Mitral Stenosis
Mitral Stenosis

- Epidemiology
- Etiology
- Symptoms
- Physical Exam
- Severity
- Natural history
- Timing of Surgery
Epidemiology

- 4 times more common in females
- Fully developed cases usually present >20 yrs
Etiology

- **Rheumatic heart disease** - 99% of cases
- **congenital**: frequently associated with other lesions causing LV outflow obstruction including AS, sub aortic stenosis, coarctation
- other rare causes:
  - calcified mitral valve ring, infective endocarditis (bulky vegetations may cause obstruction to flow), granulomatous infiltration in association with eosinophilia, SLE (treatment of Libman-Sachs endocarditis with steroids may lead to significant fibrosis with development of MS
Pathophysiology

- Normal valve area: 4-6 cm².

- Mild mitral stenosis:
  - MVA 1.5-2.5 cm²
  - Minimal symptoms

- Mod mitral stenosis
  - MVA 1.0-1.5 cm² usually does not produce symptoms at rest

- Severe mitral stenosis
  - MVA < 1.0 cm²

The stenosis here is due to fusion of the commissures.
Hemodynamic

- **Raised LAP** and hence **PCP** results in decreased pulmonary compliance, pulmonary edema and haemoptysis.

- **Persistently raised LAP** leads to reactive **pulmonary hypertension**. In late stages **RVF** occurs. Further reduces forward flow. Also **LV** distorted by bulging **RV**.

- **Raised LAP** results in **LA dilatation** and **AF**.

- Development of **AF** and loss of atrial contraction results in a marked deterioration of hemodynamic function and may lead to an exacerbation of **pulmonary oedema**.
  (Pregnancy and infection may also precipitate decompensation)

- **LV dysfunction** occurs in 1/4 of patients with severe **MS**. May be result of extension of scarring into myocardium and chronic reduction of preload.

- low cardiac output in severe cases.
Hemodynamic Changes in Mitral Stenosis With and Without Pulmonary Vascular Disease

Normal
- SVC
- RA
- RV
- PA
- PC
- PV
- LA
- LV
- Ao
- Flow: 6 L/min

Tight Mitral Stenosis
- Without Pulmonary Vascular Disease
- MVA = 1.0 cm²
- Flow: 5 L/min

Tight Mitral Stenosis
- With Pulmonary Vascular Disease
- MVA = 0.5 cm²
- Flow: 3 L/min

LA = Left Atrium
RA = Right Atrium
SVC = Superior Vena Cava
PC = Pulmonary Capillary
PV = Pulmonary Vascular Disease
LV = Left Ventricle
RV = Right Ventricle
IVC = Inferior Vena Cava
PA = Pulmonary Artery
Hemodynamic

- With long-standing elevation of the pulmonary venous pressure, anatomical changes in the pulmonary arterioles consisting of medial hypertrophy, intimal proliferation, and fibrosis lead to a significant decrease in the cross-sectional area of the pulmonary vascular bed. This, in turn, leads to a significant increase in pulmonary vascular resistance, markedly increasing RV and pulmonary artery pressure.
• Note the significant increase in both lymphatic drainage as well as the development of an engorged systemic bronchial venous circulation.

• **Brisk hemoptysis** may occur when these engorged bronchial veins rupture.
Symptoms

- Fatigue
- Palpitations
- Cough
- SOB
- Palpitation

- A fib
- Systemic embolism
- Pulmonary infection
- Hemoptysis
- Right sided failure
  - Hepatic Congestion
  - Edema

- Worsened by conditions that ↑ cardiac output.
  - Exertion, feverth, anemia, tachycardia, Afib, intercourse, pregnancy, thyrotoxicosis
**Signs**

**Palpation:**
- Small volume pulse
- Tapping apex-palpable S\(_1\)
- +/- palpable opening snap (OS)
- RV lift
- Palpable S\(_2\)

**Auscultation:**
- Loud S\(_1\)- as loud as S\(_2\) in aortic area
- A\(_2\) to OS interval inversely proportional to severity
- Diastolic rumble: length proportional to severity
- In severe MS with low flow- S\(_1\), OS & rumble may be inaudible
ECG: LAE, AFIB, RVH, RAD
CXR:

- Enlarged LA and LA appendage
- upper lobe blood diversion
- ± pulmonary edema
Timing of A2 to OS Interval

- Width of A2-OS inversely correlates with severity.
- The more severe the MS the higher the LAP the earlier the LV pressure falls below LAP and the MV opens.

- First heart sound \( (S1) \) is accentuated and snapping
- Opening snap \( (OS) \) after aortic valve closure
- Low pitch diastolic rumble at the apex
- Pre-systolic accentuation (esp. if in sinus rhythm)
Natural History

- Progressive, lifelong disease,
- Usually slow & stable in the early years.
- Progressive acceleration in the later years
- 20-40 year latency from rheumatic fever to symptom onset.
- Additional 10 years before disabling symptoms
Atrial dysrrhythmias
- Systemic embolization (10-25%)
  - Risk of embolization is related to age, presence of atrial fibrillation, previous embolic events
- Congestive heart failure
- Pulmonary infarcts (result of severe CHF)
- Hemoptysis
  - Massive: secondary to ruptured bronchial veins (pulm HTN)
  - Streaking/pink froth: pulmonary edema, or infection
- Endocarditis
- Pulmonary infections
Role of Echocardiography

- Diagnosis of Mitral Stenosis
- Assessment of hemodynamic severity
  - mean gradient, mitral valve area, pulmonary artery pressure
- Assessment of right ventricular size and function.
- Assessment of valve morphology to determine suitability for percutaneous mitral balloon valvuloplasty
- Diagnosis and assessment of concomitant valvular lesions
- Reevaluation of patients with known MS with changing symptoms or signs.
- F/U of asymptomatic patients with mod-severe MS
Therapy

Medical

- Diuretics for LHF/RHF
- Digitalis/Beta blockers/CCB: Rate control in A Fib
- Anticoagulation: In A Fib
- Endocarditis prophylaxis

Balloon valvuloplasty
Effective long term improvement

Surgical

- Mitral commissurotomy
- Mitral Valve Replacement
  - Mechanical
  - Bioprosthetic
Indications for surgery

- significant symptoms which limit normal activity
- pulmonary oedema without precipitating cause
- recurrent emboli
- pulmonary oedema in pregnancy (emergency valvotomy)
- deterioration due to AF which does not respond to medical treatment
- valve cross-sectional area < 1.3 cm² (valvotomy) or < 1 cm² if there is a possibility that valve replacement may be required