

CARDIOVASCULAR PATHOLOGY

VALVULAR HEART DISEASES | OVERVIEW

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[Valvular Heart Diseases | Overview](#)

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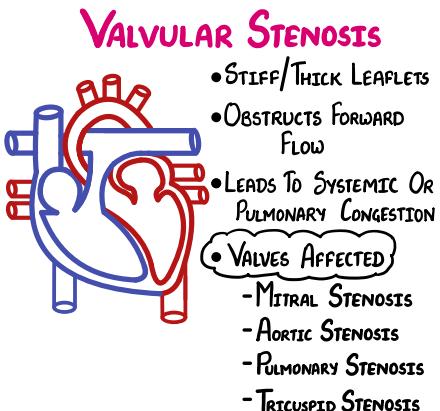
- (A) DIAGNOSTIC INVESTIGATIONS
- (B) TREATMENT OF VALVULAR HEART FAILURE

I) INTRODUCTION

(A) DEFINITIONS

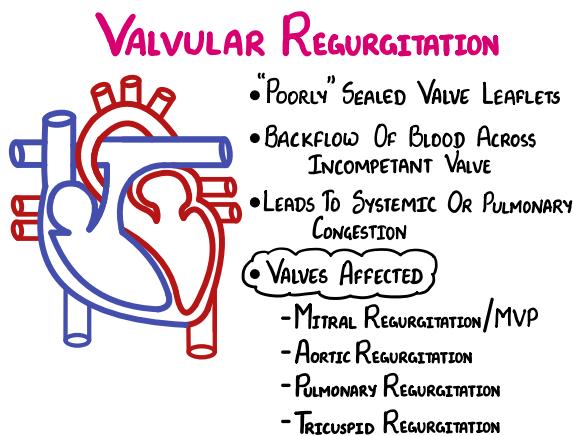
(1) Valvular stenosis

Thickening of valve, that obstructs the forward flow of blood from atria to ventricles/ ventricles to arteries



(2) Valvular regurgitation

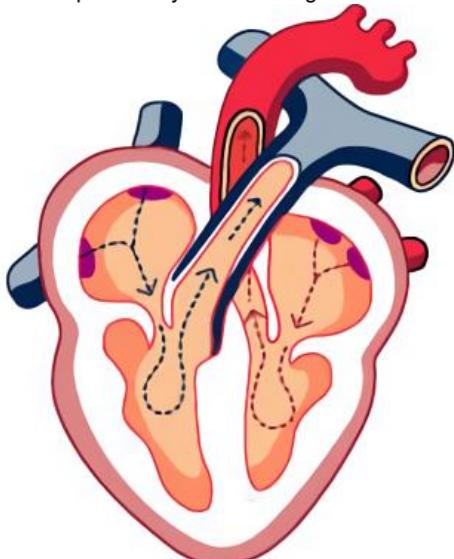
Poor seal between the leaflets of the valve, causing backflow of blood from ventricles into atria/ arteries into ventricles



(B) CONSEQUENCES OF VALVULAR STENOSIS AND REGURGITATION

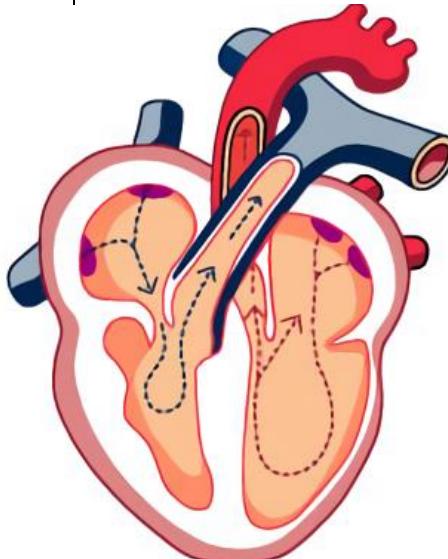
(1) Left side of heart

If blood isn't pumped out through aorta
→ Blood backs up into the pulmonary veins
→ Leads to pulmonary venous congestion



(2) Right side of heart

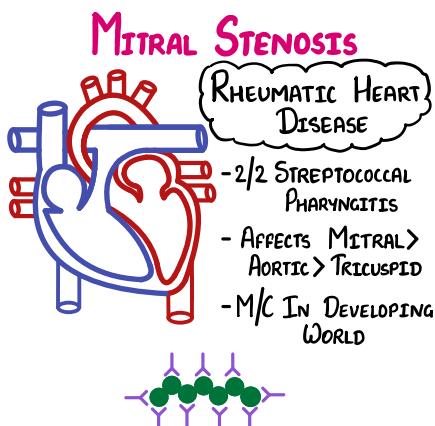
If blood isn't pumped out through pulmonary artery
→ Blood backs up into the vena cavae
→ Leads to systemic venous congestion



(C) VALVULAR STENOSIS

(1) Mitral Stenosis

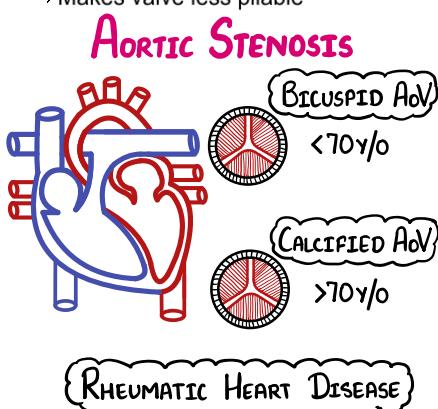
- Most common cause is **Rheumatic Heart Disease**
 - Develops from **streptococcal** infection of the throat
 - Body produces antibodies against the M antigen of Streptococcus
 - M antigen** resembles **cardiac myosin** of humans
 - Therefore, the antibodies produced against M antigen also attack the cardiac cells, causing fibrosis of the heart valves
 - This leads to narrowing of valve lumen, and obstruction to forward flow
- Predilection of valves: **Mitral valve > Aortic > Tricuspid**



(2) Aortic Stenosis

Can be due to 2 causes:

- Bicuspid aortic valve**
 - Normally aortic valve has 3 cusps
 - Congenital fusion of 2 of the cusps, resulting in only 2 cusps out of 3
 - Makes it difficult for valve to open
 - Seen in younger individuals (**<70 years**)
- Calcified aortic valve**
 - Risk factors: age (**>70 years**), hypertension, hypercholesterolemia, end stage renal disease
 - Increases calcium deposition and fibrosis of cusps
 - Makes valve less pliable



(3) Pulmonary Stenosis

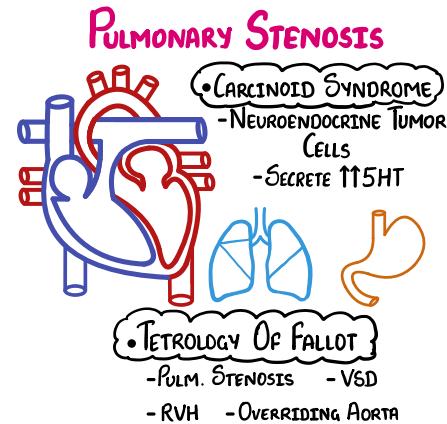
Often seen in:

(i) Carcinoid syndrome

- Presentation:
 - Diarrhea
 - Rash
 - Wheezing due to bronchospasm
 - Flushing of face
 - Pulmonary stenosis
- Neuroendocrine tumor**
 - Starts in GIT and spreads to liver and right side of heart
 - Produces large amounts of 5-hydroxy tryptamine (5-HT), which is a precursor of serotonin
 - 5-HT induces **fibrosis of pulmonary valve**

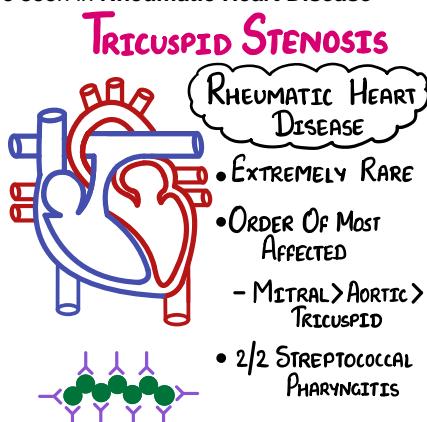
(ii) Tetralogy of Fallot

- VENTRICULAR SEPTAL DEFECT
- RIGHT VENTRICULAR HYPERTROPHY
- OVERRIDING AORTA
- PULMONARY STENOSIS



(4) Tricuspid Stenosis

- Very rare
- Can be seen in **Rheumatic Heart Disease**



(D) VALVULAR REGURGITATION

(1) Mitral Regurgitation and Mitral Valve Prolapse

Types of Mitral Regurgitation:

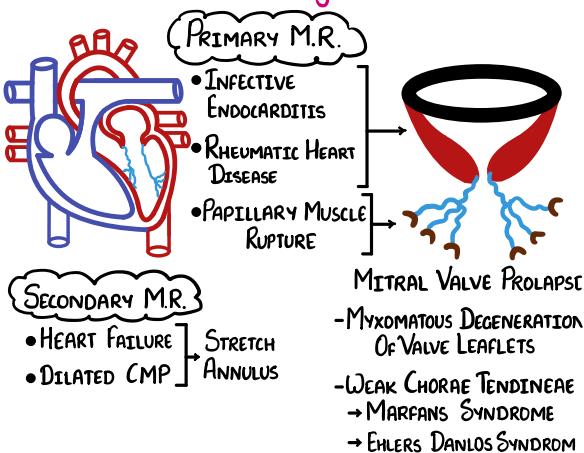
(i) Primary MR

- Involves parts of the valve apparatus
 - Valve leaflets
 - Chordae tendinae
 - Papillary muscles
- Seen in:
 - (a) Infective endocarditis
 - Destroys the leaflets of the valve
 - (b) Severe Rheumatic Heart Disease
 - Fibrosis of valves, valves become less pliable and very stiff, can't fully shut close
 - (c) Papillary muscle rupture
 - In **Myocardial Infarction (MI)**, when there is death of myocytes (due to ischemia) near the papillary muscles, they rupture off, causing the chordae tendinae to become loose
 - (d) Mitral Valve Prolapse
 - Affects leaflets, chordae tendinae and attachments to the papillary muscle
 - Instead of closing during ventricular systole, the valve bows in, creating space for a regurgitation jet
 - Pathology:
 - (i) *Myxomatous degeneration of valve*
 - Core of leaflet is very weak
 - (ii) *Weak chordae tendinae*
 - Associated with connective tissue disorders:
 - Marfan's syndrome
 - Ehler-Danlos Syndrome
 - Osteogenesis imperfecta

(ii) Secondary MR

- Involves annulus of the valve
- Annulus is stretched out
 - This increases the distance between the leaflets
- Seen in:
 - (a) Heart failure
 - (b) Dilated Cardiomyopathy

MITRAL REGURGITATION & M.VALE PROLAPSE



(2) Aortic Regurgitation

- Regurgitation jet from aorta into ventricles
- Causes:

(i) Valve damage

- (a) Bicuspid aortic valve
 - More susceptible to
 - Infective Endocarditis
 - Calcifications
- (b) Infective endocarditis
- (c) Rheumatic Heart Disease

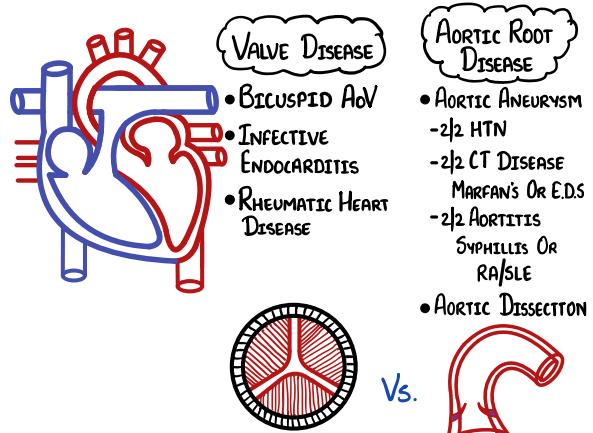
(ii) Aortic Root Disease

- Dilation of root of aorta
- Causes:
 - (a) Aortic aneurysm
 - (i) Hypertension
 - (ii) Connective tissue disease
 - Weak walls, more prone to dilate
 - **Marfans, Ehler-Danlos Syndrome**
 - (iii) Inflammation of aorta
 - Syphilis
 - Ankylosing spondylitis
 - Rheumatoid arthritis
 - Systemic Lupus Erythematosus (SLE)

(b) Aortic Dissection

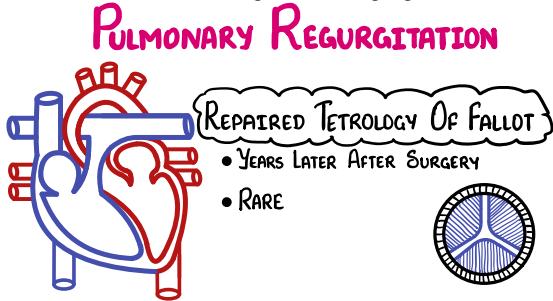
- Blood "dissects" through the tunica intima, creating and flowing through a false lumen
- The jet of blood flows down and compresses the annulus
- This distorts the shape of leaflets, weakening them

AORTIC REGURGITATION



(3) Pulmonary Regurgitation

- Rare
- Seen after procedures done to repair Tetralogy of Fallot: Balloon valvuloplasty
 - Balloon inserted into fibrotic valve and expanded, to repair the pulmonary stenosis
 - Too much stretching causes regurgitation



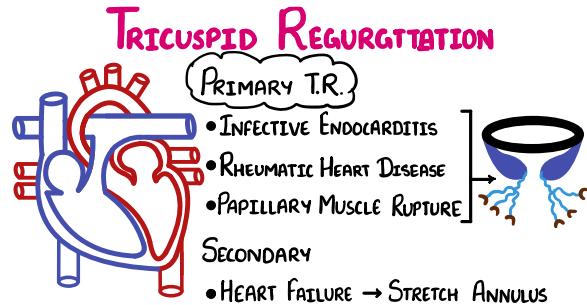
(4) Tricuspid regurgitation

(i) Primary TR

- Infective endocarditis
- Rheumatic Heart Disease
- Carcinoid Syndrome
- Rupture of papillary muscles

(ii) Secondary TR

- Right sided heart failure
 - Stretch out the annulus



II) CLINICAL FEATURES

(A) MITRAL STENOSIS

- Fibrotic, sclerotic, calcified valve obstructing the blood flow from the left atrium into the left ventricle

(1) Pathophysiology

- The build-up in left atrial pressure (LAP) results in volume overload (Figure 1)
- Volume overload or increase in the left atrial volume (LAV) leads to dilatation of the left atrium
- High pressure in the left atrium is relieved by allowing the blood to enter the pulmonary veins resulting in:

(i) Pulmonary venous congestion with edema

(ii) Pulmonary hypertension

- Because right ventricle has to pump against a much higher pressure in the pulmonary circulation, this can eventually lead to **RIGHT-SIDED HEART FAILURE**

(iii) Atrial fibrillation with associated risk for mural thrombi

- Since the mitral valve is stenotic, there is less blood entering the left ventricle
- Less blood exits the aorta but not enough to cause hypotension
 - Exception is when the patient has atrial fibrillation

(a) Risk for systemic hypotension

- When the pressure in atria increases, the atria gets thicker → contracts hard to push blood down (**atrial kick**)
- However, there is **loss of atrial kick in AF** → ↓ stroke volume → ↓ cardiac output → **HYPOTENSION**

(b) Risk for mural thrombi

- Since there is an obstructive valve, there is increased blood volume in the left atrium
- To accommodate the increased volume, left atrium dilates
- Myocardial pacemaker cells stretch out and gets agitated
- Ectopic foci and reentrant circuits** are created resulting in:
 - Increased risk of **ATRIAL ARRHYTHMIA / ATRIAL FLUTTER**
 - Increased blood stasis → Increased **EMBOLIZATION**
 - May eventually lead to **STROKE**

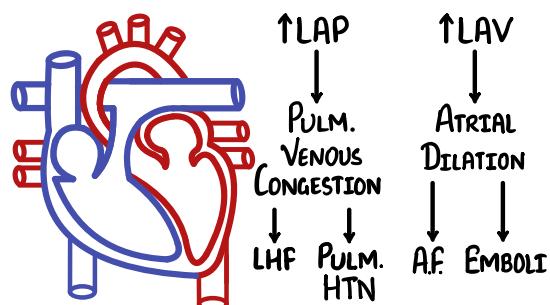


Figure 1. Pathophysiology of Mitral Stenosis



(3) Clinical Features

(i) Signs and Symptoms

- (a) Shortness of breath / Dyspnea
- (b) Orthopnea
- (c) Paroxysmal nocturnal dyspnea
- (d) Palpitations
- (e) Visible AF in EKG

(ii) Pressure Curve

(a) Left Atrial Pressure (LAP)

- o During S1, there is **rise in LAP**
- o When ventricles start to squeeze and contract, they shut the mitral valve and the tricuspid valve close.
- o The closure of the atrioventricular valves corresponds to the **FIRST HEART SOUND (S1)**

(b) Left Ventricular Pressure

- o During systole, there is **rise in LVP**

Systole is the time period between S1 and S2

(c) Aortic Pressure

- As the left ventricle ejects blood into the aorta, **aortic pressure rises**.
- The “blip” in the aortic pressure tracing occurs after closure of the aortic valve. This is called the **DICROTIC NOTCH OR INCISURA**.
- Around the time dicrotic notch occurs is the closure of the semilunar valves (aortic and pulmonic) correspond to the **SECOND HEART SOUND (S2)**
- When they are shut, the AV valves should open because the atrial pressure is higher than the ventricular pressure

(i) In Mitral Stenosis

- Since it is difficult to snap open the sclerotic and fibrotic valve in MS, what is heard is an **OPENING SNAP**
- Blood will still try to squeeze blood into the ventricles through the narrow mitral valve
 - o Initially, when the mitral valve snaps open a lot of blood rushes through it and then less and less blood
 - o Hence, the **intensity of the heart sound is DECREASING**

When looking at the pressure curves, try to correlate it with the heart sounds (S1 and S2).

(iii) Murmurs (Figure 2)

- (a) Diastolic murmur (after S2)
 - This is the beginning of the heart murmur in MS
 - During this time, there is the closure of the semilunar valves (S2) while the AV valves open
- (b) Starts with an opening snap
 - Due to the opening of the stiff mitral valve
- (c) Followed by a diastolic decrescendo rumble
 - Due to the gradual decrease in the volume of blood passing through the stenotic mitral valve

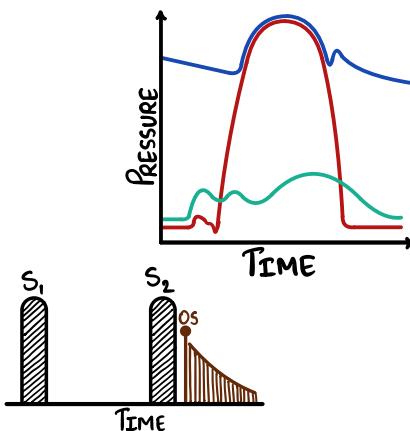


Figure 2. Abnormal heart sounds in mitral stenosis. Pressure curve for **aorta**, pressure curve for the **left ventricle**, pressure curve for **left atrium**

(iv) Physical Exam (Figure 3)

- (a) Heard at 5th left intercostal space midclavicular line
 - Positioning the patient in left lateral decubitus will accentuate the abnormal heart sound
- (b) No radiation

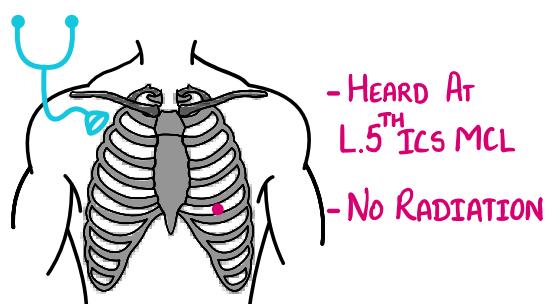


Figure 3. Auscultation of Abnormal heart murmurs in mitral stenosis



(B) MITRAL REGURGITATION

- Poorly sealed mitral valve
- **Reflux of blood from the left ventricle into the left atrium** during systole (Figure 4)
- Usually arises as a **complication of mitral valve prolapse** [Sattar]
- Other causes include [Sattar]
 - Left ventricular dilatation
 - Infective endocarditis
 - Acute rheumatic heart disease
 - Papillary muscle rupture after a myocardial infarction

(1) Pathophysiology

(i) Pulmonary venous congestion and edema

- The reflux of blood results in the increase in the pressure of left atrium
- *Like in MS*, blood is pushed back to the pulmonary venous circulation

(ii) Pulmonary hypertension and eventually right-sided heart failure

- Mechanism is the same as *MS*

(iii) Hypotension

- Since some of the blood are redirected into the pulmonary circulation, **less blood goes to the left ventricle**.
- As a result, there will be less stroke volume
→ ↓ cardiac output → **HYPOTENSION**
- Common in patients with **acute mitral regurgitation**

(iv) Cardiomegaly

- Although some of the blood are redirected into the pulmonary circulation, the left ventricle continuously receive blood from the left atrium
- Over time, there will be **increase in left ventricular end-diastolic volume (LVEDV)** causing it to dilate → **CARDIOMEGLY**
- In these patients, MR patients may present as asymptomatic (i.e. less hypotension, less pulmonary edema) because of the compensation of the left ventricle

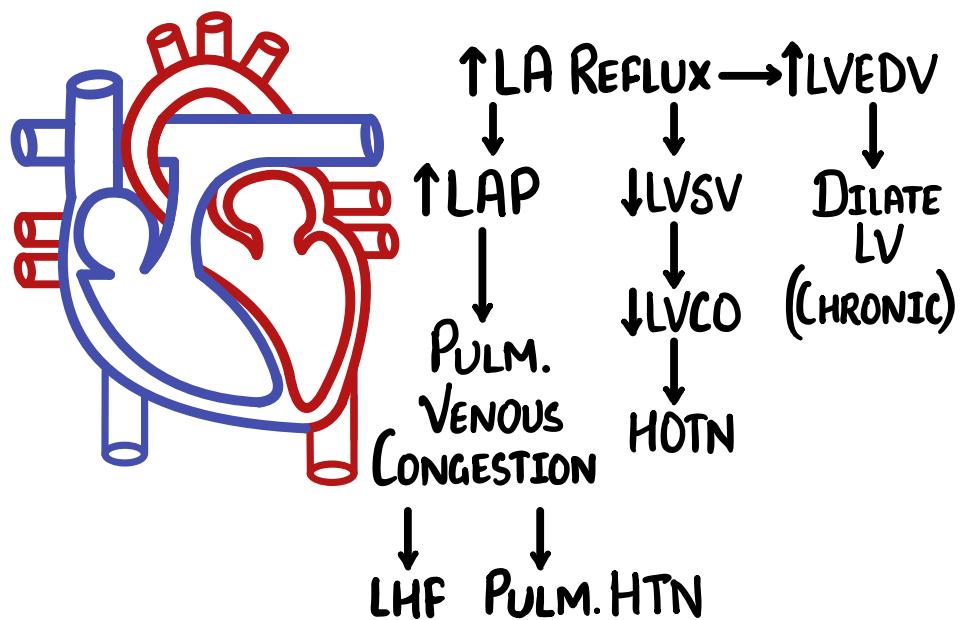


Figure 4. Pathophysiology of Mitral Regurgitation



(3) Clinical Features

(i) Pressure Curve

(a) Left Atrial Pressure

- When the left ventricular pressure goes above the left atrial pressure, the AV (mitral and tricuspid) valves close.
- Recall: The closure of the AV valves corresponds to the first heart sound (S1)

(i) In Mitral Regurgitation

- Some of the blood that the left ventricle tries to push into the aorta regurgitates into the left atrium
- Left atrium gets filled with blood, thus **left atrial pressure increases**

(b) Left Ventricular Pressure

- During systole, there is **rise in LVP**

(c) Aortic Pressure

- As the left ventricle ejects blood into the aorta, **aortic pressure rises**.

(ii) Murmurs (Figure 5)

(a) Holocystolic blowing murmur

- During systole in MR, regurgitation takes place through the mitral valve.
- The turbulence of blood flow causes the holocystolic blowing murmur
 - HOLOSYSTOLIC** means **throughout the systole** (from the onset of S1 until the onset of S2)
 - Unlike MS, the **intensity of the murmur is the same or slight crescendo**.
- During S2, the aortic and pulmonary valve close.
- Meanwhile the AV valves open

(b) Mid-systolic click

- Heard if it occurs as a complication mitral valve prolapse
- When the ventricles contract, the AV valves should close.
- In **MVP**, the **mitral valve is weak or degenerated**.
 - As a result, there is **bowing or ballooning** of the mitral valve into the left atrium during systole
 - The bowing creates the mid-systolic ejection click

(c) Followed by mitral regurgitation murmur

- When the mitral valve leaflets are bowed, a narrow space is created causing blood to regurgitate into the left atrium → **MITRAL REGURGITATION MURMUR**

If only holocystolic blowing murmur is heard, this is mitral regurgitation. But when a mid-systolic click followed by a regurgitation murmur is heard, there might also be a mitral valve prolapse.

(iii) Physical Exam (Figure 5)

(a) Heard at 5th left intercostal space

midclavicular line

- Positioning the patient in left lateral decubitus will accentuate the abnormal heart sound

(b) Radiates to the axilla

- Ejecting the blood from the left ventricle back into the left atria = going to the axilla

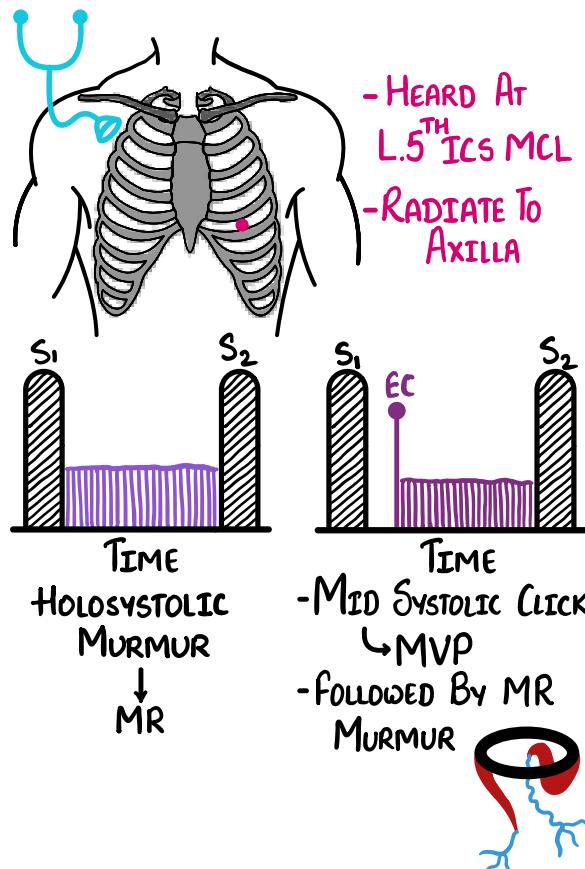
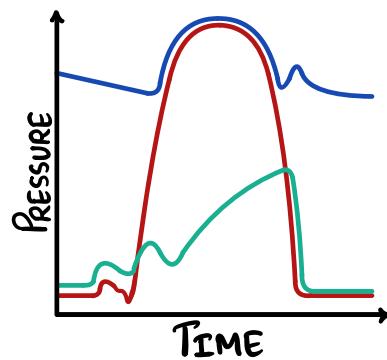
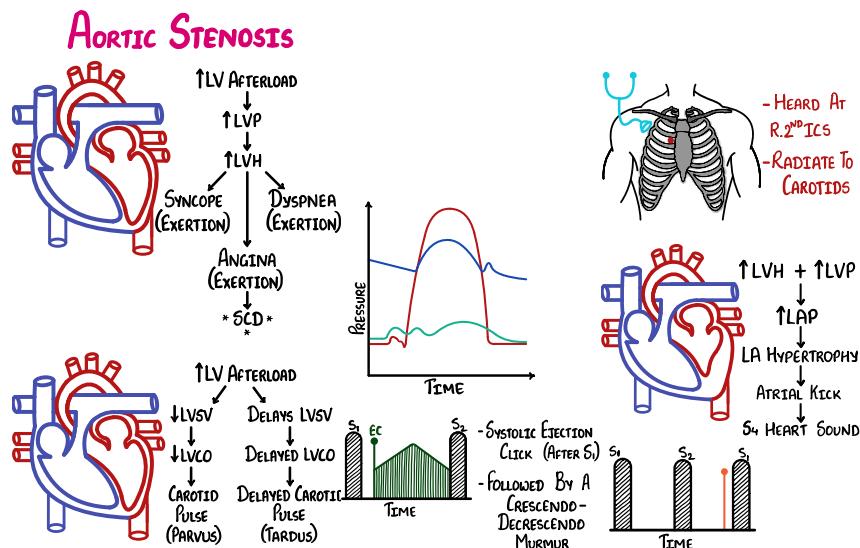


Figure 5. Abnormal heart sounds in mitral regurgitation and its auscultation. Pressure curve for **aorta**, pressure curve for the **left ventricle**, pressure curve for **left atrium**



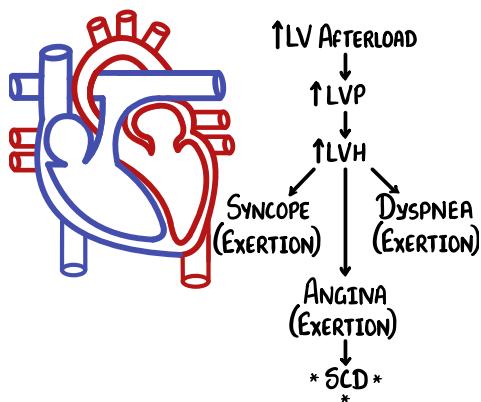
(C) AORTIC STENOSIS



- Remember there's some type of obstruction of blood flow
 - Aortic valve is very stenotic, fibrotic, immobile
 - It doesn't want to open → doesn't allow blood flow to be able to exit
 - Causes obstruction of blood flow to go from left atrium to the aorta

(1) High afterload → concentric hypertrophy of left ventricle

- Because of the valve is so stenotic, fibrotic
 - It's very difficult to get blood out of the left ventricle
 - This is equivalent to say that there's higher afterload**
 - Harder for the left ventricle to be able to eject the amount of blood into the aorta
- Decreased diameter → making the resistance to blood flow
 - Causing the pressure to be higher across the valve
 - The left ventricle has to work harder to push as much blood out
- Afterload is amount of work the left ventricle has to do to overcome the pressure or resistance in the aorta
- If the afterload is high → **the pressure that's going to develop within the left ventricle is going to increase**
 - The **left ventricle undergoes concentric hypertrophy**
 - Increased size and gets really thick



(i) Symptoms

Syncope

- When we have difficulty pushing blood out of the left ventricle to the brain
 - Less amount of blood (perfusion) that's getting into the carotid circulation into the brain**
 - Leads to syncope**
- In other words, loss of cerebral perfusion for a small period of time

Angina

- The ventricles are really thick → they're going to have **higher demand**
 - We're going to **need more blood and O₂ to supply the muscle**
- We're not pushing much blood because of the narrow, stenotic valve
 - Lower the coronary perfusion → leads to anginal chest pain**

Worst scenario

- So little blood flow to the muscle
 - Significant ischemia
 - Causes fatal arrhythmia
 - Causes them to go into **sudden cardiac death**

Dyspnea on exertion

- Remember we're having difficulty being able to deliver blood to the actual systemic circulation
 - Where it helps us to be able to move and perform all the certain types of activities
 - We can have very short of breath with exertion

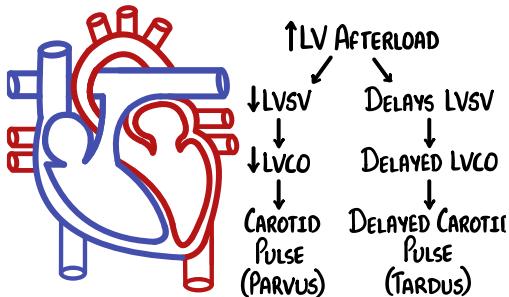
- Mnemonic: **SAD**
 - S**yncope
 - A**ngina
 - D**ysspnea

- These happens primarily in patients with exertional activities**



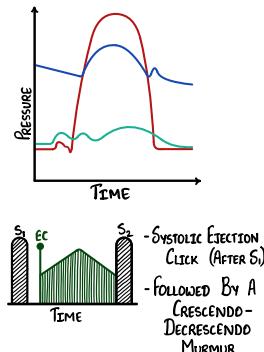
(2) High afterload → decreased stroke volume → parvus

- The ventricle is under a lot of afterload
 - Lots of resistance that we have to overcome to push blood out
- Afterload increases → stroke volume decreases**
 - Less blood getting out into the aorta → **less blood filling the carotid**
 - Causes the carotid pulse not to be very strong
 - Diminished or decreased intensity of the carotid pulse → parvus**



Murmur

- Graph**
 - Blue = aortic pressure
 - Red = left ventricular pressure
 - Green = left atrial pressure
- We're looking for the comparison between left ventricular pressure vs aortic pressure
- Refer to minute [46:21 – 49:27](#) of the lecture video for better visualization



Underfilling of the aorta

- Left ventricle is going to work really hard to push as much blood into the aorta
 - But **underfilling of the aorta** happens
 - Because the afterload is really high → decreases stroke volume**
- The aorta should be equally filled**
 - The pressure that the left ventricle exerts should equally distributed into the aorta
 - But because of the stenotic valve, the pressure is not equally distributed**
- If that happens
 - We have the spaces in the curve that's giving an interesting type of murmur
- Remember
 - S₁ is the onset of when the ventricular pressure overcomes the atrial pressure
 - Closing the tricuspid valve and mitral valve
 - Opening the aortic valve and the pulmonary semilunar valve
 - Dicrotic notch → closure of the aortic valve and pulmonary semilunar valve → begin S₂
- If the aortic valve is supposed to open
 - We shouldn't hear any sounds between the aortic valve opens and closes**
 - Nor we shouldn't be able to hear the blood spurting through between the systole

(3) High afterload → delayed ventricular contraction → tardis

- The ventricle has to generate a lot of pressure** to push blood across the stenotic valve
 - Takes a little bit more time**
- So, this delays the amount of blood that's getting out of the left ventricle into the aorta**
 - Less blood is getting into the aorta** due to delayed left ventricular cardiac output
 - Less blood that is actually stretching the carotids** that induce pulse
 - The pulse that's taking a long time and coming later → tardis**

- Combination of decreased or diminished carotid pulse and pulse coming later** is called **parvus et tardis**
 - Common **pathognomonic finding in aortic stenosis**

Early systolic click + crescendo & decrescendo murmur

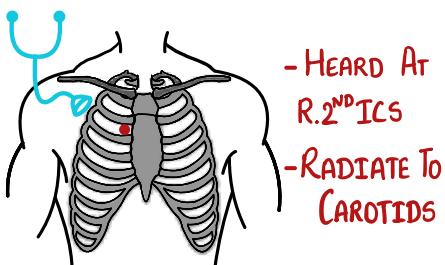
- But whenever the left ventricle is ejecting
 - It so difficult to push blood through the stenotic valve
 - Makes a little "clicking" sound**
 - Because it bows the valve a little bit and then we shoot a ton of blood out
- Look at the curve
 - Starting off with less blood flowing through
 - Then more and more blood is flowing through → then less and less
 - This configuration is called
 - Crescendo**
 - Because it's **increasing in intensity**
 - And then **decrescendo**
 - Because it's **decreasing in intensity**

Remember the murmur begins right after S₁ → systolic murmur

- Gives a little ejection click
- After that we have a murmur which is
 - Crescendo and decrescendo fashion



Auscultation location

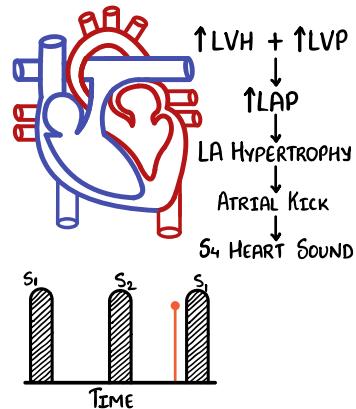


- We have this patient that has early systolic ejection click with crescendo decrescendo murmur
 - We think it's aortic stenosis
 - We would listen this in **right 2nd intercostal space parasternal border**

Murmur radiation

- We want to listen whether the murmur radiates?
- Generally, in this harsh type of murmur
 - We want to listen the radiation
 - Particularly into the carotids
 - Usually the radiation is the direction which the blood is flowing**
 - This murmur is harsher in comparison of regurgitation
 - Regurgitation is more of a blowing musical type of murmur
- For example in
 - Mitral regurgitation**
 - Flowing from **left ventricle into the left atria toward the axilla**
 - Aortic stenosis**
 - Left ventricle into the carotids**
 - We should have that **radiation of that murmur in the carotid**
- Big thing to think about
 - To differentiate this between hypertrophic obstructive cardiomyopathy
- Refer to the minute 50:27 – 50:47 of the lecture video for the aortic stenosis murmur

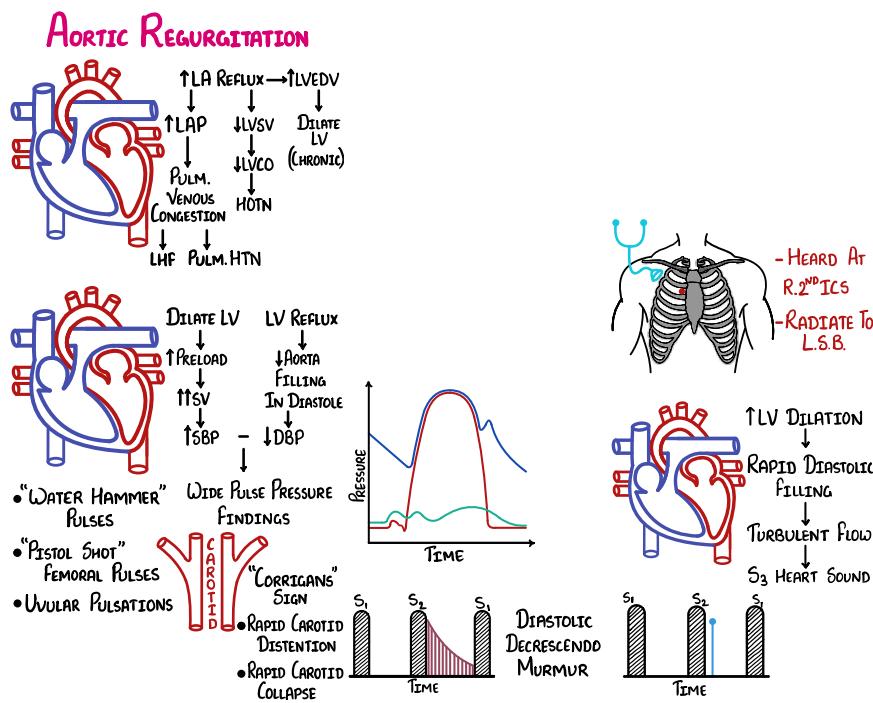
S₄ heart sound



- Important for the boards
- Whenever the left ventricle is getting thicker because of the concentric hypertrophy**
 - We **decrease the diameter of the lumen** of the left ventricle
 - Increase the pressure inside of the cavity**
- High left ventricular pressure** → it's so difficult for left atrium to push blood
 - Left atrium starts thickening up → **undergoes left atrial hypertrophy**
 - To accommodate the high pressure in the left ventricle
- What happens is **during diastole**
 - When the blood is supposed to be filling into the heart, it does in 3 phases
 - Rapid diastolic filling (early phase)**
 - The blood was in the atria fills down
 - Mid-diastolic filling**
 - Blood within the superior and inferior vena cava just moving into the atrium to the ventricles
 - Late-diastolic filling**
 - When the atria contract and push down the blood into the ventricles
 - When the left atrium decides to contract
 - It's going to really squeeze the blood down into the left ventricle → **atrial kick**
 - Way louder than usual
 - Occurs in late diastole right before S₁**
 - Called S₄ heart sound**
 - Refer to the minute 52:59 – 53:08 of the lecture video for the S₄ heart sound



(D) AORTIC REGURGITATION



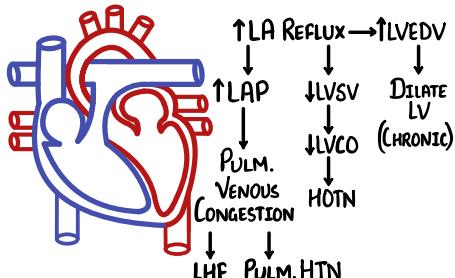
- We have poorly-sealed aortic valve
 - Blood is supposed to be going from left ventricle into the aorta
 - Blood should not be going back from the aorta to the left ventricle during diastole
- But, we have damaged valve or aortic root dilation/disease
 - Causing regurgitation of blood
 - Flow back from the aorta into the left ventricle
 - There's left ventricle regurgitation or reflux

Backflow from left ventricle → left atrium

- When we reflux the blood back into the left ventricle
 - The blood accumulates in the left ventricle
- This will cause backflow from the left ventricle into the left atrium → **increases left atrial pressure**

Backflow from left atrium → pulmonary vein → pulmonary artery

- Remember whenever the pressure is higher in an area, it likes to go to the area of lower pressure
 - Pushes some of the blood to the pulmonary vein → causes **pulmonary venous congestion**
- We may get features of left heart failure
 - Pulmonary edema
- If the pressure in the pulmonary artery starts building up from all of these congestions
 - It can cause **pulmonary hypertension**
 - Which can subsequently lead to right-sided heart failure



Hypotension → cardiogenic shock

- Every time we reflux the blood that was supposed to be in aorta which contributes to stroke volume and cardiac output
 - **Stroke volume drops**
- Example
 - We want to push out 70mL of blood into the aorta → stroke volume
 - 40mL stays in order
 - 30mL of it refluxes back down into the left ventricle
 - True stroke volume: 40mL
- If we drop the left ventricular stroke volume
 - Less blood is filling the aorta
 - **Drops cardiac output**
 - Drops blood pressure in the actual systemic circulation (**hypotension**)
 - Sometimes to the point of **cardiogenic shock**
 - Especially if it's acute
- Acute mitral regurgitation can develop
 - Acute pulmonary edema
 - Acute hypotension with cardiogenic shock
 - **Same thing can happen in acute aortic regurgitation**

Left ventricle overfilling → pulmonary edema

- Just like mitral regurgitation
 - If the left ventricle dilates because we have all of the blood → **overfilling**
 - **Causes pulmonary edema**



Chronic aortic regurgitation → left ventricular dilation to accommodate blood

- If the left ventricle dilates to accommodate all of this blood
 - Less pulmonary venous congestion → less hypotension
 - This will happen in chronic type of cases

Chronic vs acute cases

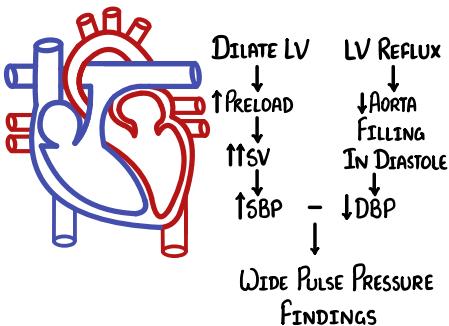
• Chronic cases

- They are usually more asymptomatic
 - No significant pulmonary edema
 - No significant hypotension

• Acute cases

- They're likely going to have pulmonary edema, hypotension, murmur

Increased preload → increased stroke volume in chronic aortic regurgitation



- Go back to patient with chronic aortic regurgitation
- When they accumulate of the volume
 - They don't want to keep backing it up into the left atrium → pulmonary circulation
- They dilate their left ventricle
 - Making it bigger → accommodate more volume
 - More volume in the left ventricle → increased preload
 - Remember preload is volume in the left ventricle before it contracts
- What is the relationship between preload and stroke volume assuming that contractility is normal and afterload is constant?
 - Increase stroke volume

Clinical signs → loves to be asked in exams

(i) Corrigan's pulse

- Take a look at the carotids
- Whenever the ventricles contract
 - They're smashing blood into the aorta and then filling the carotids quickly during systole
- During systole
 - They have very quick distension of the carotid artery
- During diastole
 - What happens to the aorta is the pressure drops → gets underfilled
 - The blood that was in carotid circulation gets yanked back into the aorta → left ventricle
 - This causes sudden drop or collapse of the carotid
- Sudden rise and sudden collapse of the carotid → Corrigan's pulse

Increased systolic blood pressure

- Increase preload → increase stroke volume → push more blood out into the aorta
 - Some may reflux back down
 - But we're pushing more volume of blood into the aorta
- When we push blood into the aorta
 - We fill the aorta
 - **Systolic blood pressure will go up**

Decreased diastolic blood pressure

- During diastole
 - **Aorta accommodates that volume of blood and then it recoils**
- When it recoils
 - In normal patients, it will shut the aortic valve and no blood will go back in
 - But in this patient, **they have damaged aortic valve or aortic root disease**
 - This doesn't happen
- Less aortic filling during diastole → drops diastolic blood pressure
- What is it called when we take **systolic blood pressure minus diastolic blood pressure? Pulse pressure**
 - Pulse pressure in this patient is really wide
 - Because the **systolic blood pressure is so high**
 - Because the left ventricle is slamming blood out into the aorta
 - Aorta is getting filled during systole
 - During diastole, because of that incompetent valve
 - Blood is slamming back into the left ventricle
 - **Dropping their aortic pressure during diastole → low diastolic pressure**
- Exaggerated example
 - Blood pressure of 220/40mmHg
 - Insane difference in comparison to someone who has 120/80mmHg
 - This could be one particular finding

(ii) Water hammer pulse

- We take and fill their radial artery
 - It's blasting rapidly (the distension and collapse)
 - And then we lift their arm up
 - We'll see the accentuated rapid distension and collapse of the radial artery

(iii) Pistol shot pulse

- We'll feel like a "boom boom" across the femoral artery
 - Due to the systolic and diastolic pressure differences

(iv) Uvular pulsation or Muller sign

- We can see the uvula
 - **Punching bag in the back of throat kind of pulsating**

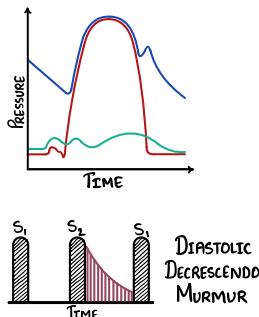
(v) Other signs

- Quincke signs
- Durozies sign



Murmur

- Graph
 - Blue = aortic pressure
 - Red = left ventricular pressure
 - Green = left atrial pressure
- We're looking for the comparison between left ventricular pressure vs aortic pressure
- Refer to minute 1:00:38 – 1:03:15 of the lecture video for better visualization



- Whenever the left ventricular pressure overcomes the left atrial pressure (green)
 - It will shut the tricuspid and mitral valve close → S₁
- All the way from S₁ to S₂ → ventricular systole
 - The aortic valve is opening well and push blood out

The problem during diastole

- We have dicrotic notch
 - It's supposed to signal the sign of closure of the aortic valve
 - But the aortic valve is not closing very well**
 - We have regurgitation jets flow in

Diastolic decrescendo murmur

- During ventricular diastole
 - The ventricles are supposed to get filled by the atria
 - Left atrium → left ventricle
 - Right atrium → right ventricle
- But now the aorta is filling the left ventricle because the aortic valve is incompetent
 - Not having a good seal
 - Dropping the aortic pressure significantly
- We have big difference in the curve
 - Where the intensity looks like it's decreasing as we go through the process of diastole → decrescendo type of murmur
- Starts at the onset of S₂
 - Right after S₂, the aortic valve and pulmonary valve are supposed to close
 - Aortic valve doesn't close fully → regurgitation jet into the left ventricle
 - Decreases the intensity throughout the process of ventricular diastole
- These patients have diastolic decrescendo murmur
 - That is blowing in intensity and quality

Auscultation location

- Right 2nd intercostal space parasternal border
- What we're going to listen → diastolic decrescendo type of murmur
 - Blowing in quality, very loud
 - Usually grade 3 or above

Murmur radiation

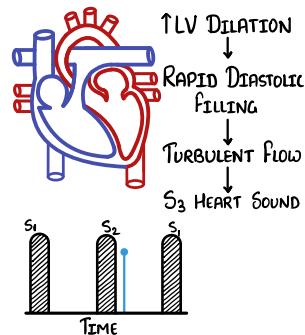
- Radiation
 - We have to think about the direction of the regurgitated blood flow
 - It's going to flow from the aorta down into the left ventricle
 - Radiates from the right 2nd intercostal space to the left sternal border
- Refer to the minute 1:04:10 – 1:04:25 of the lecture video for the aortic regurgitation murmur

Chronic aortic regurgitation → may produce S₃ heart sound

- In chronic patients
 - Opposite of the aortic stenosis patient
- They dilate their ventricles
 - Accumulate more volume
 - Can take a lot of volume very rapidly in the early phases of diastole**
- During diastole
 - Whenever blood's coming down into the atria, it comes down in 3 phases
 - Rapid diastolic filling
 - Blood's sitting in the atria rapidly flows down when tricuspid and mitral valve open
 - Mid-diastolic filling
 - Blood within the venous channel waiting to get into atria → move down to the ventricles
 - Late-diastolic filling
 - Atria decides to contract and push the remaining amount of blood into the ventricles
 - In patient with very distended, compliant, very large left ventricles

- They're going to be able to take in tons of blood during early phase of diastole
 - Rapid flow of blood and bang the wall**
 - Produce the turbulent blood flow → produce low-pitched murmur → S₃ heart sound

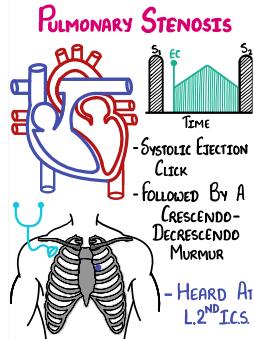
- Remember S₄ heart sound was the atrial kick from the left atrial hypertrophy
- Refer to the minute 1:06:29 – 1:06:39 of the lecture video for the S₃ heart sound



PULMONARY STENOSIS, PULMONARY REGURGITATION, TRICUSPID STENOSIS, TRICUSPID REGURGITATION

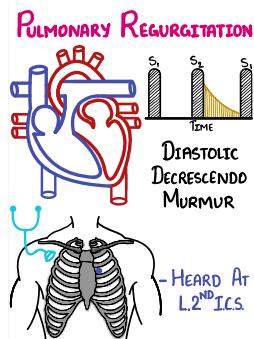
- Primary clinical features of these 4
 - Some type of right heart failure symptoms
 - Jugular venous distension
 - Hepatomegaly
 - Pedal edema
 - We're focusing on murmurs

(E) PULMONARY STENOSIS



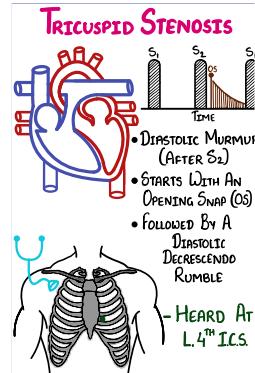
- Exactly like in **aortic stenosis**
- Except it's **heard at left 2nd intercostal space parasternal border**
- Also, no radiation that listed in the literature that's significant
- In pulmonary stenosis
 - We're having difficulty to get blood from the right ventricle into pulmonary artery
 - Difficult to click open or bow open the pulmonary valve → **produce early systolic ejection**
 - A lot of blood will start to flow out → **crescendo**
 - Less blood will leave → **decrescend**

(F) PULMONARY REGURGITATION



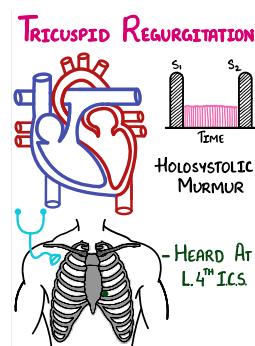
- Exactly like **aortic regurgitation**
- Except it's **heard at left 2nd intercostal space parasternal border**
- We have pulmonary valve that's incompetent
 - Not having a good seal
 - So blood will backflow from the pulmonary artery into the left ventricle
- When it does backflow
 - There are tons of blood initially that flows through and less over time → causes **diastolic decrescendo murmur**

(G) TRICUSPID STENOSIS



- Exactly like **mitral stenosis**
- Except it's **heard at left 4th intercostal space parasternal border**
- Very stenotic, fibrotic tricuspid valve
 - Difficult to get blood from the right atrium to right ventricle
- When it does, causes **opening snap right after during the beginning of diastole (S₂)**
 - We'll have **opening snap followed by a diastolic decrescendo type of rumbling murmur**

(H) TRICUSPID REGURGITATION



- Tricuspid valve is incompetent
 - It's not forming a good seal
- **Blood is backflowing from the left ventricle into the left atria during systole**
 - Occurring **throughout the entire time of systole → holosystolic regurgitation**
- Remember blowing musical intensity
 - Heard at **left 4th intercostal space parasternal border**



III) MURMUR MANEUVERS

(A) INSPIRATION

(1) Physiology

- When taking a deep breath:
 - Intrathoracic pressure drops
 - Increase of blood flow into the right side of the heart
 - More blood flowing through tricuspid and pulmonary valve
 - Increase of murmurs associated with those valves.
 - **Inspiration increases the intensity of right-sided murmurs.**

(2) Murmurs

- 2) Tricuspid stenosis
- 3) Tricuspid regurgitation
- 4) Pulmonary stenosis
- 5) Pulmonary regurgitation

(B) EXPIRATION

(1) Physiology

- When breathing out:
 - Intrathoracic pressure increases
 - Increase of blood flow from the pulmonary veins into the left atrium
 - More blood flowing through mitral and aorta valve
 - Increase of murmurs associated with those valves.
 - **Expiration increases the intensity of left-sided murmurs.**

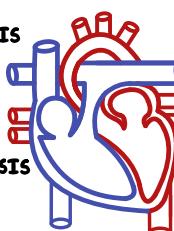
(2) Murmurs

- 1) Mitral stenosis
- 2) Mitral regurgitation
- 3) Aortic stenosis
- 4) Aortic regurgitation

RECAP

INSPIRATION

- ↑R. HEART FILLING
- ↑INTENSITY Of R. HEART MURMURS
- TRICUSPID STENOSIS
- TRICUSPID REGURGITATION
- PULMONARY STENOSIS
- PULMONARY REGURGITATION



EXPIRATION

- ↑L. HEART FILLING
- ↑INTENSITY Of L. HEART MURMURS
- MITRAL STENOSIS
- MITRAL REGURGITATION
- AORTIC STENOSIS
- AORTIC REGURGITATION

(C) SQUAT & PASSIVE LEG RAISE

(1) Physiology

(i) When doing a squat

- Contraction of muscles of lower extremities
- Squeezes blood up towards the heart

(ii) When having someone lying flat and raising their leg

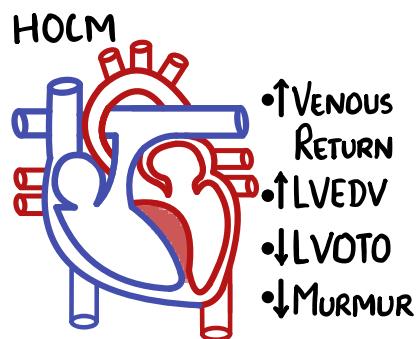
- Blood flows down from legs towards the heart due to gravity force

→ Increase of venous return with both maneuvers

- Increase intensity of all murmurs
- Two exceptions:
 - Hypertrophic obstructive cardiomyopathy
 - Mitral valve prolapse

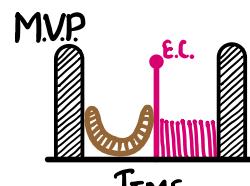
(2) Hypertrophic Obstructive Cardiomyopathy

- When squatting or raising legs
- More blood into the ventricles
- Blood stretches out the hypertrophic walls of the heart
- Decrease of obstruction
- Increases blood flow through the valves
- Decreases intensity of murmur



(3) Mitral Valve Prolapse

- When squatting or raising legs
- More blood into the ventricles
- Takes more time to generate a full contraction
- Later click
- Shorter murmur



- ↑VENOUS RETURN (↑LVEDV)
- LATER CLICK
- SHORTER MURMUR



(D) VALSALVA & STANDING UP SUDDENLY

(1) Physiology

(i) In Valsalva maneuver

- Increase of intraabdominal pressure and intrathoracic pressure
 - Difficulty for blood to flow into the heart

(ii) Standing up suddenly

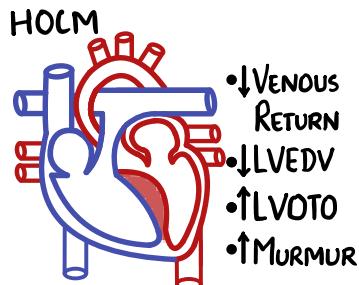
- Blood drops down to the lower extremities due to gravity force

→ Decrease of venous return with both maneuvers

- Decrease intensity of all murmurs
 - Two exceptions:
 - Hypertrophic obstructive cardiomyopathy
 - Mitral valve prolapse

(2) Hypertrophic Obstructive Cardiomyopathy

- When doing Valsalva or standing up suddenly
- Decrease of venous return
- Less blood in left ventricle
- Increases obstruction
 - Increases intensity of murmur



(3) Mitral Valve Prolapse

- When doing Valsalva or standing up suddenly
- Decrease of venous return
- Takes less time to generate a contraction
 - Earlier click
 - Longer murmur



RECAP

SQUAT/P.L.R.

- ↑VENOUS RETURN
- ↑INTENSITY OF ALL MURMURS
- 2 EXCEPTIONS -
- HOCM
- M.V.P

VALSALVA/STANDING

- ↓VENOUS RETURN
- ↓INTENSITY OF ALL MURMURS
- 2 EXCEPTIONS -
- HOCM
- M.V.P

(E) HANDGRIPS

(1) Physiology

- When squeezing a handgrip
- Increase of vascular resistance
 - Increases left ventricle afterload
 - Decrease of stroke volume
 - **Decrease of blood flow through the aortic valve**
 - **Decrease aortic stenosis murmur**
 - Increases blood into the left ventricle
 - **Increase of retrograde flow**
 - **Increase of aortic regurgitation murmur**

(i) Other effects of a stroke volume decrease:

- More flow into the ventricle
- **Decreases intensity of an hypertrophic obstructive cardiomyopathy murmur**
- Takes more time to generate a full contraction
 - **Later click and a shorter murmur in a mitral valve prolapse**
- More blood into the left ventricle
 - **Increases mitral regurgitation murmur**

(F) AMYL NITRATE

(1) Physiology

- Amyl nitrate vasodilate vessels
- Decrease of vascular resistance
 - Increases left ventricle afterload
 - Increase of stroke volume
 - **More blood flow through the aortic valve**
 - **Increase of aortic stenosis murmur**
 - **Less retrograde flow**
 - **Decrease of aortic regurgitation murmur**

(i) Other effects of a stroke volume increase:

- Less flow into the ventricle
 - **Increases intensity of an hypertrophic obstructive cardiomyopathy murmur**
- Takes less time to generate a full contraction
 - **Earlier click and longer murmur in a mitral valve prolapse**
- Less blood into the left ventricle
 - **Decreases mitral valve regurgitation murmur**

(G) LEANING FORWARD

- Brings aorta and aortic valve closer to the chest wall
- Increases intensity of aortic stenosis and aortic regurgitation murmurs

(H) LEFT LATERAL DECUBITUS

- Brings mitral valve close to the chest wall
- Increases intensity of mitral stenosis, mitral valve prolapse and mitral regurgitation murmurs.

LEANING FORWARD

- BRINGS AORTA + AORTIC VALVE CLOSE TO CHEST WALL
- ↑INTENSITY OF
 - A. STENOSIS
 - A. REGURGITATION

LEFT LATERAL DECUBITUS

- BRINGS MITRAL VALVE CLOSE TO CHEST WALL
- ↑INTENSITY OF
 - M. STENOSIS
 - M. V.P.
 - M. REGURGITATION

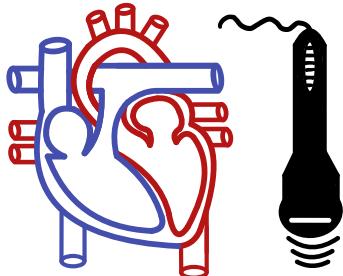


IV) DIAGNOSIS AND TREATMENT

(A) DIAGNOSTIC INVESTIGATIONS

ECHOCARDIOGRAM

- BEST TEST



→ Echocardiogram: BEST TEST

(i) Transthoracic (T.T.E.)

- For initial diagnosis
- Poor sensitivity and specificity but less invasive

(ii) Transesophageal (T.E.E.)

- For confirmatory diagnosis

• What you need to look for with an echo:

- Estimate valve diameter
- Estimate velocity across valve
- Estimate pressure gradients across valve
- Assesses EF + any regurgitation jets

(B) TREATMENT OF VALVULAR HEART FAILURE

(1) Heart Failure Management

→ Symptomatic treatment

- Fluid restriction
- Sodium restriction
- Diuresis
- Beta Blockers or ACE inhibitors

(2) Surgery

→ When symptomatic treatment isn't enough

(i) Balloon Valvuloplasty

- Preferred for stenosis lesions
- More specifically for mitral stenosis
- A balloon gets inflated between the valves and stretches the fibrotic valves opening the space for blood to flow.

(ii) Valve Repair / Replacement

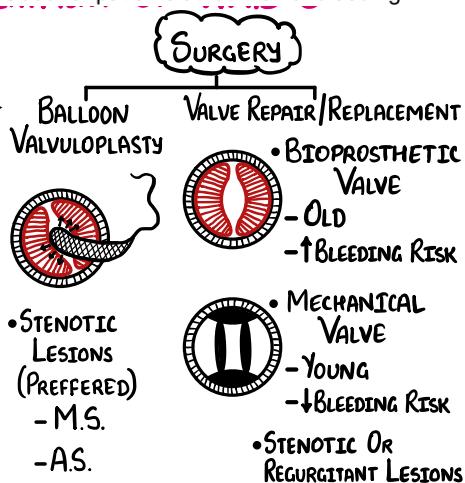
- For stenotic lesions that didn't respond to balloon valvuloplasty or severe regurgitant lesion

1) Bioprosthetic Valve

- Usually good for around 10 years
- Better for older patients
- No need of anticoagulation therapy
- Good for patients with high risk of bleeding

2) Mechanical Valve

- Usually good for many years
- Indicated in younger patients
- Anticoagulation therapy
- Good for patients at low risk of bleeding

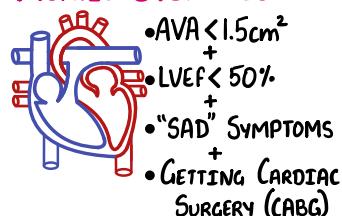


(3) Aortic Stenosis

(i) Indications:

- 1) Aortic Valve Area (AVA) $<1.5 \text{ cm}^2$
- 2) Ejection Fraction dropping $<50\%$
- 3) Symptoms like dyspnea, syncope on exertion or angina on exertion
- 4) They're getting a cardiac surgery for something other than aortic stenosis

AORTIC STENOSIS



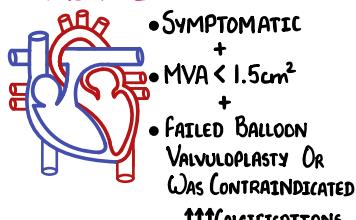
*SURGICAL Vs T.A.V.R INDICATIONS
(OPEN) (CLOSED)

(4) Mitral Stenosis

(i) Indications:

- 1) Failure after balloon valvuloplasty or contraindication to balloon valvuloplasty
 - Excessive amount of calcifications
- 2) Pulmonary edema symptoms
 - Dyspnea, orthopnea, paroxysm nocturnal
- 3) Mitral Valve Area (MVA) $<1.5 \text{ cm}^2$

MITRAL STENOSIS



*SURGICAL INDICATIONS
(OPEN)

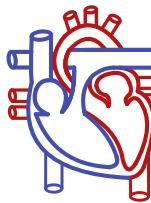


(5) Aortic Regurgitation

(i) Indications:

- 1) Left ventricle dilation >50 mm
- 2) Left ventricle ejection fraction <50%
- 3) They're getting a cardiac surgery for something other than aortic regurgitation
- 4) Symptoms

AORTIC REGURGITATION



- LV DILATION ($>50\text{mm}$) +
- LVEF $< 50\%$ +
- GETTING CARDIAC SURGERY (CABG) +
- SYMPTOMS

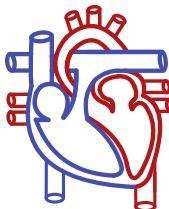
* SURGICAL INDICATIONS (OPEN)

(6) Mitral Regurgitation

(i) Indications:

- 1) Left ventricle ejection fraction 30-60%
- 2) Left ventricle dilation >40 mm
- 3) Left heart failure symptoms

MITRAL REGURGITATION



- LVEF 30-60% OR
- LV DILATION ($>40\text{mm}$) ±
- SYMPTOMS

* VALVE REPAIR VS REPLACEMENT INDICATIONS

(7) Prosthetic Valve Dysfunction

(i) Regurgitant Prosthetic Valve

1) Paravalvular leak

- Due to the prosthetic valve dehiscence
 - Back flow of blood
 - More commonly seen with mechanical valves

2) Transvalvular leak

- Due to some type of destruction of the bioprosthetic valve or a thrombus that blocks closure

(ii) Stenotic Prosthetic Valve

- Due to a valve thrombus causing an obstruction
- Cusp malfunction
 - Fail to open

(iii) Clinical features:

- A new murmur
- Macroangiopathic hemolytic anemia (MAHA)
 - AKA thrombotic microangiopathies
 - Rupture of RBC when trying to cross a very narrow space between stenotic valves
 - Anemia, hyperbilirubinemia, increase of LDH
- Acute onset of heart failure symptoms
- Increased risk of thromboembolism

PROSTHETIC VALVE DYSFUNCTION

REGURGITANT PROSTHETIC VALVE

• PARAVALVULAR LEAK

→ M/C \ominus MECHANICAL VALVES

→ 2/2 ANNULAR DEHISCENCE

• TRANSVALVULAR LEAK

→ M/C \ominus BIOPROSTHETIC VALVES

→ 2/2 CUSP DEHISCENCE
Or VALVE THROMBUS THAT BLOCKS CLOSURE



STENOTIC PROSTHETIC VALVE

• 2/2 VALVE THROMBUS

→ VALVE OBSTRUCTION

• 2/2 CUSP MALFUNCTION

→ FAIL TO OPEN



CLINICAL FEATURES

- NEW MURMUR
- MAHA → HEMOLYSIS OF RBC's
- HF SYMPTOMS
- ↑ THROMBOEMBOLISM

