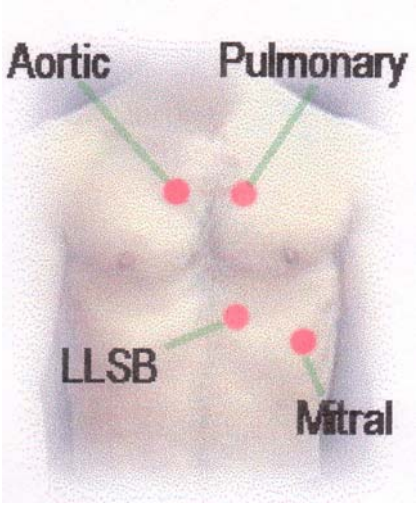
Heart Sounds.

IMENE BENAYACHE.MD

I/.Location of the valves:

- Mitral valve → 5 ICS (apex).
- Tricuspid valve → LLSB (left lower sternal border).
- Aortic valve → 2ICS (right).
- Pulmonic valve → 2 ICS (left).



II/. Anology of heart sounds:

A. First heart sound S_1:

- LUB/ is produced by the closure of the AV (mitral and tricuspid) valves during systole.

- Mitral valve closure comes before tricuspid valve closure (higher pressure on the left side).
- Occurs immediately following the beginning of the QRS.
- Preceeds carotid pulse.
- Best heard at the apex.
- Is a high frequency sound.
- Hard to hear a split.

B/. Second heart sound S_2:

- Produced by closure of aortic and pulmonic valves
- Occurs just after S_1.
- Aortic valve closure comes before pulmonic valve closure.
- Comes just after T wave.
- Normally splits during inspiration as the blood is sucked into the right heart → delays closure of PV, so PV separates from AV.
    - Inspiration: A_2……P_2
    - Expiration: A_2..P_2.
- Best heard at the base of the heart.
- Shorter and sharper and of higher frequency than S1.

C/. Third heart sound S_3:

- Ventricular gallop.
- Occurs shortly after S_2, during the early passive, rapid diastolic filling.
- Is normal in children and adults < 40 year-old.
- Abnormal in patients > 40 year-old and indicates:
    - CHF
    - MR or TR
    - Constrictive pericarditis.
    - Anemia.
- It is not caused by valves, but created by sudden tensing of ventricular wall as blood rush in.
- Low in volume and frequency.

D/. Fourth heart sound S4:

- Atrial gallop or presystolic gallop or atrial sound.

- Heard just before S_1.
- Coincides with late active diastolic filling.
- It is created when the atrial contraction rapidly distends the ventricle. When the stiff, non-complient ventricular wall reaches its physical limits it tenses, and the S_4 is created.
- Only patient swith atrial contraction can have an S4 (atrial fibrillation and junctional rhythms should not have it).
- Low in volume and frequency.
- Heard in trained athletes and elderly without cardiac disease.
- <u>Is abnormal and can indicate</u>:
  - AS
  - AMI
  - Hypertension
  - CAD

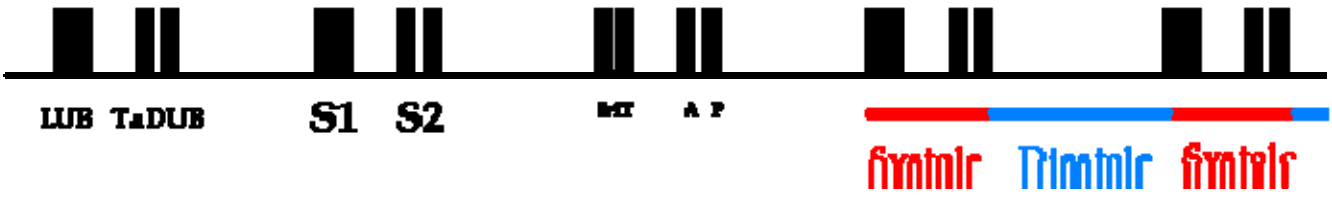
III/. Split of S_1:

- S_1 is created when the ventricles contract and close the mitral and tricuspid valves.

If the splitting is mild, it could be normal but if big it is due to:
RBBB.

IV/. Normal Splitting of S_2:

- S_2 is created when the ventricles relax and pressure from the aorta and pulmonary arteries exceeds the ventricular pressure ⇒ aortic and pulmonic valves close.
- During inspiration you can hear the splitting of S_2.
- If the split is wide or fixed it indicates disease.



V/. Quadruple gallop:

- Is produced by combining S4, S1, S2 and S3.
- Because S3 and S4 are low in intensity, low pitch (frequence), it is best heard at the apex with the bell of the stethoscope.
- Sounds like a galloping horse.
- It suggests CHF.

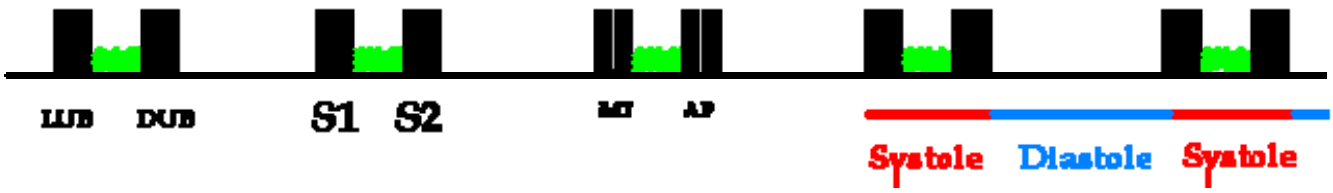
VI/. Summation gallop:

- Is the overlap of S3 and S4 as the patient`s heart rate reaches 110 beats/min.
- This is due to the fact that diastolic is shortened more than systolic as heart rate increases.

VII/. Systolic Murmurs:

A. Holosystolic Murmur.

- It is a pansystolic murmur, occupies the entire interval of systole.



B. Diamond Shapped murmur:

- It is a crescendo-decresendo murmur.
- Seen in aortic and pulmonic stenosis.

VIII/. Diastolic Murmurs:

- Occur during diastole.
- Seen in mitral and tricuspid stenosis and aortic and pulmonic regurgitation.

IX/. Systolic Clicks:

- Occur from abnormal ballooning of the MV into the left atrium as the mitral valve prolapses.
- Maneuvers that:
  - Decrease venous return (standing, Valsava, vasodilators) bring the click closer to S1
  - Increase venous return (lying down, squatting) move the click closer to S2.

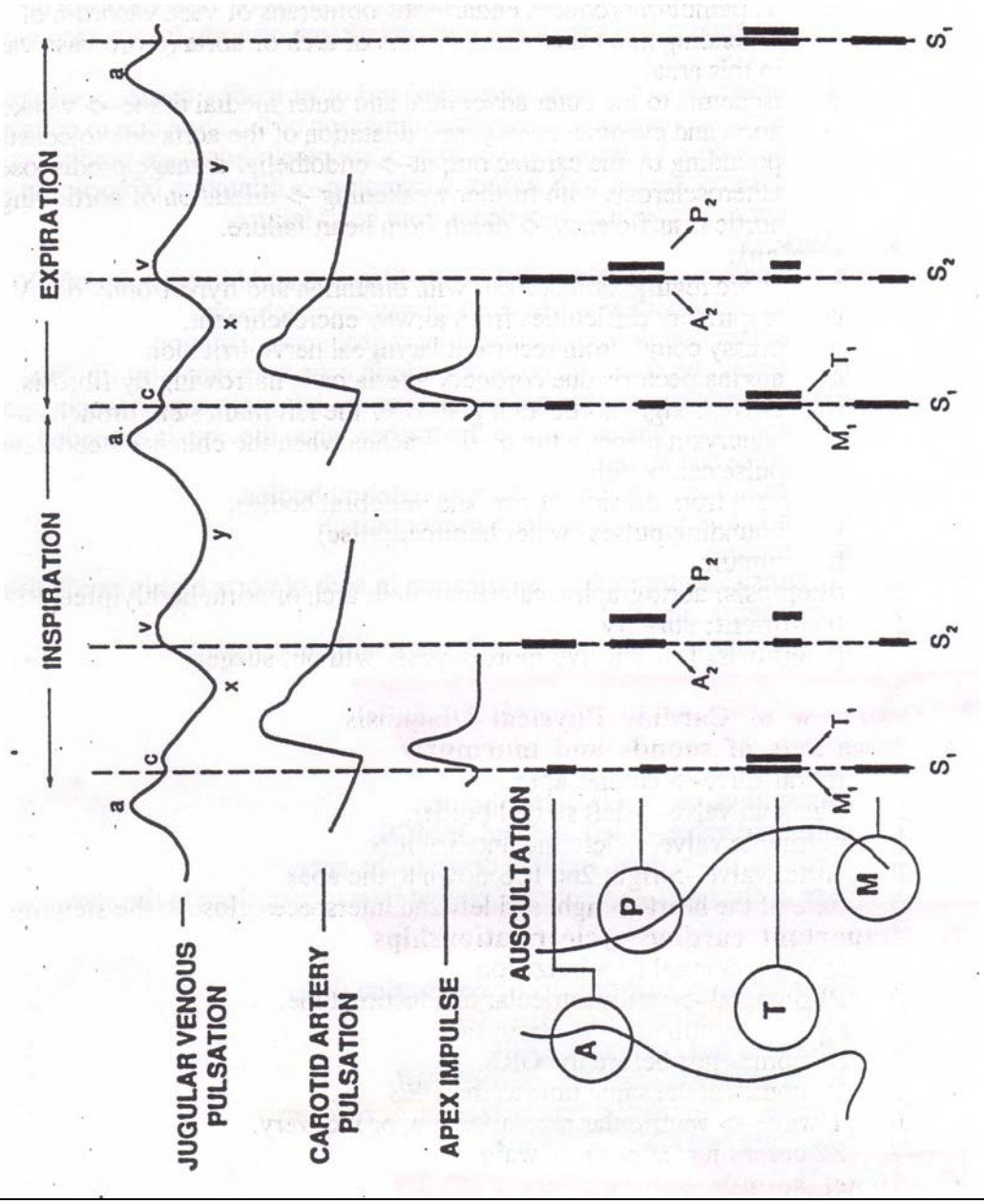
X/. Opening Snap:

- Feature of mitral and tricuspid stenosis, where valve is less pliable than normal.

- The earlier the OS, the worse the disease, because it means that LA pressures must have been very high to open the valve fast.
- Later in diastole the OS, the better the prognosis.

XI/. Factors influencing murmurs:

- The intensity of a murmur is influenced by:
  - Thickness of the chest wall.
  - Presence of intervening tissue.
- Murmurs ↓ in intensity in obesity and patients with ↑ anteroposterior diameters from COPD.
- As a general rule, inspiration increases the murmurs originating from the right heart (blood sucked into the thorax through right ventricle during inspiration).
- Expiration increases those originating from the left heart due to positive (or less negative) pulmonary pressures.
- **Summery:**



"First and Second heart sound".		
	"S1"	**"S2"**
Normal.	➢ **Represents closure of the "MV" and "TV" during systole.** ➢ **MV comes before TV (higher pressure in left ventricle).** ➢ **S1 is a low frequency sound.** ➢ **Signals the onset of ventricular systole.** ➢ **Normally we do not hear the split.** ➢ **Comes immediately following "QRS".** ➢ **Preceeds carotid pulse.**	➢ **Represents closure of "AV" and "PV".** ➢ **AV comes before PV.** ➢ **S2 is a high frequency sound (louder than S1).** ➢ **Signals the onset of ventricular diastole.** ➢ **Normally we hear a split during inspiration of S2 into (A2 & P2).** ➢ **S2 splits on inspiration as blood is sucked into the right heart → delays closure of PV, so PV separates away from AV.**

<table>
<tr><td></td><td colspan="2"></td><td colspan="2">➢ Comes just after “T” wave.
➢ Best heard at the base of the heart.</td></tr>
<tr><td><u>Abnormal.</u></td><td><u>Increased S1.</u></td><td><u>“Decreased S1”</u></td><td><u>“↑ S2”</u></td><td><u>“↓ S2”</u></td></tr>
<tr><td></td><td>➢ The wider the valve is spread, the greater the intensity of the sound when it closes.
➢ ↓ PR interval (valves are still wide open).
➢ Early mitral stenosis when the valve is more pliable and closes slower and it combines with closure of TV.</td><td>➢ Pulmonary emphysema (increased AP diameter).
➢ Pericardial effusion (muffles the sound).
➢ Prolonged PR interval (1st degree heart block, valve had time to partially close).
➢ Severe mitral stenosis (fixed opening and no movement of the valve).</td><td>➢ Aorta closure, because of systemic hypertension.
➢ In pulmonary closure (pulmonary hypertension).</td><td>➢ Aortic and pulmonic stenosis.</td></tr>
<tr><td></td><td colspan="2">➢ Split S1 MC delay in closure of TV from a RBBB.</td><td colspan="2">➢ <u>Paradoxical split of S2:</u> P2 comes before A2, the split occurs on expiration rather than inspiration.
• Due to a delay in closure of AV or early closure of the PV.
• MCC is LBBB, which delays AV closure.
➢ <u>Fixed splitting of S2:</u> is split in both inspiration (normal) and expiration (abnormal).
• Delay closure of PV or early closure of AV.
• Delay of closure of PV in ASD or VSD.
• Early closure of AV in mitral regurgitation.</td></tr>
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