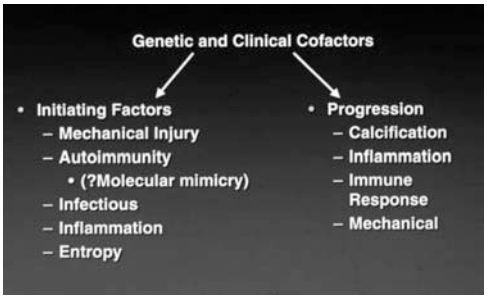
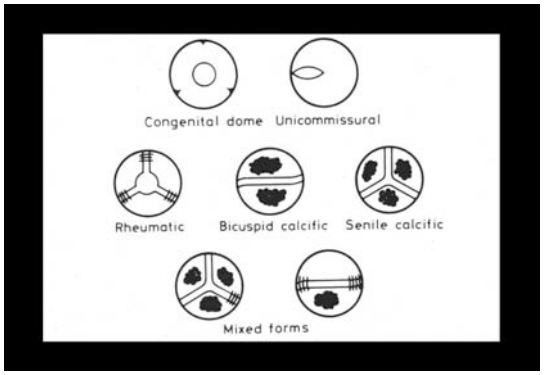
Valvular Heart Disease

General Principles

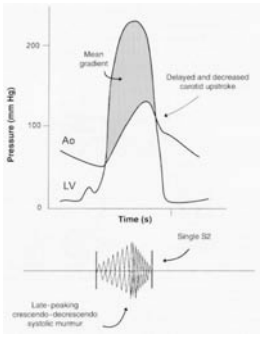
- Etiology
- Cellular and molecular mechanism of valve damage
- Structural pathology
- Functional pathology - stenosis/regurgitation
- Loading conditions - pressure/volume
- Compensation
- Decompensation
- Natural history
- Treatment - type and timing





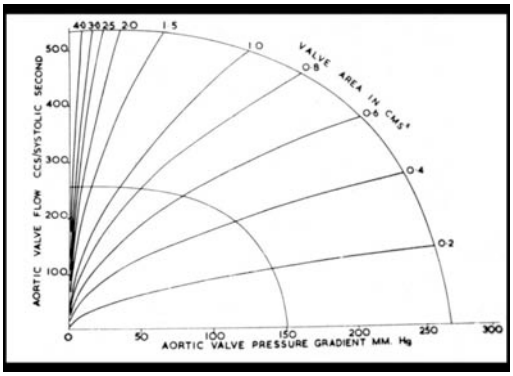
Effect of AV Stenosis on LV Loading

- **Ejection across stenotic aortic valve requires a systolic pressure gradient between the LV and aorta.**
- **This places a pressure load on the LV.**



Relation of AV Gradient to Flow and Valve Area

- $\Delta P = k (\text{Systolic Flow}/AVA)^2$
- Systolic Flow = Cardiac output/(HR x SEP)



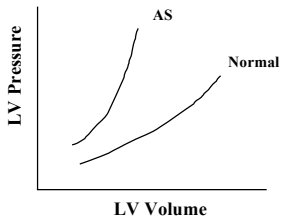
Compensation

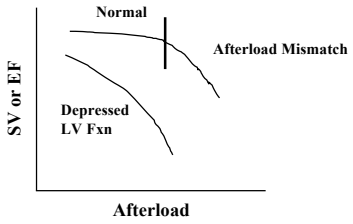
- Pressure load -> concentric LVH (increased wall thickness)
- Increased wall thickness -> increased LV pressure generation
- Increased wall thickness -> normalize wall stress (= LVP/2 x Wall Thickness)

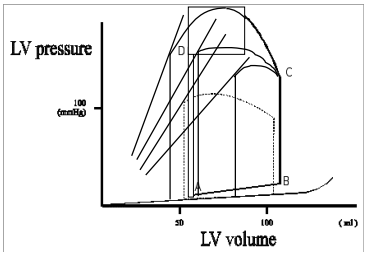
Decompensation

- LVH -> decreased LV compliance
- Inadequate LVH -> afterload mismatch
- Eventual irreversible depression of contractility

Diastolic Pressure-Volume Relationship

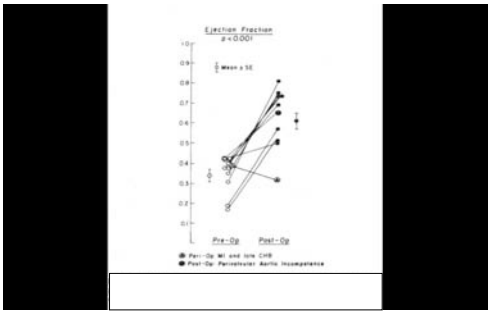


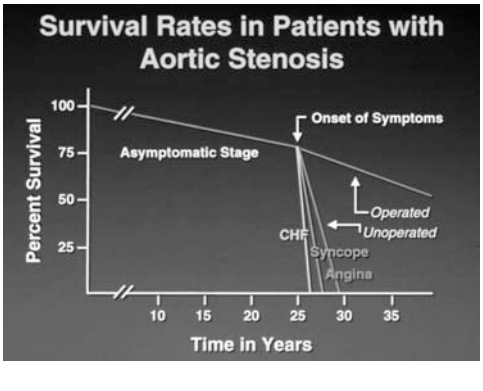


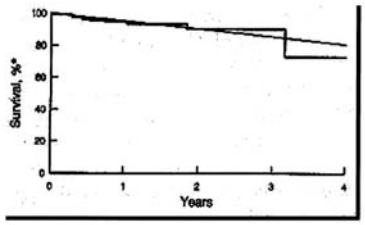


AS - compensated
AS - afterload mismatch
Normal
AS - depressed contractility

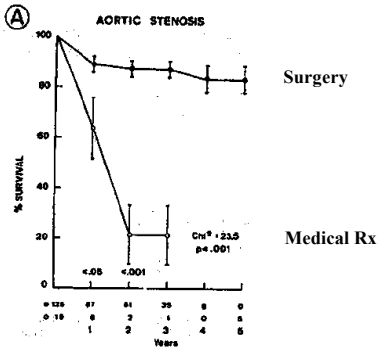
EF Improvement after AVR for AS







Survival for 113 patients with asymptomatic, severe aortic stenosis (thick line) who did not receive early intervention (group 2) compared with survival for age- and gender-matched control subjects (thin line). * Censored at symptoms, aortic valve replacement or valvuloplasty.



Surgery

Medical Rx

Symptoms

- **Exertional**
  - **Dyspnea**
  - **Syncope**
  - **Angina**

Dyspnea

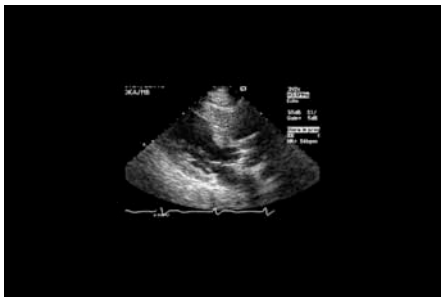
- Decreased LV compliance -> increased LVDP -> increased PCW
- Afterload mismatch -> decreased LVEF (or SV)
  - Increased LV ESV
  - Increased LV EDV
  - Increased LVEDP
- Irreversible decreased LV contractility -> increased LVEDP

Syncope

- CO = HR x SV
- During exercise (normal response):
  - Mean arterial BP (--) = CO (increased) x total peripheral resistence (decreased)
- Severe AS response to exercise:
  - Less of increase in CO due to a smaller increase in SV
  - MAP (decreased) = CO (less increase) x total peripheral resistence (decreased)
  - Increased LVP -> LV baroreceptors -> further decreased in total peripheral resistence

Angina

- Increased myocardial oxygen demand: increased muscle mass, increased afterload
- Decreased supply
  - Coronary perfusion pressure (decreased) = Aortic diastolic pressure - LVDP (increased)

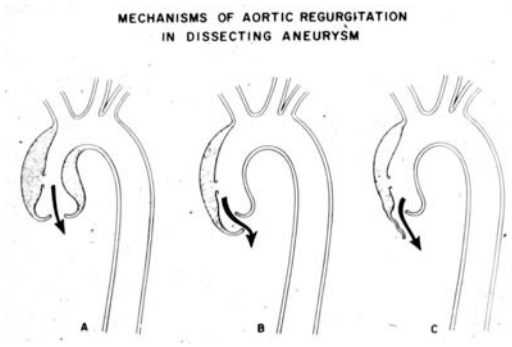


Aortic Regurgitation - Pathoanatomy

- **Aortic valve**
- **Aortic root**
- **Both**

Acute AI-Etiology

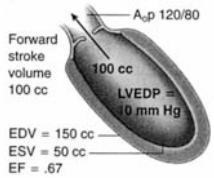
- Infective Endocarditis
- Aortic Dissection
- Failed Bioprosthesis
- Mechanical Valve Failure
- Blunt Trauma
- Cusp Rupture or Prolapse
  - Spontaneous
  - VSD
  - Myxomatous Valve
- Inflammatory disease
- Post PABV or AV Surgery



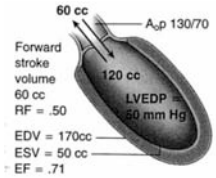
Acute AR - Pathophysiology

- **Normal: CO = SV x HR**
- **Valve Regurgitation**
  - **CO = SV (1 - RF) x HR**
  - **RF (Regurgitation fraction) = Regurgitation volume/SV**
- **Normal LV size and compliance limits increase in EDV and SV and therefore CO.**
- **Rapid rise to high level of LV diastolic pressure due to filling of LV from Ao as well as LA. LV diastolic pressure closes MV in diastole and further limits forward flow.**
- **This leads to pulmonary congestion and decreased CO -> death.**

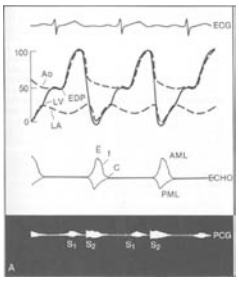
Normal

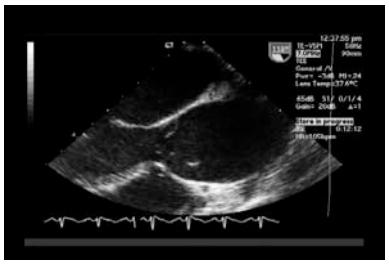


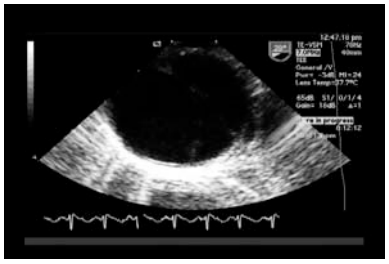
Acute AR

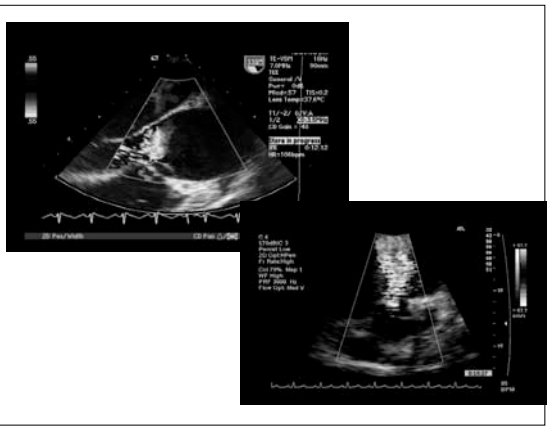


Acute AR

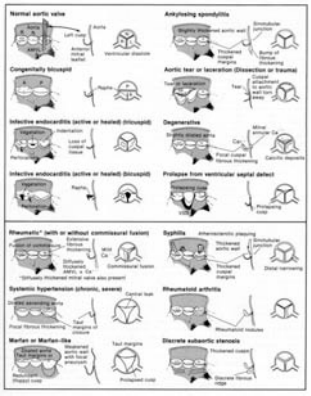








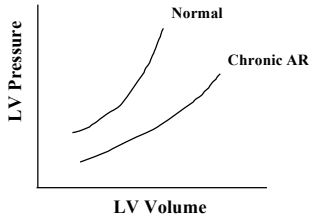
Chronic AR - Etiology



Chronic AR - Pathophysiology

- **Chronic AR develops slowly enough to allow eccentric LVH -> increased LV chamber size, changing LV pressure/volume relationship. The LV can then accommodate a large regurgitant volume at normal LV diastolic pressure.**

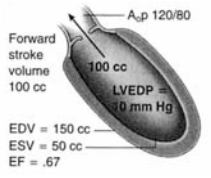
Diastolic Pressure-Volume Curves



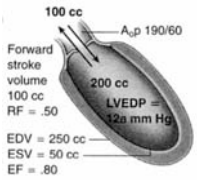
Chronic AR - Pathophysiology

- **CO = SV (1-RF) x HR**
- **CO is maintained because of increased eccentric hypertrophy -> increased SV. Thus, normal CO and LVDP allow chronic severe AR to be asymptomatic**
- **Increased SV -> increased SBP and widened pulse pressure -> increased afterload**
- **Increased LVEDV = increased volume load**

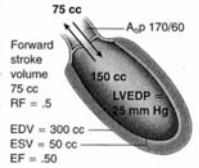
Normal



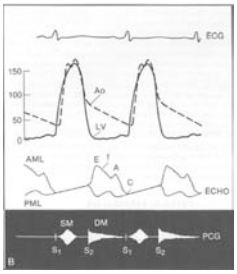
Chronic AR - Compensated



Chronic AR - Decompensated



Chronic AR



Symptoms

- **Dyspnea**
  - **Increased LVEDV -> Increased LVEDP -> Increased LAP**
  - **Afterload mismatch -> Increased LVESV -> Increased LVEDV-> Increased LAP**
  - **Decreased myocardial contractility -> Increased LVEDV-> Increased LAP**
- **Angina**
  - **Increased demand due to increased afterload**
  - **Decreased supply due to decreased coronary perfusion pressure (CPP = Ao diastolic pressure - LVDP); Ao diastolic pressure is decreased and LVDP is increased**

Natural History of AR

- **Chronic AR**
  - **Low risk until decreased LVEF or symptoms**
  - **Symptom onset or decreased LVEF -> progression to death or irreversible LV dysfunction over several (1-5) years**
- **Acute AR**
  - **Pulmonary congestion, low cardiac output, death (over hours to days)**

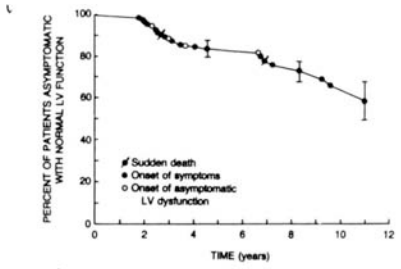
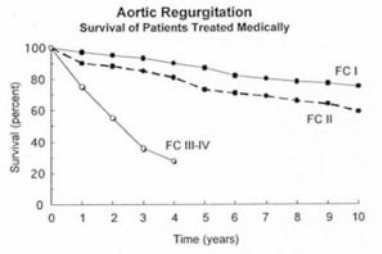


Figure 13. Low incidence of development of symptoms or LV dysfunction in younger patients with chronic severe AR. From Reference 59.



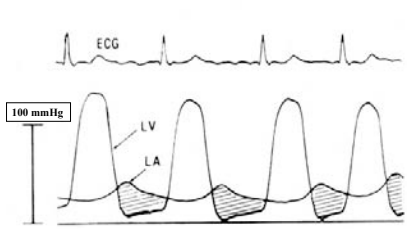
Mitral Stenosis

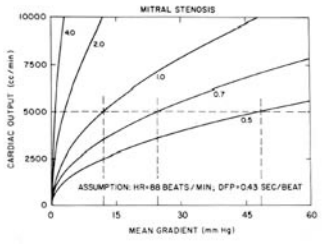
Etiology

- **Rheumatic**
- **Prosthetic Valve Dysfunction**
- **Rare - myoma, mitral annular calcification**

Mitral Stenosis - Pathophysiology

- **High LA pressure is needed to maintain flow across stenotic MV**
- **This leads to diastolic gradient across MV**
- **Flow and gradient related by:**
  - **Δ P = (Flow/MVA)^2**
  - **Flow = CO/(DFP x HR)**





Mitral Stenosis - Pathophysiology

- Increased LA pressure -> Increased PCWP -> Increased PAP -> RV pressure overload
- A chronic increase in PCWP -> pulmonary arteriolar constriction -> Increased PVR -> Further increase in PAP -> Increased RV pressure overload

Symptoms

- Exercise -> Increased flow
- Increased HR -> Increased flow (due to decreased diastolic time)
- Increased flow -> Increased gradient -> Increased LAP = Increased PCWP -> dyspnea
- Increased PCWP and increased PVR -> RV pressure overload -> decreased RVEF and stroke volume -> decreased CO and increased RA pressure
- Increased RA pressure and decreased CO -> fluid retention -> edema, ascites, liver congestion, increased JVP

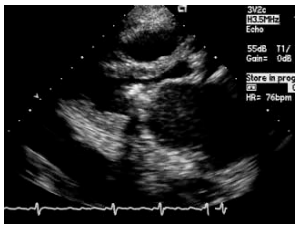
Symptom Onset

- Gradual and initially on exertion
- Rapid and abrupt if increased CO and HR
  - Fever/infection, pregnancy, anemia, hyperthyroid etc

Natural History

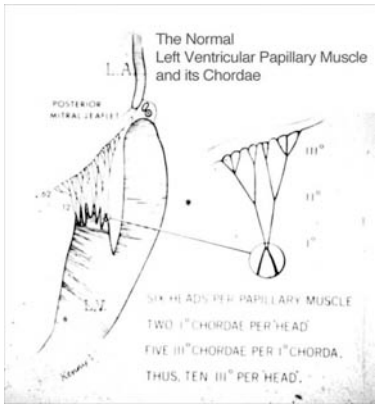
- **ARF**
- **Symptom onset**
- **Severe disability**
- **Death**

5-40 years



Mitral Apparatus

- Mitral Annulus
- Mitral Leaflets-Anterior and Posterior
- Chordae Tendineae
- Papillary Muscles
- Left Ventricle



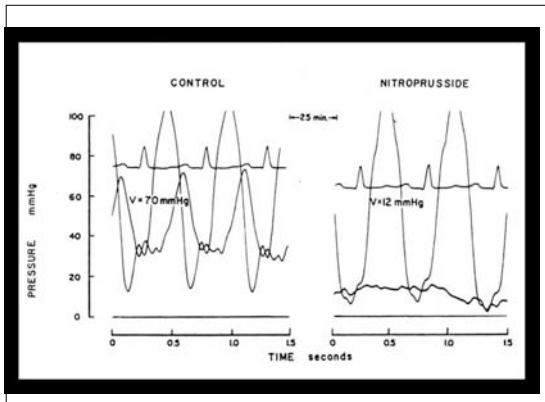
Mitral Regurgitation

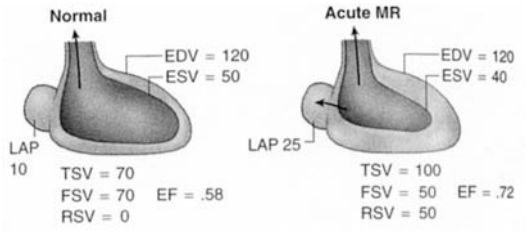
Etiology

- **Acute MR**
  - **Infectious endocarditis**
  - **Spontaneous chordal rupture (myxomatous degeneration)**
  - **Papillary muscle rupture (acute MI)**
  - **Prosthetic valve failure**
- **Chronic MR**
  - **Same as acute**
  - **MVP/Myxomatous degeneration**
  - **Rheumatic**
  - **Ischemic**
  - **Functional (dilated cardiomyopathy)**

Acute MR - Pathophysiology

- **Regurgitation into normal size and normally compliant LA**
  - **Marked increase in LA pressure**
  - **Increased PCWP -> pulmonary congestion**
- **CO = SV (1 - RF)xHR; CO decreases because SV increase is limited by normal LV size and compliance**
- **Sudden increase in PCWP may lead to increased PVR -> RV failure**





Natural History

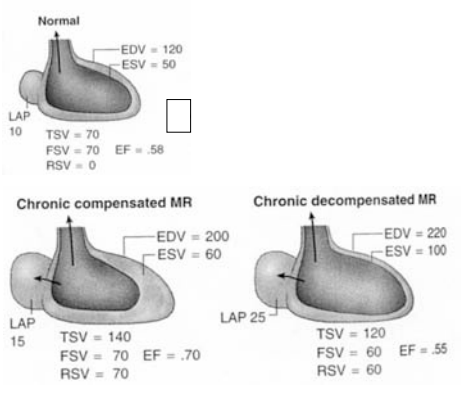
- **If severe -> pulmonary edema, death**
- **If LV systolic function abnormal -> shock, pulmonary edema, death**

Chronic MR - Pathophysiology

- **Eccentric Hypertrophy allows large regurgitant volume to be accommodated at normal LV diastolic pressure (i.e. shift of LV diastolic P-V relationship)**
- **LA enlargement accommodates regurgitant volume at normal pressure**
- **Ejection into LA provides decrease in afterload (i.e. extra preload is ejected into low pressure LA, no isovolumetric contraction period decreases systolic wall stress)**
- **Usual measures (i.e. EF, ESD) of systolic function may be normal with abnormal contractility, and are greater than normal while myocardial contractility is maintained.**

Chronic MR - Pathophysiology

- **When systolic function worsens (decreased LVEF) -> further increase in LVEDV and LA pressure -> pulmonary congestion.**



M R
Symptoms

1) LAP and C.O. (and therefore symptoms) determined by

i) degree of MR
ii) rate of development of MR
iii) LA compliance
iv) LV compliance
v) LV systolic function
vi) Afterload
vii) preload

Natural History of Chronic MR

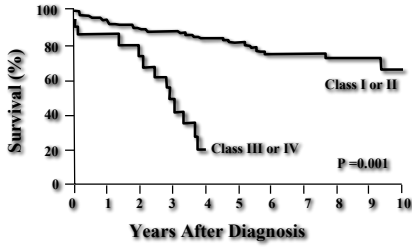
- **Structural MR**
  - **Onset of symptoms or fall of LVEF into normal range mark transition to development of irreversible LV dysfunction, CHF, and death over several year period**
- **Functional MR**
  - **National history tied to underlying disease (i.e. dilated CM, ischemic heart disease)**

Long Term Survival With Medical Therapy Stratified by Initial EF

No. at Risk											
EF >60%	185	109	94	83	69	61	45	30	19	11	6
EF <60%	44	24	21	20	15	9	6	4	1	1	1

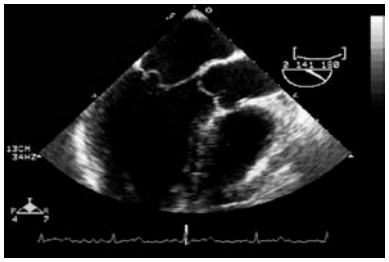
Ling et al. NEJM 1996;335:1417-23

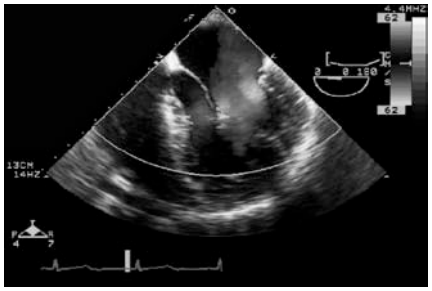
Long Term Survival With Medical Therapy Stratified by NYHA Class



No. at Risk											
Class I or II	162	117	102	95	80	69	50	33	20	12	7
Class III or IV	66	15	12	7	3						

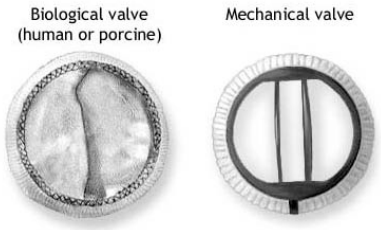
Ling NEJM 1996;335:1417-23

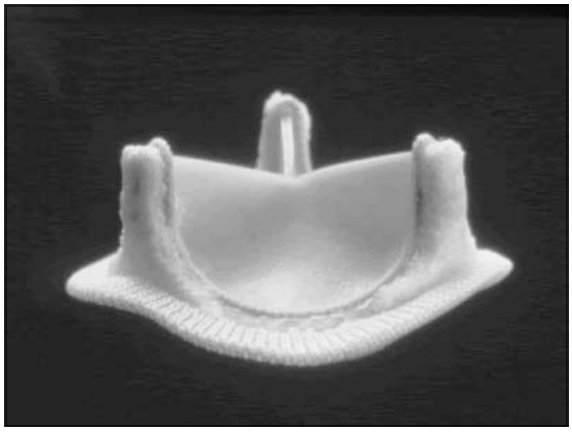




Mechanical Treatments

- **Prosthetic Heart Valves**
  - **Mechanical (durable, thrombogenic)**
  - **Tissue (less durable, less thrombogenic)**
  - **Repair preferred**
- **Percutaneous Treatments**
  - **Balloon valvuloplasty for MS, PS**
  - **Mitral repair**
  - **Aortic and pulmonic valve replacement**





Percutaneous Aortic Valve