

# Cardiac pathology

## lecture 6

# VALVULAR HEART DISEASE

by

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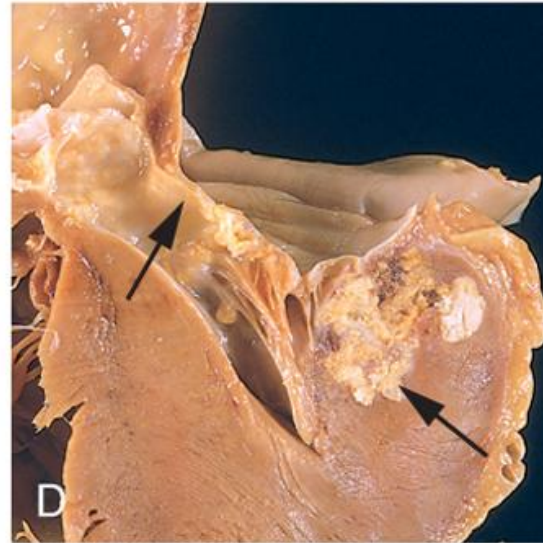
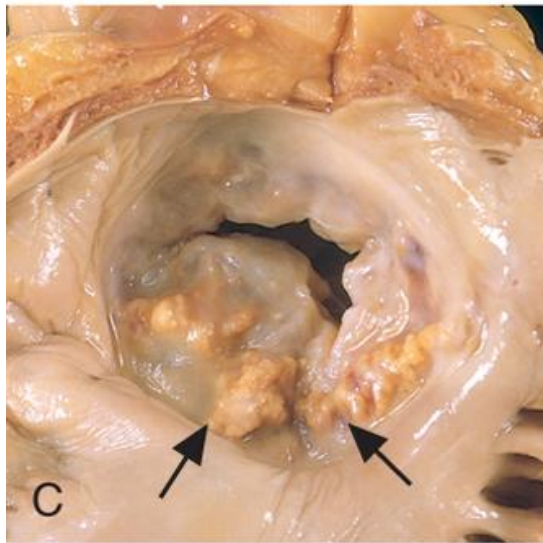
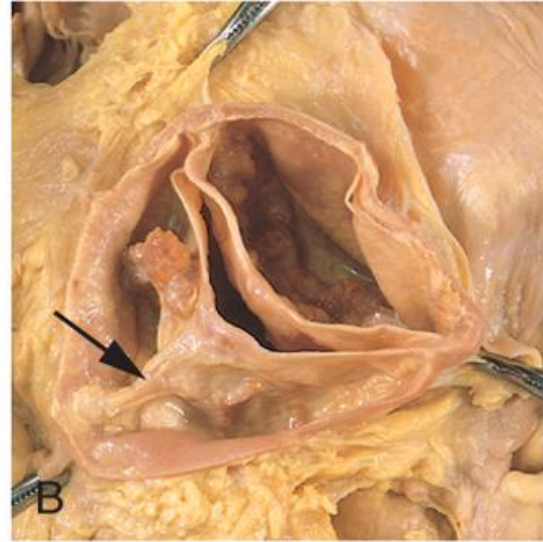
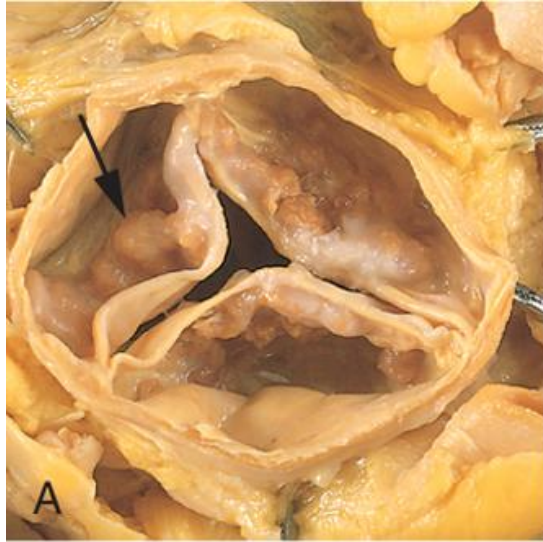
- **Stenosis** *is the failure of a valve to open completely, obstructing forward flow.*
- Valvular stenosis is almost always a chronic process caused by a primary cuspal abnormality (e.g., calcification or valve scarring).
- **Insufficiency** *results from failure of a valve to close completely, thereby allowing reversed flow.*

- Valvular insufficiency may result from:
  - ❑ **Intrinsic disease** of the valve cusps (e.g., valve destruction)
  - ❑ Distortion of the **supporting structures** (e.g., the **aorta**, mitral **annulus**, tendinous **cords**, **papillary muscles**, **ventricular free wall**) without primary changes in the cusps.
- It can appear **acutely**, as with chordal rupture, or **chronically** due to leaflet scarring and retraction.

- Stenosis or regurgitation can occur in pure forms, or may coexist in the same valve.
- Valvular disease may affect only a single valve (the mitral valve is most commonly affected), or more than one valve.
- Abnormal flow through diseased valves typically produces abnormal heart sounds called murmurs.
- Valve abnormalities can be congenital or acquired.
- acquired stenoses of the aortic and mitral valves account for approximately two-thirds of all valve disease.

## Calcific Aortic Stenosis

- It is the **most common cause of aortic stenosis** and is usually the consequence of calcification from **progressive age**-associated "wear and tear" of either anatomically **normal aortic valves** or **congenitally bicuspid** valves.
- It typically begins to manifest when patients reach their **70s and 80s**; onset with bicuspid aortic valves is at a much earlier age (**40-50 years**).



- **Clinical features:**

- **Angina** : due to left ventricular outflow obstruction with raised intraventricular pressure to >200 mm Hg and subsequent left ventricular hypertrophy and relative ischemia.
- **Syncope**: brain hypoperfusion.
- **CHF**

## Myxomatous Mitral Valve (prolapsed)

- In *myxomatous degeneration of the mitral valve*, one or both mitral leaflets are "floppy" and *prolapse*, meaning that they balloon back into the left atrium during systole.
- *Mitral valve prolapse* is a primary form of myxomatous mitral degeneration affecting 3% to 5% of adults.
- women seven times more frequently than men.
- Secondary myxomatous mitral degeneration can occur in any of a number of settings in which mitral regurgitation is caused by some other entity (e.g., IHD).



- Clinical Features

- Most patients with mitral valve prolapse are asymptomatic, and the valvular abnormality is usually discovered only incidentally on physical examination.
- A minority of patients may complain of palpitations, dyspnea, or atypical chest pain.
- Auscultation discloses midsystolic click(s) ± regurgitant murmur.

- **3% experience one of several complications:**
  - **Mitral regurgitation and CHF**, particularly if the chordae or valve leaflets rupture.
  - Increased risk for the development of **infective endocarditis**.
  - Sudden death caused by **ventricular arrhythmias**.
  - **Stroke** may occur from **embolism** of thrombi formed in the left atrium.

## Rheumatic Valvular Disease

- Rheumatic fever (RF) is an acute, immunologically mediated, multisystem inflammatory disease that occurs a few weeks after an episode of group A  $\beta$ -hemolytic streptococcal pharyngitis; it can also rarely occur with streptococcal infections at other sites (e.g., skin).

- Acute rheumatic heart disease (RHD) is the cardiac manifestation of RF and is associated with inflammation of the valves, myocardium, or pericardium.
- RHD leads to diffuse and dense scarring of valves resulting in permanent dysfunction (**mitral stenosis** being most common).

- **Acute RF** is characterized by **Aschoff bodies** (consist of a central zone of degenerating, hypereosinophilic extracellular matrix infiltrated by lymphocytes, occasional plasma cells, and plump activated macrophages called **Anitschkow** cells).

- Aschoff bodies can be found in any of the three layers of the heart-pericardium, myocardium, or endocardium (including valves)-so-called **pancarditis**.
- The pericardium shows a **fibrinous or serofibrinous exudate**, which generally resolves without sequelae.

- The myocardial involvement (myocarditis) takes the form of scattered **Aschoff bodies** within the interstitial connective tissue.
- Valve involvement results in **fibrinoid necrosis** along the lines of closure forming 1- to 2-mm vegetations (**verrucae**) that have little effect on cardiac function.

- **Chronic RHD** is characterized by organization of the acute inflammation and subsequent **scarring**.
- The cardinal anatomic changes of the mitral (or tricuspid) valve include **leaflet thickening, commissural fusion and shortening, and thickening and fusion of the chordae tendineae**.
- Fibrous bridging across the valvular commissures and calcification create "**fish mouth**" stenoses.



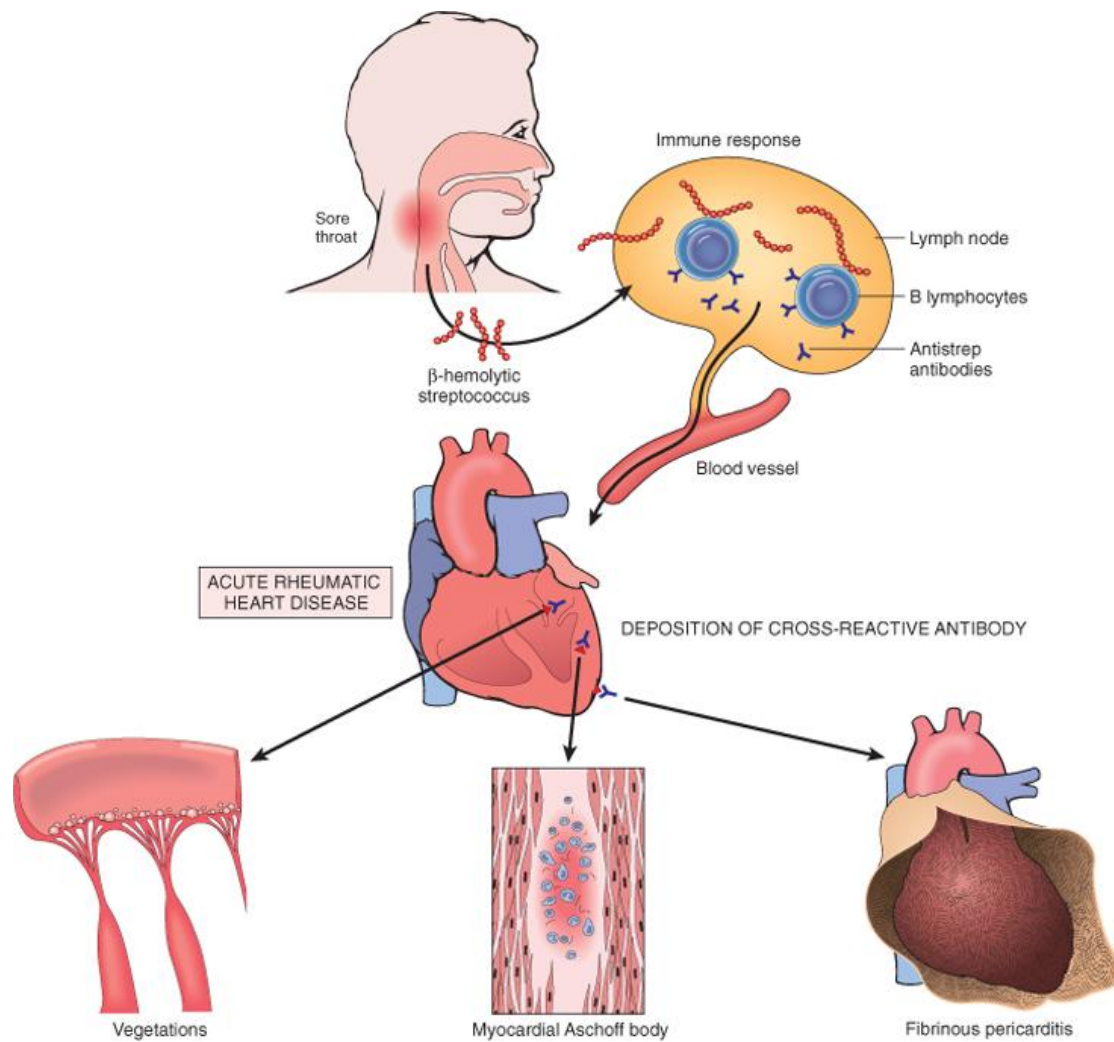
- Microscopically there is neovascularization, with diffuse fibrosis that obliterates the normal leaflet architecture.
- Aschoff bodies are rarely seen in chronic RHD.

- ❖ The functional consequence of RHD is **valvular stenosis and regurgitation** (stenosis tends to predominate).
- ❖ RHD is overwhelmingly the most frequent cause of mitral stenosis accounting for 99% of cases.

- The **mitral valve alone is involved in 70% of cases of RHD.**
- Combined mitral and aortic disease account for 25% of cases.
- the tricuspid valve is usually less frequently and less severely involved, and the pulmonic valve almost always escapes injury.
- **With tight mitral stenosis, the left atrium progressively dilates and may harbor mural thrombi.**

- **Pathogenesis**

- *Acute RF is a hypersensitivity reaction induced by host antibodies elicited by group A streptococci.*
- The M proteins of certain streptococcal strains induce host antibodies that cross-react with glycoprotein antigens in the heart, joints, and other tissues.
- A genetic susceptibility is likely to influence the development of the pathogenic antibodies.
- The chronic sequelae result from progressive fibrosis due to healing of the acute inflammatory lesions.



Thank  
you