- **Mitral valve replacement (MVR)**—Is required
  - In MS (orifice <1 cm^2).
  - NYHA class-III symptomatic despite optimal medical therapy.
  - When MS is associated with significant MR or whose valve in severly disorganized by PMBV or prior operative manipulation should have valve replacement.
    Operative mortality is 6% and 10 years survival is about 70%.

AORTIC REGURGITATION

AORTIC REGURGITATION CAUSED BY

1. Primary aortic valve disease
2. Aortic root dilation.

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Causes of Aortic Valve Disease

- Rheumatic heart disease.
  - Puckering of valve cusp due to infiltration of cusp by fibrous tissue.
  - Commissural fusion prevent proper apposition.
- Calcific AS in elderly is the most common cause of AS with AR.
- Infective endocarditis
  - Destruction or perforation of valve leaflet.
  - Vegetation interfering with proper apposition of cusp.
- Iatrogenic
  - Catheterization.
  - Percutaneous aortic balloon valvotomy.
  - Radiofrequency catheter ablation.
- Bicuspid aortic valve.
- Large VSD.
- Membraneous subaortic stenosis.
- Myxomatous degeneration of aortic valve.
- Structural deterioration of bioprosthetic valve.

Causes of Aortic Root Disease

- Degenerative aortic dilation (age-related)
- Ankylosing spondylitis, RA, SLE, Crohn's disease
- Psoriatic and other seronegative spondyloarthritis
- Systemic HTN
- Dissecting aortic aneurysm
- Cystic medial necrosis (Marfan)
- Aortic dilation secondary to bicuspid valve
- Osteogenesis imperfecta
- Syphilitic aortitis
- Appetite-suppressant drug.

PATHOPHYSIOLOGY OF CHRONIC AORTIC REGURGITATION

- In chronic AR, blood is ejected into comparatively high pressure aorta which results in left ventricular hypertrophy along with dilation due to increase EDV.
- In compensated AR there is sufficient wall thickening so that the ratio of wall thickness : cavity radius remains normal.
- In long-standing chronic AR with increasing severity over time wall thickening fails to keep peace with the hemodynamic load and the end systolic wall stress rises. At this point after load mismatches results in declining of systolic function and EF falls.
- Left ventricular mass is greatly increased often greater than 1 kg. Severe chronic AR has the largest EDV of all heart diseases.
- Regurgitant flow may exceed 20 L/min in severe cases, so that LV output reaches about 25 L/min.
- LV function—Due to decompensation of LV function and reduction of LV compliance, LV end diastolic pressure rises which leads to rising LA, PCWP, RV and RA pressure which at first occurs during exercise later at rest. It leads to fall in EF causing pulmonary congestion.
- Myocardial ischemia is due to
  - Increase in LV wall tension.
  - Increase in LV mass.
  - Decrease in coronary blood flow in diastole as DBP is less than normal.

Clinical Features

History

Patients remain asymptomatic when patients are in compensated state but LV gradually enlarges.

Most patients develop symptoms in 4th or 5th decade only after considerable cardiomegaly and myocardial dysfunction have developed.

Principal symptoms

- Dyspnea—On exertion.
- Orthopnea.
- PND.
- Palpitation—Usually during exertion and lying down posture.
- Pain chest—Due to pounding of heart against chest wall.

Out of these symptoms, dyspnea develops early and rest are the late features which are more prominent with stress, exertion and tachycardia.

These complaints are present for many years before the symptoms of overt LVF develops.

Past history

- Past history of rheumatic fever, SABE may be there.
- Features of RA, SLE, ankylosing spondylitis may be present.

Peripheral signs of AS

The following peripheral signs are seen in chronic AR due to increased pulse pressure—

- **Lighthouse sign**—Alternate blanching and flushing of forehead.
- **Ladolfi sign**—Change in pupil size with cardiac pulsation.
- **Müller sign**—Pulsation of uvula.
- **De Musset's sign**—Head nodding with carotid pulsation.
- **Corrigan's sign**—Dancing carotid in neck.
- **Suprasternal pulsation**—Visible.
- **Locomotor brachialis**—Visible brachial artery pulsation.
- **Bisferiens pulse**—More commonly palpable in brachial and femoral artery than carotid artery. In this condition

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percussion wave and dicotic wave are separately palpable also known as "double kicking pulse".
- **Wide pulse pressure**—Korotkoff 's sound often persists up to zero even though intraarterial pressure rarely falls below 30 mmHg.
- **Water hammer pulse (high volume collapsing pulse)**—Pulse pressure greater than 60 mm Hg. Diastolic blood pressure usually less than 60 mm Hg.
- **Prominent digital arterial pulsation**.
- **Quincke's sign**—Visible capillary pulsation at finger nail and lip.
- **Rosenbach sign**—Pulsation of liver.
- **Gerhardt sign**—Pulsation of spleen.
- **Traube's sign**—Also known as pistol shot sound—A booming systolic and diastolic sound heard over femoral artery.
- **Duroziez's sign**—Systolic murmur heard over the femoral artery when it is compressed proximally and a diastolic murmur when it is compressed distally.
- **Hill sign**—Popliteal SBP 60 mm or more higher than brachial SBP.

Palpation

- **Apex heaving LV type** (forceful and ill-sustained) appear with features of LV enlargement.
- **Systolic retraction** over left parasternal region.
- **Systolic thrill** at the base of the heart (aortic area) which is also palpable at suprasternal notch or over carotid artery called '**carotid shudder**' (due to increased flow through carotid artery in systole).

Auscultation

- **Soft S_1**.
- **S_2 may be absent or single or exhibit narrow paradoxical splitting**.
- **A_2 is soft or absent in valvular AR but A_2** may be normal or rarely may be accentuated in aortic root disease.
- **P_2 may be obscured** by early diastolic murmur.
- **Systolic ejection sound** (click) may be present due to abrupt distension of aorta by augmented stroke volume.
- **S_3 gallop** reflects increased EDV of left ventricle or may be a feature of LV dysfunction who are candidate for surgical treatment.
- **S_4 may be audible in late stage of AR with severe LV dysfunction**.
- **A high pitch decrescendo, blowing early diastolic murmur starting immediately after A_2, best heard at left 3rd ICS parasternal region (neoaortic area) with radiation towards apex with the diaphragm of stethoscope and the patient in sitting posture leaning forward with the breath held at the end of expiration (when the AR is due to primary valve defect)**.
- **When the AR is due to aortic root dilation the same murmur radiates along the right sternal border**.
- The murmur may be holodiastolic and may have a rough quality in severe AR.
- **When murmur have musical quality (cooing dove murmur)** it indicates eversion or perforation of aortic cusp.
- **The severity of regurgitation correlates better with the duration rather than intensity of the murmur. The greater the severity of the AR, shorter the length of the murmur**. In long-standing severe AR the late diastolic component is abolished *due to equilibration of LV pressure and aortic pressure*.
- **An enjection systolic flow murmur radiating to carotid** (but more high pitched and less rasping than murmur of organic AS) **may be heard associated with a systolic thrill due to increased flow through aortic valve in systole**.
- **A functional mid and late diastolic apical rumble known 'Austin Flint murmur'** may be heard in severe AR in presence of normal mitral valve due to impinging of aortic reflux jet on the aortic leaflet of mitral valve (absence of OS and loudness differentiate it from organic MS).
- **Dynamic auscultation**—Murmur of AR may be accentuated by increasing the afterload as in equating posture, with leaning forward and isometric exercise.

Laboratory Investigations

- **ECG**
  - Left axis deviation.
  - LVH.
  - LV strain pattern—ST depression with upright T or more commonly inverted T with down-slopping ST segment.
  - Q wave in I, AVL, V_3–V_6 with small R-wave (precordial) leads due to LV diastolic overload.
  - ECG is not a accurate predictor of cardiac weight.
- **Chest X-ray**
  - In acute AR—Minimum cardiac enlargement.
  - In chronic AR—Marked cardiac enlargement with downward and outward shifting of apex.
    Apex may be displaced below diaphragm.
    Left lateral view shows encroachment in the posterior mediastinum by LV.
  - Calcification of aortic valve—Specially in com- bined AS with AR.
  - Linear calcification in the wall of aorta — Specially in syphilitic aortitis and degenerative aortic diseases.
  - Dilation of ascending aorta (unfolding of aortic arch)—Specially in aortic root disease.

Echocardiography

- **M-Mode echo identify**
  - Cause of AR
  - Thickening of valve cusp

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  - Presence of congenital abnormality like bicuspid valve
  - Presence of prolapse of the valve cusp
  - Presence of vegetation and dilation of aortic root.
- **2-D echo quantify**
  - LV–EDV
  - LV–ESV
  - Ejection fraction
  - Venticular mass.
- **TEE** (transesophageal echocardiography) gives more detailed information about
  - LV size
  - Regurgitant blood volume
  - Presence of vegetation.
- **High frequency fluttering of anterior leaflet of mitral valve due to impingement of regurgitant blood even in mild AR is an important echo sign** but is not seen in rheumatic involvement of mitral valve as the valve is rigid.
- **Doppler**—Most sensitive and accurate noninvasive technique for detecting mild AR, regurgitant flow, orifice size, progression of regurgitation and its effect on ventricle, ejection fraction and timing of surgical intervention.

Radionucleotide imaging is necessary for

Accurate noninvasive assessment of severity of AR by allowing determination of regurgitant fraction and of the LV/RV stroke volume ratio. Usually radionucleotide imaging is only done when—

- Echo resolution is suboptimal
- Discrepancy between clinical and echo finding.

MRI

- Accurate measurement of the regurgitant volume and regurgitant orifice size.
- Most accurate noninvasive technique for detecting LV mass and LV end diastolic volume.

Management

- **Medical therapy**
  - All patients with AR of any severity should receive prophylaxis for infective endocarditis.
  - Asymptomatic patients with mild to moderate AR and normal or minimal increase in cardiac size require no therapy but should be followed up clinically and by echo every 12–24 months.
  - Patients with mild to moderate AR and patients with severe AR but normal EF and mild ventricular dialation may engage in aerobic exercise.
  - Asymptomatic patients with chronic severe AR and normal LV function should be examined 6 monthly.
  - Patients with severe AR having limitation of cardiac reserve and evidence of decreased LV function should not engage in vigorous exercise.
  - Systemic arterial diastolic hypertension, if present, should be treated (because it increases the regurgitant flow) with nifedipine and ACEI. (β-blocker should be used with great caution).
  - Atrial fibrillation and bradyarrhythmia are poorly tolerated and should be treated if possible.
  - Vasodilator therapy—Chronic AR with significant volume overload and increased EDV should be considered for vasodilator therapy with hydralazine, nifedipine or ACE.
    Chronic medical therapy may be necessary for the patient who refuses surgery or considered inoperable due to serious comorbid condition. Chronic medical therapy consists of agressive management of heart failure and vasodilator therapy with
    - Diuretic
    - Salt restriction
    - ACEI or nifedipine (vasodilator).
    - Digoxin.
- **Surgical therapy**—Since severe symptoms (NYHA class III or IV) and left ventricular dysfunction with an ejection fraction less than 50% are independent risk factor for poor postoperative survival, surgery should be carried out in NYHA class-II patient before severe left ventricular dysfunction has developed.
  - *Indications for surgery*
    - Symptomatic chronic severe AR
    - Poor exercise tolerance
    - EF less than 50%
    - LV EDD more than 70 mm
    - LV ESD more than 50 mm.
  - *Operative procedure*
    - Annuloplasty for primary aortic root disease.
      Two procedures
      - » Encircling suture of the aorta
      - » Subcommissural annuloplasty.
    - Excision of a part of aorta with replacement by a graft or prosthetic valve in case of aneurysmal dilation of aorta.
    - AVR (aortic valve replacement) in severe AR, if it is due to primary valve disease.

AORTIC STENOSIS

Types of Aortic Stenosis

There are three types of aortic stenosis present depending on to anatomical site

- **Valvular**
  - Congenital
  - Rheumatic
  - Degenerative.

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- **Supravalvular**
  - Hourglass constriction of aorta
  - Membranous—Diaphragm in the aorta.
- **Subvalvular**.

Valvular

- **Congenital**
  - *Unicuspid*—Aortic valve produces severe obstruction in infancy.
  - *Bicuspid aortic valve* have abnormal architecture causes turbulent flow which traumatises the valve cusp resulting in fibrosis and calcification.
    It may be stenotic due to commissural fusion.
  - Tricuspid valve cusps are unequal in size with commissural fusion.
- **Acquired**
  - Rheumatic valvular AS usually with mitral valve involvement
    - It is mostly associated with AR or mitral valve disease.
  - Degenerative—Leading cause of AS in developed countries.
    - Two types
      - » Atherosclerotic
      - » Calcific
      - » Rare causes are
        - ▫ Rheumatoid involvement of cusp
        - ▫ Ochronosis with alcaptonuria.

Supravalvular

- Hourglass constriction (most common)—It is associated with marked thickening and disorganization of tunica media of aorta produce a constricting annular ridge at the superior margin of the sinus of Valsalva.
- Membranous type—Result of fibrous or fibromuscular semicircular diaphragm with a small central opening stretched across the lumen of the aorta.

Subvalvular

- Static obstruction—due to fibromuscular band.
- Dynamic obstruction—HOCM (hypertrophic obstructive cardiomyopathy).

Pathophysiology of Aortic Stenosis

In adult obstruction usually develops and progresses gradually.

This form of chronic progressive AS is well-compensated by LVH maintaining stroke volume for many years.

Late in the course of AS, left ventricular aortic pressure gradient, stroke volume, COP all decline resulting in PHTN and RVH.

LVEDV remains normal until late but LV mass increases due to concentric hypertrophy of left ventricle.

- Mild stenosis : Valve orifice size—1.5–2 cm^2.
- Moderate stenosis : Valve orifice size—1–1.5 cm^2.
- Severe stenosis : Valve orifice size—less than 1 cm^2.
- Critical stenosis : Valve orifice size—less than 0.8 cm^2.

Cardiac output is often within normal limit in most of the patients of severe AS in resting condition but often fails to rise during exercises.

Clinical Features of Aortic Stenosis

History

There is a long latent period for onset of symptoms. Manifestations most commonly commence at the 5th decade in congenital and rheumatic AS whereas 7th–9th decade in degenerative AS.

Cardinal manifestations are—

- **Angina** (seen in 66% of critical AS) is due to—
  - Decreased oxygen supply—
    - Due to decreased BP
    - Due to increased LVEDP.
  - Increased demand of O_2 due to increased LV mass.
- **Syncope** is due to—
  - Decreased cerebral perfusion, specially during exercise.
  - Malfunction of baroreceptor mechanism.
  - Vasopressor response due to greatly increased LV systolic pressure during exercise.
  - Ventricular fibrillation causing graying out spell or dizziness.
  - Transient AV block.
- **Exertional dyspnea**.
- **Heart failure**.

Dyspnea may be associated with PND and orthopnea, pulmonary edema reflecting varying degree of pulmonary HTN.

Marked fatiguability, debilitation, peripheral cyanosis are not prominent until late stage of the disease.

Survival curve shows that interval from the onset of the symptom to the time of death is approximately—

- 2 years with heart failure
- 3 years with syncope
- 5 years with angina.

Infective endocarditis is more common in younger with mild valvular disease but less common with old people with rock-like calcific aortic stenosis.

Physical Examination

- **Palpation—Outside precordium**
  - **Pulsus parvus et tardus**—Slow and low rise pulse.
  - **Systolic thrill** felt most prominent over carotid producing carotid shudder.
  - **Simultaneous palpation of apex and carotid** reveals a lag in the later.
  - **Pulsus alternans**—Suggests left ventricular dysfunction.

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- **Jugular venous pulse**—Prominent a-wave reflecting diminished RV compliance consequent to pulmonary hypertension and hypertrophy of septum.

Due to pulmonary hypertension secondary RVF and TR develop for which 'C V' or 'V' wave may seen in neck venous pulse.

- **Palpation of precordium**
  - **Apex**
    - Usually normal in position but may be shifted down and out which forceful and well-sustained.
    - Systolic thrill best appreciated when patient in upright position leans forward during full expiration. Palpated most readily in 2nd ICS on right side of sternum or suprasternal notch and is frequently transmitted to carotids (carotid shudder).
  - **Features of RVH and RVF**
    - Right parasternal heave
    - Engorged pulsatile neck veins (earliest)
    - Pedal edema
    - Hepatomegaly } Late feature
    - Ascites.

Auscultation of Aortic Stenosis

- S_1—Normal or soft.
- S_4—Prominent due to vigorous atrial contraction and partially closed mitral valve during presystole.
- **S_2**—
  - S_2 may be single due to inaudibility of A_2.
  - A_2 may be inaudible due to immobility caused by calcification of valve cusps.
  - P_2 is burried in prolonged ejection systolic murmur.
  - Prolongation of LV systole causes overlapping of A_2 with P_2 or there may be reverse splitting.
- **Aortic ejection click**—Due to sudden halting of upward movement of aortic valve—heard 0.06 second after S_1.
- **Ejection systolic murmur of AS is low pitch, rough and grasping in character**—Best heard at the base of the heart on right 2nd ICS close to sternum and breath held at the end of expiration, with the diaphragm of the stethoscope and well-transmitted to the carotids. The high-pitched component may be transmitted to the apex (**Gallavardin's** phenomenon) comes in differential diagnosis of murmur of MR.

  In patient with severe aortic stenosis but with pre serve COP, the murmur is of grade III/IV whereas in severe aortic stenosis with heart failure and low COP the murmur is relatively soft and brief.
- **More severe is the stenosis**.
  - More prolonged is the murmur of AS
  - Peaks later in systole.
- **Dynamic auscultation**—Murmur of valvular AS is augmented by squatting and reduces in intensity during starting of Valsalva maneuver and on standing.

Investigations ofAortic Stenosis

- Doppler echocardiography for diagnosis and assessment of aortic stenosis and LV function every yearly in symptomatic patients and at 2–5 years interval in asymptomatic patients with a mild to moderate AS.
- Cardiac catheterization—When AS is associated with coronary artery disease and when there is discrepancy between the clinical picture and echocardiographic finding.

Management of Aortic Stenosis

- **Medical management**—In patient with severe AS (<1 cm^2).
  - Avoid strenuous physical activity even in asymptomatic stages.
  - Avoid dehydration, low COP and hypovolemia.
  - β-blocker and ACEI are safe in asymptomatic patient used for CAD or HTN.
  - Nitroglycerine may be used for angina.
  - Statin (atorvastatin or rosuvastatin) is beneficial for degenerative calcific AS.
- **Surgical treatment of AS**—(mortality <1%)

Indications

- When orifice size is less than 1 cm^2.
- Symptomatic AS.
- Aneurysmal dilation of aortic root in moderate AS.
- When hypotensive response to exercise develops in AS with left ventricular dysfunction.

Valvoplasty can be done in small number of selected patients.

- In children—In noncalcified congenital AS who commonly have bicuspid valve—two types of procedure.
  - Commissural incision under direct vision (mortality less than 1%).
  - Balloon aortic valvoplasty when valve orifice area >0.8 cm^2. The development of regurgitation and restenosis after 10–20 years requires reoperation or valve replacement.
- In adult—Aortic valve replacement (AVR) is recommended.

MITRAL REGURGITATION

Abnormality of any one of the five components of mitral valve apparatus may cause MR. But the major causes of MR are

1. Rheumatic endocarditis
2. Mitral valve prolapse
3. Infective endocarditis
4. Cardiomyopathy
5. Ischemic heart disease.

- **Other rare causes are**
  - Endocardial cushion defect

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- Calcification of mitral annulus
- Carcinoid
- Collage vascular disorder
- Trauma.

- **Acute MR**—
  - Papillary muscle rupture (post-MI)
  - Endocarditis
  - Trauma
  - Rupture chordal/flail leaflet (MVP).
- **Chronic MR**—
  - Myxomatous MVP
  - Rheumatic (33%)
  - Endocarditis
  - Annular calcification
  - Congenital
  - HOCM
  - DCM.

Irrespective of cause, severe mitral regurgitation (MR) is most often pro- gressive, since enlargement of LA places tension on the posterior mitral leaflet pulling it away from the mitral orifice, thereby aggravating valvular dysfunction.

Similarly LV dilatation (LVEDD >6 cm) increases the regurgitation which in turn enlarges LA and LV further causing chordal rupture resulting in a vicious cycle hence it is said that, mitral regurgitation begets mitral regurgitation.

PATHOPHYSIOLOGY OF MITRAL REGURGITATION

In MR the volume of the regurgitant blood varies directly with LV systolic pressure and size of the regurgitant orifice. But later it is profoundly influenced by extent of LV dilatation.

- Normally ejection fraction rises in severe MR and in presence of normal LV function. Even a modest reduction in ejection fraction (EF) reflects significant dysfunction.
- V-wave in the LA pressure pulse in usually prominent with rapid Y descent.
- In chronic MR there is often increase in LV compliance so that LV volume rises with little elevation of diastolic pressure. The cardiac output is usually not affected but reduced in seriously symptomatic patient.
- In severe cases as much as 50% of total LV stroke volume regurgitates with each beat. Qualitative and clinically useful estimate of the severity of MR is made by the degree of LA opacification after injection of contrast material in LV or by color Doppler.
- Patient with acute MR usually have normal or reduced LA compliance with marked pulmonary congestion and edema.
- Patient with long-standing chronic severe MR usually have marked increase in LA compliance and are at the opposite end of the spectrum. These patients usually complain of severe fatigue and exhaustion secondary to low COP. AF is invariably present.

CLINICAL FEATURES OF MITRAL REGURGITATION

Symptoms

1. **In chronic severe MR**
   - Fatigue.
   - Exertional dyspnea.
   - Orthopnea.
   - Right-sided heart failure or its other presentation may be found where MR is associated with pulmonary vascular disease or marked PHTN.
2. **In acute severe MR**
   - LV failure with acute pulmonary edema is the common presenting feature.
3. **Physical finding**—
   a. *General examination*
      - BP—Normal.
      - JVP—Prominent a-wave in patient with sinus rhythm and marked PHTN.
      - Prominent v-wave in patient with MR with TR.

   b. *Systemic examination*—
      - Laterally displaced apex.
      - Palpable systolic thrill at the apex in 25% patients.
      - Hyperdynamic apex with brisk systolic impulse.
      - Left parasternal thrust and diastolic knock over pulmonary area due to pulmonary hypertension.

   c. *Auscultation*—
      - Soft S_1 buried (in murmur).
      - Wide splitting of S_2.
      - Low-pitched S_3 due to sudden tensing of papillary muscle, cordae tendinae and valve leaflet.
      - ***Pansystolic blowing murmur of grade 3 or 4 intensity found at the apex radiating to axilla, best heard during expiration with the bell of the stethoscope in case of dysfunction of anterior cusp of mitral valve.***
      - *In case of primary involvement* ***of posterior mitral leaflet, or ruptured chordae the regur- gitant jet strikes LA wall adjacent to aortic root; hence the murmur is transmitted to base of the heart*** *and in these cases it may be confused with murmur of AS.*
      - In MVP the murmur usually commences at late systole after a midsystolic click.
      - A middiastolic rumble over mitral area due to increase in flow across the mitral valve during first rapid filling phase of diastole may be heard. S_4 is audible in patient with acute severe MR who are in sinus rhythm.
      - In case of ruptured cordae the murmur has **cooing dove quality called Seagull murmur**.
      - In case of flail leaflet the murmur may have musical quality.

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INVESTIGATIONS OF MITRAL REGURGITATION

1. **Chest X-ray**—Enlargement of LA and LV but later LA enlarges massively and forms double contour on the right border of cardiac silhouette in chest X-ray (PA).
2. **ECG**—Shows features of
   - LA enlargement
   - RA enlargement
   - Atrial fibrillation
   - LV hypertrophy.
3. **Echocardiography**
   - Color flow Doppler is most accurate for diagnosis of MR.
   - 2D echo can identify the etiology.
   - LA is usually enlarged and exhibits increased pulsation.
   - LV may be hyperdynamic.
   - Mitral valve calcification and LV dyskinesia in ischemic MR.
   - Ruptured chordae, flail leaflet in post-AMI mitral regurgitation can easily be detected in echo.
   - Coarse erratic motion of involved leaflet in infective endocarditis can be seen.

TEE (transesophageal echocardiography)—Provides greater detailing.

MANAGEMENT OF MITRAL REGURGITATION

Medical Therapy (Table 4.1)

- Rest.
- Reduced NaCl intake.
- Enhanced NaCl excretion by diuretic.
- Vasodilator (nitrates) increase cardiac output by lowering BP and reducing regurgitant flow.
- Digitalis to slow ventricular rate.

IV GTN or nitroprusside reduces afterload thereby reduces the volume of regurgitant flow in acute severe MR with AMI.

- **ACE inhibitors** in ischemic MR.
- **Anticoagulant** and tight elastic stocking to reduce venous thrombi and pulmonary embolism.
- Prophylaxis for endocarditis should be given.

INDICATIONS OF SURGERY

1. **In severe MR** whose limitations do not allow full time employment or the performance of normal household activities despite optimal medical management.
2. **In asymptomatic MR with progressive LV dysfunction** with LVEF <60% and end systolic cavity dimension on echo >45 mm.
3. **In asymptomatic patient when PHTN or atrial fibrillation are present**.

Procedure

1. **Mitral valve replacement with prosthesis** is indicated where the valves are markedly deformed, shrunken, with calcified leaflet secondary to rheumatic fever.
2. **Mitral valvoplasty or annuloplasty** is indicated in patient with—
   a. Severe annular dilation.
   b. Flail leaflet.
   c. MVP (mitral valve prolapse).
   d. Ruptured chordae.
   e. Infective endocarditis.
      - MVR (mitral valve replacement) carries operative risk ~ 6%.
      - Valvoplasty or annuloplasty caries operative risk ~ 3%.
      - Thromboembolic or hemorrhagic complications occur with mechanical prosthesis requires replacement by bioprosthesis.

Table 4.1: Concise conservative medical therapy for valvular heart disease

		Symptoms control		Long-term therapy
A.	Mitral stenosis	1. β-blocker 2. Non-DHP CCB 3. Digoxin 4. Cardioversion for new onset AF may be tried 5. Diuretic for HF	For rate Control in AF	1. Penicillin prophylaxis for RF 2. Warfarin for AF or thromboembolism
B.	Mitral regurgitation	1. Diuretic for HF 2. Vasodilator therapy for acute MR with nitrate, ACEI 3. Counterpulsating aortic balloon for acute MR (in AMI)		1. Warfarin for AF or thromboembolism 2. Vasodilator for HTN
C.	Aortic stenosis	1. Diuretic for HF		1. Nil
D.	Aortic regurgitation	1. Diuretic and vasodilator, for HF ACEI and nitrate		1. Vasodilator for HTN

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ISBN 978-93-5270-072-1