# Valvular heart diseases

- Objectives:
  - Know the clinical presentation of Valvular Heart Disease (VHDs)
  - Identify the Etiology of VHDs.
  - Do clinical assessment of VHDs.
  - Do Laboratory & non-invasive assessment of VHDs.
  - Apply medical management of VHDs.
  - Apply invasive management of VHDs.

[ Color index : **Important** | **Notes** | **Extra** ]

- Resources:
  - 435 slides
  - Kumar clinical medicine 8th edition, Step-Up to medicine, Internal Medicine Kaplan USMLE Step 2 CK, Organ systems - first aid.

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# Mind Map

## VHDs

### Mitral Stenosis

Almost all cases are due to rheumatic heart disease.
**Murmurs:**
- Opening snap followed by low-pitched diastolic rumble.
- Loud S1.

### Mitral Regurg.

**Murmurs:**
- Holo\Pan-systolic murmur
- S3 gallop.

❖ **Mitral prolapse**:
- Large mitral valve leaflets.
- Midsystolic or late systolic click.

Mid-to-late systolic murmur

### Aortic Stenosis

**MCC:**
Calcification and degeneration of a normal valve in elderly.
**Murmur:**
- Harsh crescendo–decrescendo systolic murmur.
- S4 gallop.

### Aortic Regurg.

- Widened pulse pressure—markedly increased systolic BP, with decreased diastolic BP.
- Diastolic decrescendo murmur.
- Apical Rumble – "Austin Flint Murmur"

## Vulvular heart diseases that cause:

### Pressure overload

- Aortic stenosis
- Mitral stenosis

### Volume overload

- Chronic mitral regurg.
- Chronic tricuspid regurg.

The most imp things are: Mitral & Aortic valve diseases. **Focus on them**!

# Introduction

**Cardiac cycle + Heart sounds**
14 min

Start this lecture by watching this video, by one of our colleagues: (it includes the introduction part of female slides.)

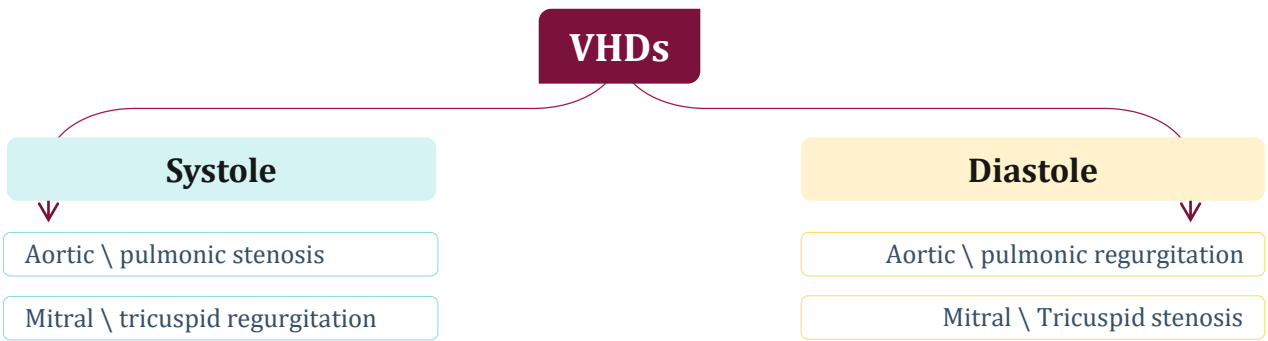
## Characteristic of heart valves

| Valve | Structure | Site of auscultation | Phase when valve is open | Sound |
|---|---|---|---|---|
| Mitral | Bicuspid | Left 5th IS at the midclavicular line | Diastole | S1 (Lub) |
| Tricuspid | Tricuspid | Left 5th IS at the SB | | |
| Aortic | Semilunar (3 cusps) | Right 2nd intercostal space (IS) at the sternal border (SB) | Systole | S2 (Dub) |
| Pulmonic | Semilunar (3 cusps) | Left 2nd IS at the SB | | |

## Heart sounds & significance:

| Sound | Significant |
|---|---|
| **$S_1$** | MV & TV closure; the MV closes before the tricuspid valve, so $S_1$ may be split. |
| **$S_2$** | AV & PV closure; the AV closes before the PV; inspiration causes increased splitting of $S_2$. |
| **$S_3$** | During rapid ventricular filling (early diastole) normal in children; in adults, associated with dilated ventricle (ie, dilated CHF) & increased filling pressure. |
| **$S_4$** | Late diastole; not audible in normal adults; its presence suggest high atrial pressure or stiff ventricle (ie. Ventricular hypertrophy). The left atrium must push against a stiff LV wall ("atrial kick") |

- **Regurg/ Insuff:** leaking (backflow; against its direction) of blood across a **closed** valve.
- **Stenosis:** Obstruction of (forward) flow across an **opened** valve.




The most imp things are: Mitral & Aortic valve diseases. **Focus on it**!

# Mitral Stenosis

Most common lesion caused by rheumatic fever consisting of thickened mitral valve leaflets, fused commissures, and chordae tendineae. May result in right ventricular failure.

## Etiology

- **Rheumatic Fever**; related to streptococcus infections, causing damage to the mitral valve and leading to mitral stenosis later in life. (**the most common**) (more common in female)
- Other less common causes:
  - Congenital Mitral Stenosis, Systemic Lupus Erythematosus, Rheumatoid Arthritis, Atrial Myxoma (tumor), Malignant Carcinoid, Bacterial Endocarditis

## Pathophysiology

- **Pathogenesis**:
  - Cusps thicken → commissures (the area bet 2 leaflets) fused together → chordae tendinae becomes thickened & shortened → calcium deposits form.

1. **LA hypertension**
   - **Pulmonary interstitial edema.**
   - **Pulmonary hypertension**
     - Passive → obligatory to preserve forward flow
     - Reactive → vascular changes in 40%
       - Protects interstitium from edema
       - **Leads to right heart failure** → pts w\ MS come w\ right side HF first!!
   - LA stretch & atrial fibrillation
     - ↑ HR → ↓ LV filling.
     - ↓ atrial "kick" → ↓ LV filling
     - Atrial thrombus formation & embolus.
2. **Limited LV filling & cardiac output.**
   - When the orifice is reduced to approximately $2cm^2$, which is considered mild mitral stenosis, blood can flow from the left atrium to the left ventricle only if propelled by an abnormal pressure gradient – the hemodynamic hallmark of Mitral Stenosis.

## Symptoms

- ✓ Normal MV area = 4-6 $cm^2$, symptoms begin when the MV area = < $2cm^2$
- **Dyspnea on exertion** → pulmonary venous congestion.
- Fatigue → ↓ cardiac output.
- Inability to tolerate the increased volume,
- **Inability to tolerate the increased HR**
  - ↓ filling
  - ↑ LA pressure & ↑ pulmonary vein congestion.
  - The increased HR→ ↓ flow rate in the valve, bc the valve is narrow, the diastole filling time is ↓ → more tension & pressure
- Hemoptysis → due to rupture of thin dilated bronchial veins.
- Orthopnea, paroxysmal nocturnal dyspnea (PND), pulmonary edema (developed when there is a sudden glow rate across a markedly narrowed mitral orifice), palpitation (arrhythmias), Chest pain, Peripheral edema.
- Hoarseness (due to impingement of an enlarged left atrium on the recurrent laryngeal nerve)
- Systemic embolism (due to stagnation of blood in an enlarged left atrium)

# Mitral Stenosis (Cont.)

## Signs

- **Mitral stenosis murmur**:
  - The **opening snap** is followed by a low-pitched diastolic rumble (mid-diasyolic murmur) (due to turbulent blood flow across the stenotic valve) and **presystolic accentuation.** (squatting & leg raising increase the intensity)
- S2 is followed by an opening snap.
- **Loud S1**, due to abrupt leaflet closure → wont occur if the valve is calcified. It might be the most prominent physical finding.
- loud S2 – due to pulmonary HTN (if present)
- May be associated with:
  - Mitral regurgitation or aortic stenosis.
  - Right sided murmurs
    - Pulmonary insufficiency → Graham Steel Murmur (early diastolic murmur)
    - Tricuspid regurgitation.
- Atrial fibrillation (irregular cardiac rhythm)
- Sternal lift (due to right ventricular enlargement)

## Diagnosis

- Clinical evaluation of Mitral Stenosis begins with an in-depth history and physical exam.
- **ECG**: (ECG is helpful but not diagnostic!)
  - Atrial fibrillation (AFib), right atrial enlargement, right ventricular hypertrophy.
  - left atrial enlargement (biphasic P wave in leads $V_1$ and $V_2$)
- **Echocardiography** (Echo 2D/color doppler) → test of choice.
  - TransEsophageal Echocardiography.
- Cardiac Cath → helpful, confirmatory. Needed if the pt is older (look at the coronaries)
- Chest radiology.

## Complication

- **Atrial fibrillation**
- Lung congestion.
- Blood clots with systemic embolization (due to stagnation of blood in an enlarged left atrium)
- Pulmonary hypertension
- Congestive heart failure (CHF)

## Treatment

- Treatment of symptomatic mitral stenosis:
  1. Medical therapy → treat the symptoms not the cause.
     - ✓ Diuretics → for congestion.
     - ✓ Digoxin, Beta & Ca channel blockers → for AFib rate control.
     - ✓ Anticoagulation → for AFib & LA clots
     - ✓ SBE prophylaxis → prevent endocarditis.
  2. Surgical therapy → treat the cause:
     - **Percutaneous Ballon Valvulaoplasty** → for Non-calcified, pliable valve. Done by a catheter.
     - Open Commisurotomy – valve repair
     - Mitral Valve Replacement → when a catheter procedure can not be done.

# Mitral Regurgitation

Backflow of blood from the left ventricle into the left atrium, due to inadequate functioning (insufficiency) of the mitral valve. Most commonly from ischemia. Nowadays, it is more common than MS.

## Etiology

- **Alterations of the Leaflets, Commissures, Annulus:**
  - Rheumatic heart disease
  - **MVP**
  - Endocarditis
- **Alterations of LV or LA size and Function:**
  - Papillary Muscle (Ischemic, MI, Myocarditis, DCM)
  - HOCM
  - LV Enlargement
  - Cardiomyopathies (dilated , hypertrophic)
  - **LA Enlargement** from MR –
    - MR begets MR.

If the LV is dilated → the mitral valve will be affected → (MR)

## Pathophysiology

1. A portion of the left ventricular stroke volume is pumped backward into the left atrium instead of forward into the aorta, resulting in increased left atrial pressure and decreased forward cardiac output. (LAv is increased bc of increased LVEDv)
2. Volume overload occurs, increasing preload.
3. Afterload is decreased as the left ventricle empties part of its contents into the relatively low-pressure left atrium.
4. This helps to compensate for the regurgitation by augmenting ejection fraction.
5. Left ventricular dysfunction occurs after prolonged compensation.

## Symptoms

- Fatigue & weakness → bc of the reduced cardiac output.
- Dyspnea & orthopnea → owing to pulmonary venous hypertension occuring as a direct result of mitral regurg. & 2ry to left ventricular failure.
- Right sided HF → in the late stages of the disease & eventually may lead to CHF.
- Mitral valve syndrome (if present)

## Signs

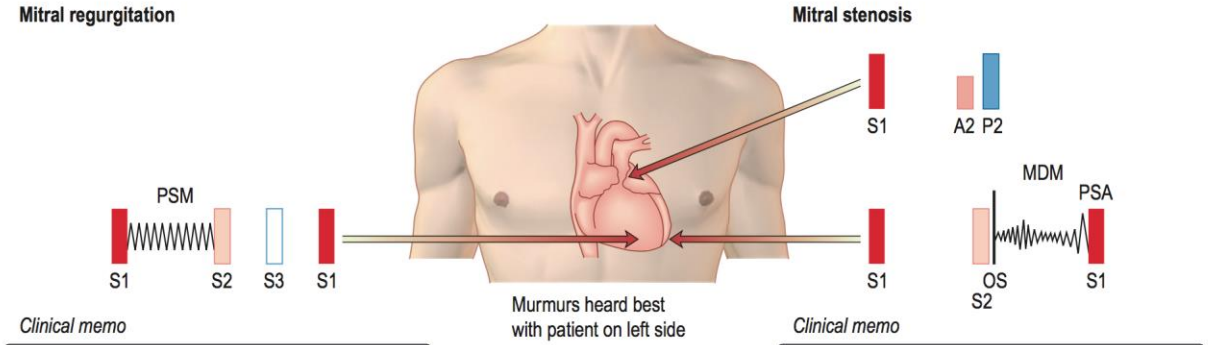
- Laterally displaced (forceful) diffuse apex beat and a systolic thrill.
- Soft first heart sound S1.
- **Pan-systolic murmur**.
- **S3 Gallop** (increased volume during diastole) (gallop= S3 + tachycardia)
- Split S2 (but is obscured by the murmur)
- **Holosystolic apical murmur** radiating to the **axilla** and often accompanied by a thrill.

## Diagnosis

- ECG:
  - Left Atrial Enlargement & LVH.
- Echocardiography (Echo 2D/color doppler) → test of choice.
- Cardiac Cath – helpful, confirmatory, needed if the pt is older – look at the coronaries.

## Treatment

- Treatment of <u>symptomatic</u> mitral regurgitation:
  1. Medical therapy → Diuretics, Vasodilators (ACE inhibitors→ it ↓ afterload) & SBE prophylaxis.
  2. Surgical therapy:
     - MV replacement:
       - Relief of symptoms, MVR survival similar to natural history, Operative mortality 8-10% & 10 yrs survival = 50%.
     - MV repair:
       - Operative mortality 2-4% , 10 yrs survival = 80%, **preservation of mitral apparatus**!!, No risk of thrombotic complication.




*Clinical memo*

**Mitral regurgitation**

| | |
|---|---|
| Pulse: | Sinus rhythm or atrial fibrillation |
| Apex: | Forceful, displaced, systolic thrill |
| Sounds: | Soft S1, S3 |
| Murmurs: | Pansystolic |

*Clinical memo*

**Mitral stenosis**

| | |
|---|---|
| Face: | Mitral facies |
| Pulse: | Atrial fibrillation |
| RV: | Heaving, sustained |
| Apex: | Localized, tapping |
| Sounds: | Loud S1, loud P2 (if pulmonary hypertension), opening snap |
| Murmurs: | Mid-diastolic rumble at apex |

**Figure 14.71 Features associated with mitral regurgitation and mitral stenosis.** A2, aortic component of the second heart sound; MDM, mid-diastolic murmur; OS, opening snap; P2, pulmonary component of the second heart sound (loud with pulmonary hypertension); PSA, presystolic accentuation; PSM, pansystolic murmur; S1, first heart sound; S2, second heart sound; S3, third heart sound.

# Mitral Regurg. – **Mitral valve prolapse**

- Large mitral valve leaflets, an enlarged mitral annulus, abnormally long chordae or disordered papillary muscle contraction.
- Demonstrate **myxomatous degeneration** of the mitral valve leaflets.
- Associated with Marfan's syndrome, thyrotoxicosis, rheumatic or ischaemic heart disease.

| | | | |
|---|---|---|---|
| Pathophysiology | 1. Abnormal mitral apparatus → mitral leaflet prolapse → papillary muscle traction, activation of stretch receptors → papillary muscle & subendocardial ischemia → pain, ventricular arrhythmias. | | |
| Symptoms | ○ **Atypical chest pain** is the most common symptom<br>○ **Palpitations** may be experienced because of the abnormal ventricular contraction or because of the atrial and ventricular arrhythmias.<br>○ **Sudden cardiac death** due to fatal ventricular arrhythmias is a very rare but recognized complication. | Signs | ○ **Mid-systolic click** (most common) → Produced by the sudden prolapse of the valve and the tensing of the chordae tendineae that occurs during systole.<br>○ Late systolic murmur (if associated with MR)<br>→ **Mid-or-late systolic click** |
| Prognosis | ○ Often benign.<br>○ Rare complication:<br>• Endocarditis<br>• Progressive MR<br>• Acute or chronic.<br>• Thromboembolism.<br>• Atrial & ventricular arrhythmias. | | |
| Diagnosis | ❖ Diagnosis by: Echo 2D/Color<br>❖ Treatment:<br>• B-Blockers (for hyperadrenergic symptoms, atypical chest pain & Palpitations)<br>• Aspirin (TIAs without etiology)<br>• SBE Prophylaxis (only if associated with MR)<br>• Severe Symptomatic MR – same as chronic MR | | |

# Aortic Stenosis

It is a chronic progressive disease that produces obstruction to the left ventricular stroke volume leading to symptoms of chest pain, breathlessness, syncope and presyncope and fatigue. It is the most common VHDs.

**Etiology**

- Calcification and degeneration of a normal valve; more common in the **elderly** population. (**the most common cause**) → > 60 yrs.
- Calcification and fibrosis of a congenitally bicuspid aortic valve. → 30-50 yrs.
- Rheumatic valvular disease (3rd most common cause)→ 30-60 yrs.
  - If the aortic valve is affected by the rheumatic fever, the mitral valve is also invariably affected.

**Pathophysiology**

1. Obstructed left ventricular emptying → ↑ left ventricular pressure (**pressure overload**) → compensatory left ventricular hypertrophy (reduce wall stress, reduce vent. Compliance, ↑ LVEDp & ↑ LAp ) Forceful atrial contraction augments filling at the thick, noncompliant ventricle and generates a prominent **$S_4$ gallop** that **elevates the left ventricular end-diastolic pressure**.
2. Left ventricular hypertrophy and high intramyocardial wall tension account for the increased oxygen demands and, along with decreased diastolic coronary blood flow, account for the occurrence of **angina pectoris**.
3. As the myocardium fails, mean left ventricular diastolic pressure increases, and symptoms of **pulmonary congestion** ensue.

**Symptoms**

- **Angina** → imbalance between supply & demand → 5 yrs survival.
  - ↑ LVEDp → leads to ↓ perfusion pressure.
  - Myocardial hypertrophy ↑ demand.
- **Syncope with exertion** → 3 yrs survival.
  - Inability to ↑ cardiac output & meet reduced SVR demands.
- **Congestive heart failure (CHF)** → 2 yrs survival (the worst). (such as dyspnea on exertion, orthopnea, or PND)
  - ↑ LVEDp → ↑ LAp → pulmonary venous congestion.

**Signs**

- In severely AS → low BP, bc of low blood ejected from the ventricle to aorta.
- Pulse:
  - **Pulsus Parvus et Tardus** (narrow pulse pressure) (**Carotid Impulse**) → The carotid pulse is of small volume & slow-rising or plateau in nature. (Slow & late impulses)
  - Sustained Bifid LV impulse (from LVH).
- Auscultation:
  - **Harsh Systolic Ejection Murmur** – late peaking (diamond-shaped, **Crescendo-decrescendo**), usually with thrill, radiates to **carotids**.
  - **Prominent $S_4$ gallop (from LVH)** → it is heard unless co-existing mitral stenosis prevent this.
    - In case there is left atrial fib → no S4 is heard bc there is no atrial contraction.

**Diagnosis**

- ECG:
  - Left ventricular hypertrophy & left atrial enlargement (Bifid & wide p wave)
    - Absence LVH, doesn't rule out aortic stenosis.
  - ECG is helpful but not diagnostic!
- Echocardiography (Echo 2D/color doppler) → test of choice.
- Cardiac Cath → helpful, confirmatory. Needed if the pt is older (look at the coronaries)

**Treatment**

- Treatment of symptomatic aortic stenosis or ↓ LV function:
  1. Medical therapy → treat the symptoms not the cause.
  2. Aortic valve replacement (the only truly effective therapy for AS)
     - Bioprosthetic vs Mechanical AVR.
       - Bioprosthetic given to pt 10-15 yrs, while Mechanical to >30 yrs.

# Aortic Regurgitation

## Etiology

| Acute | Chronic |
|---|---|
| ○ Acute rheumatic fever<br>○ Infective endocarditis<br>○ Dissection of the aorta<br>○ Ruptured sinus of Valsalva aneurysm<br>○ Failure of prosthetic heart valve<br>Acute AR is very bad news!<br>Pt will have acute pulmonary edema immediately!! | ○ Rheumatic heart disease<br>○ Syphilis Arthritides:<br>• Reiter's syndrome, Ankylosing spondylitis, Rheumatoid arthritis<br>○ Hypertension (severe)<br>○ Bicuspid aortic valve<br>○ Aortic endocarditis<br>○ Marfan's syndrome<br>○ Osteogenesis imperfecta |

AR result from either problems with aortic valve or aortic root

- **Abnormalities of the leaflets:**
  - Rheumatic, Bicuspid, Degenerative. - Endocarditis.
- **Dilation of the aortic annulus:**
  - **Aortic aneurysm** \ Dissection. - Inflammatory (connective tissue diseases) (syphilis, Fiant cell Artetitis, Coll Vasc Dis-Ankylosis spondylitis, Reiters) - Inheritable (Marfans, Osteogensis imperfecta)

## Pathophysiology

- **Widened pulse pressure:**
  - Stroke volume increased → (high Systolic BP)
  - Regurgitant volume increased → (Low Diastolic BP)
- Imbalance between myocardial supply & demand:
  - ↓ Diastolic BP → ↓ perfusion pressure → ↓ supply.
  - ↑ LV size (thus ↑ wall stress) → ↑ demand → Those pts may get angina

1. Aortic regurgitation results in a **volume overload** of the left ventricle.
2. The ventricle compensates by increasing its end-diastolic volume according to the Frank-Starling mechanism.
3. The left ventricular dilation is thought to overstretch the myofibrils, leading to less actin–myosin interaction and decreased contractility.
4. In acute severe aortic regurgitation, the left ventricle has not had the opportunity to dilate, its compliance is relatively high, and the aortic regurgitation therefore leads to very high left ventricular end-diastolic pressure.

If mitral regurgitation ensues, the elevated left ventricular diastolic pressure is reflected back to the pulmonary vasculature, and acute pulmonary edema may occur.

## Symptoms

- Pulmonary venous congestion
  - **Dyspnea** on exertion (most common complaint)
- Inadequate cardiac output
  - Fatigue.
  - Diminished exercise tolerance. & Angina pectoris.

## Signs

- **Diastolic decrescendo murmur** is the most typical.
- Hyperdynamic LV apical impulse (bc of ↑ SV)
- Bounding Pulses (widened pulse pressure) "water hammer" pulse, (collapsing pulse)
- **S4, S3 Gallop-advanced AI.** → any pt has ↑ LV pressure → will have ↑ LA pressure → S4.
- Apical Rumble – "Austin Flint Murmur" (low-pitched diastolic rumble due to competing flow anterograde from the LA and retrograde from the aorta) (its sounds is like mitral stenosis)

## Dx

- ECG → left atrial enlargement & LVH.
- Echo 2D/color doppler – **test of choice.**
- Cardiac Cath – helpful, confirmatory, needed if the pt is older – look at the coronaries

## Treatment

❖ Treatment of Asymptomatic Aortic Regurg
- Medical Therapy – treats the symptoms not the cause.
- Serial Check ups with Echos (eval EF, Severity AR)
- SBE Prophylaxis
- Vasodialators (Nifedipine, ACE-I)
- Diuretics

❖ Treatment of Symptomatic Aortic Regurg

✓ Aortic Valve Replacement
- Bioprosthetic vs Mechanical AVR

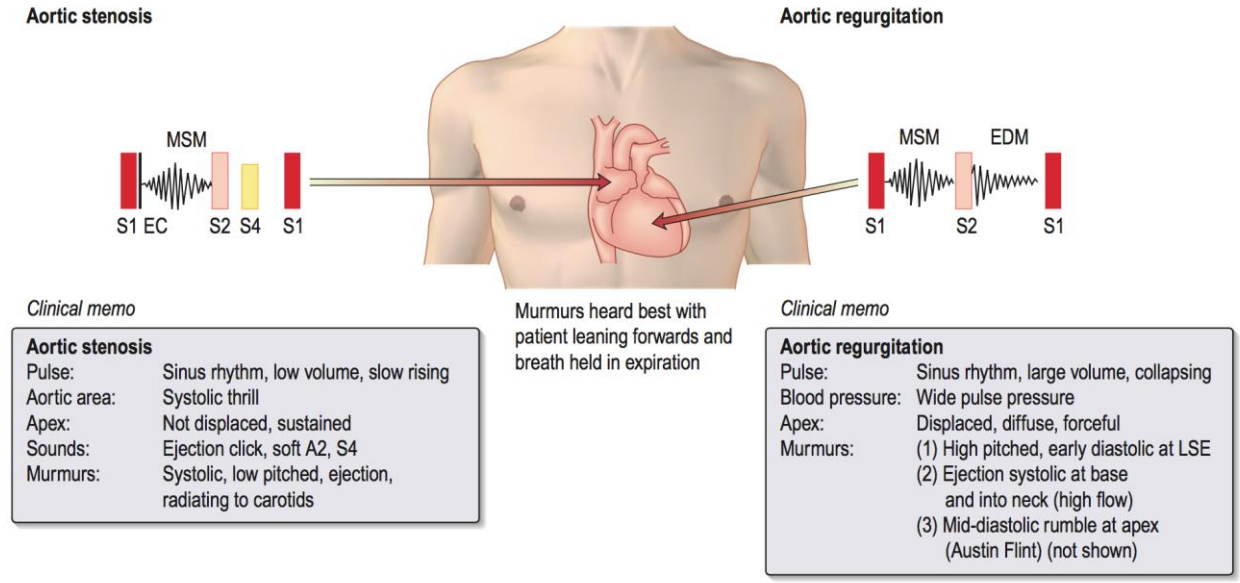



**Figure 14.80 Features of aortic stenosis and aortic regurgitation.** EC, ejection click; EDM, early diastolic murmur; MSM, mid-systolic murmur; S1, first heart sound. LSE, left sternal edge.

# Right sided VHDs:

## Tricuspid valve

### Endocarditis

- **IV drug abusers** or inpt with IVs.
  - Usually come to young pts, bc they insert the drug in their veins → goes to right side of heart → result in tri S or R.

### Carcinoid heart disease

- Tricuspid **stenosis**. (less common)

### Tricuspid Regurgitation

- Common, benign, may be 2ry to **Pulm HTN**.
  - Most of TR cases are 2ry to something else. It is usually a result of some Right side cardiac diseases, rarely seen as a major problem.

## Pulmonary valve

### Pediatrics

- Pulmonary **stenosis**

### Rheumatic HD

- Pulmonary **insufficiency** (regurg.)
  - **Graham steel Murmur**

When PI is caused by RHD, most likely there are other valves affected.

**Right** sided valvular lesions change in intensity with **inspiration**.

**TABLE 7.1 Features of important valve lesions and congenital abnormalities**

| | Site | Timing | Radiation | Character | Accentuation and manoeuvres | Other features |
|---|---|---|---|---|---|---|
| **Aortic regurgitation** | Aortic area | Early diastolic | Lower left sternal edge | Decrescendo | Expiration, patient leaning forwards | Wide pulse pressure, eponymous signs |
| **Aortic stenosis** | Aortic area | Systolic | Carotids | Ejection | Expiration | Separate from heart sounds, slow-rising pulse |
| **Mitral stenosis** | Apex | Middle and late diastolic | — | Low-pitched (use stethoscope bell) | Presystolic accentuation, left lateral position, exercise | Loud S1, opening snap |
| **Mitral regurgitation** | Apex | Pansystolic or middle and late systolic (mitral valve prolapse) | Axilla or left sternal edge | Blowing (MVP) | Longer and louder with Valsalva (MVP) | Parasternal impulse (enlarges left atrium) |
| **Ventricular septal defect** | Lower left sternal edge | Pansystolic | None | Localised | — | Often associated with a thrill |
| **Tricuspid regurgitation** | Lower left and right sternal edge | Pansystolic | — | — | Louder on inspiration | Big *v* waves, pulsatile liver |
| **Hypertrophic cardiomyopathy** | Apex and left sternal edge | Late systolic at left sternal edge, pan-systolic at apex | — | — | Louder with Valsalva, softer with squatting | S4, double-impulse apex beat, jerky carotid pulse |

MVP = mitral valve prolapse.

# MCQs

**1- You see a 57-year-old woman who presents with worsening shortness of breath coupled with decreased exercise tolerance. She had rheumatic fever in her adolescence and suffers from essential hypertension. On examination she has signs which point to a diagnosis of mitral stenosis. Which of the following is not a clinical sign associated with mitral stenosis?**

A. Malarflush
B. Atrial fibrillation
C. Pan-systolic murmur which radiates to axilla
D. Tapping, undisplaced apex beat
E. Right ventricular heave

**2- An asymptomatic 31-year-old woman has been referred for cardiological assessment. After her ECG she was told that she had mitral valve prolapse and would like further information on this condition. Which of the following statements is correct?**

A. Beta-blocker therapy is indicated
B. Angiotensin-converting enzyme (ACE) inhibitor therapy is indicated
C. One or both leaflets of the mitral valve are pushed back into the leftatrium during systole
D. Significant mitral regurgitation will eventually develop
E. Exercise should be restricted

**3- A 49-year-old woman presents with increasing shortness of breath on exertion developing over the past three months. She has no chest pain or cough, and has noticed no ankle swelling. On examination, blood pressure is 158/61mmHg, pulse is regular at 88 beats per minute and there are crackles at both lung bases. There is a decrescendo diastolic murmur at the left sternal edge. What is the most likely diagnosis?**

A. Aortic regurgitation
B. Aortic stenosis
C. Mitral regurgitation
D. Mitral stenosis
E. Tricuspid regurgitation

**4- A 78-year-old woman is admitted with heart failure. The underlying cause is determined to be aortic stenosis. Which sign is most likely to be present?**

A. Pleural effusion on chest x-ray
B. Raised jugular venous pressure (JVP)
C. Bilateral pedal oedema
D. Bibasal crepitations
E. Atrial fibrillation

**5- A patient is admitted with pneumonia. A murmur is heard on examination. What finding points to mitral regurgitation?**

A. Murmur louder on inspiration
B. Murmur louder with patient in left lateral position
C. Murmur louder over the right 2nd intercostal space midclavicular line
D. Corrigan's sign
E. Narrow pulse pressure

**6- Myxomatus degenration of MV, rheumatic fever, infective endocarfitis, calcification of mitral annulus associated with HTN/DM, and hypertrophic cardiomyopathy, can cause which kind of valvular problem?**

A. Mitral stenosis
B. Chronic mitral regurgitation
C. Acute mitral regurgitation
D. Aortic stenosis

**7- Dyspnea in valvular heart disease usually denotes:**

A. Elevated left atrial pressure
B. Elevated left ventricular systolic pressure
C. Elevated right atrial pressure
D. Elevated right ventricular diastolic pressure

**8- What is the most frequent valvulopathy in the elderly?**

A. Aortic insufficiency
B. Mitral regurgitation
C. Mitral valve prolapse
D. Aortic stenosis
E. Mitral stenosis

1- **C**, 2- **C**, 3- **A**, 4- **D**, 5- **B**, 6- **B**, 7- **A**, 8- **D**

# Answers

**1- C**

Malar flush (A), atrial fibrillation (B), a tapping apex beat (D) and right ventricular heave (E), which occurs secondary to pulmonary hypertension, are all clinical signs associated with mitral stenosis. On auscultation of the praecordium, a mid-diastolic murmur (±opening snap, representing a mobile valve) is heard rather than a pan-systolic murmur (C) which is usually heard in mitral regurgitation, tricuspid regurgitation and ventricular septal defects.

**2- C**

There is no indication for ACE inhibitor therapy (B), while beta-blockers (A) may be used for management of arrhythmias if these occur. Mitral regurgitation (D) is unlikely to occur, although it is a possibility. There is no need to limit exercise (E) in an asymptomatic patient. As mentioned elsewhere, endocarditis is a persistent risk, with the need for antibiotic prophylaxis a topic of current debate.

**3- A**

This is a typical clinical scenario for an aortic regurgitation (A), with early cardiac failure. Note the wide pulse pressure, and it is also usual for the pulse to be rapidly collapsing. The only lesion producing a diastolic murmur, among those listed, is of course mitral stenosis (D). No other valve abnormality (B), (C) or (E) produces a wide pulse pressure as seen here, but remember that in older people, almost always over the age of 60, similarly wide or even wider pulse pressures may be noted. This would be due to isolated systolic hypertension, i.e. systolic pressure 140 mmHg and diastolic  90 mmHg.

**4- D**

Aortic stenosis will first result in left ventricular failure as a result of increased ventricular pressure as the ventricle tries to pump blood across a narrowed valve. Initially the pressure load will cause a backlog of blood into the lungs, resulting in pulmonary oedema – the first sign of which will be bibasal crepitations (D) before enough fluid accumulates as pleural effusions visible on chest x-ray (A). Earlier signs of pulmonary oedema include upper lobe blood diversion and Kerley B lines as fluid infiltrates the interstitium. If the backlog continues back into the right heart, eventually signs of right-sided heart failure will be evident including raised JVP (B) and bilateral pedal oedema (C). Atrial fibrillation (E) may coexist with aortic stenosis, however it is more commonly associated as a result of mitral stenosis as the enlarged atrium disrupts the normal electrical pathways.

**5- B**

A murmur heard loudest on inspiration (A) points to a right-sided valve lesion. The right intercostal space midclavicular line (C) is the anatomical landmark for the aortic valve. The mitral area is over the apex. A murmur louder with the patient in the left lateral position (B) (as opposed to leaning forward) is associated with mitral lesions. If heard, you should determine whether the murmur radiates to the axilla. Corrigan's sign (D) (visibly exaggerated pulsating carotids) is one of the many signs of a hyperdynamic circulation associated with aortic regurgitation (including de Mussets, Traubes, Quinkes, Duroziez and a whole host of others). A narrow pulse pressure (E) is a sign of aortic stenosis.