I/. Location of the valves:

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

II/. Anology of heart sounds:

A. First heart sound S₁:
- LUB/ is produced by the closure of the AV (mitral and tricuspid) valves during systole.
Cardiovascular Section.

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

**B/. Second heart sound S₂:**
- Produced by closure of aortic and pulmonic valves
- Occurs just after S₁.
- 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: A₂…………P₂
  - Expiration: A₂..P₂.
- Best heard at the base of the heart.
- Shorter and sharper and of higher frequency than S₁.

**C/. Third heart sound S₃:**
- Ventricular gallop.
- Occurs shortly after S₂, 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.
Cardiovascular Section.

- Heard just before S₁.
- Coincides with late active diastolic filling.
- It is created when the atrial contraction rapidly distends the ventricle. When the stiff, non-compliant ventricular wall reaches its physical limits it tenses, and the S₄ is created.
- Only patients with 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.
- Is abnormal and can indicate:
  - AS
  - AMI
  - Hypertension
  - CAD

III/. Split of S₁:

- S₁ 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 S₂:

- S₂ 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 S₂.
- 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 (frequency), 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-decreased 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".

<table>
<thead>
<tr>
<th>Normal</th>
<th>“S1”</th>
<th>“S2”</th>
</tr>
</thead>
</table>
| Represents closure of the “MV” and “TV” during systole. | 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. |
| MV comes before TV (higher pressure in left ventricle). | 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. |
| S1 is a low frequency sound. | S1 is a low frequency sound.  
S1 represents closure of “MV” and “TV” during systole.  
S1 comes immediately following “QRS”.  
Precedes carotid pulse. |

Cardiovascular Section.

UHS.
<table>
<thead>
<tr>
<th>Abnormal.</th>
<th>Increased S1.</th>
<th>“Decreased S1”</th>
<th>“↑ S2”</th>
<th>“↓ S2”</th>
</tr>
</thead>
</table>
| ✓ 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. | ✓ Pulmonary emphysema (increased AP diameter).  
✓ Pericardial effusion (muffles the sound).  
✓ Prolonged PR interval (1° degree heart block, valve had time to partially close).  
✓ Severe mitral stenosis (fixed opening and no movement of the valve). | ✓ Aortic closure, because of systemic hypertension.  
✓ In pulmonary closure (pulmonary hypertension). | ✓ Aortic and pulmonic stenosis. |

✓ Split S1 MC delay in closure of TV from a RBBB.  

✓ **Paradoxical split of S2:** 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.  
✓ **Fixed splitting of S2:** 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.