THE SECOND HEART SOUND (S2), PULSUS PARADOXUS

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I S2: PHYSIOLOGY AND COMPONENTS

- **Definition:** The second heart sound represents the sudden deceleration of blood flow following the closure of the semilunar valves at the end of ventricular systole. It consists of two components:
- **Aortic Component (A2):** Represents aortic valve closure. It is normally louder and occurs first because the electrical activation of the left ventricle precedes the right ventricle, and the pressure gradient across the aortic valve is significantly higher.
- **Pulmonic Component (P2):** Represents pulmonary valve closure. It is softer and occurs slightly later.

II S2: NORMAL PHYSIOLOGICAL SPLITTING

- **Mechanism:** During normal inspiration, the intrathoracic pressure becomes more negative. This draws more venous return into the right heart, prolonging right ventricular systole and delaying the closure of the pulmonic valve (delayed P2). Simultaneously, the increased pulmonary vascular capacitance temporarily pools blood in the lungs, slightly reducing venous return to the left heart, leading to earlier aortic valve closure (early A2). Therefore, **S2 splits during inspiration and fuses during expiration**.

III S2: ABNORMAL SPLITTING PATTERNS (HIGH-YIELD EXAM CLASSIFICATION)

- **Wide Splitting:** S2 is split during both inspiration and expiration, but the split becomes even wider during inspiration.
  - **Mechanism:** Delayed electrical or mechanical emptying of the Right Ventricle, or early emptying of the Left Ventricle.
  - **Causes: Right Bundle Branch Block (RBBB)**, severe **Pulmonary Stenosis**, Pulmonary Arterial Hypertension, or early left ventricular emptying seen in severe **Mitral Regurgitation** or a **Ventricular Septal Defect**.
- **Fixed Splitting:** S2 is widely split, but the interval between A2 and P2 does not change with respiration.
  - **Mechanism:** Chronic volume overload of the right ventricle abolishes the normal respiratory variations in venous return.
  - **Causes:** The absolute hallmark of an **Atrial Septal Defect (ASD)**.
- **Paradoxical (Reversed) Splitting:** P2 occurs before A2. The splitting is heard during expiration and disappears (fuses) during inspiration.
  - **Mechanism:** Severe delay in left ventricular electrical or mechanical emptying, pushing A2 out past P2.
  - **Causes: Left Bundle Branch Block (LBBB)**, severe **Aortic Stenosis**, **Hypertrophic Obstructive Cardiomyopathy (HOCM)**, or active myocardial ischemia.

IV S2: INTENSITY OF THE COMPONENTS

- **Loud A2:** Seen in Systemic Hypertension or a dilated aortic root.
- **Soft or Absent A2:** A classic sign of severe, calcific **Aortic Stenosis** or severe Aortic Regurgitation.
- **Loud P2:** The clinical hallmark of **Pulmonary Arterial Hypertension**. If P2 is louder than A2 at the cardiac apex, pulmonary hypertension is strongly suggested.
- **Soft P2:** Seen in severe **Pulmonary Stenosis** or Tetralogy of Fallot.

V PULSUS PARADOXUS: DEFINITION AND HEMODYNAMICS

- **Definition: Pulsus Paradoxus** is a physical examination finding defined as an abnormally large decrease in systolic blood pressure—specifically a drop of **greater than 10 mmHg**—during normal, quiet inspiration. Despite the name, it is not a true "paradox"; it is simply an exaggeration of the normal physiological drop in blood pressure during inspiration.

VI PULSUS PARADOXUS: PATHOPHYSIOLOGY (VENTRICULAR INTERDEPENDENCE)

- **Setting:** The classic mechanism occurs within a tight, unyielding space, such as a pericardium filled with fluid.
- **The Septal Shift:** During inspiration, venous return to the right ventricle increases. Because the fluid-filled pericardium prevents the right ventricle from expanding outward, the right ventricle bulges inward, pushing the **interventricular septum to the left**.
- **Left Ventricular Compromise:** This septal bowing severely compromises left ventricular diastolic filling. Consequently, the left ventricular stroke volume drops sharply, leading to a sudden, measurable drop in the systemic systolic blood pressure during inspiration.

VII PULSUS PARADOXUS: CLINICAL MEASUREMENT TECHNIQUE

- Inflate the blood pressure cuff above the patient's systolic pressure.
- Deflate the cuff extremely slowly. Note the pressure at which the very first Korotkoff sounds are heard. At this point, the sounds will only be heard during **expiration**.
- Continue to deflate slowly until the Korotkoff sounds are heard equally throughout the entire respiratory cycle (during both inspiration and expiration).
- The difference between these two pressure readings is the measurement of the pulsus paradoxus.

VIII PULSUS PARADOXUS: MAJOR ETIOLOGIES

- **Cardiac Tamponade:** The most classic and life-threatening cause. Pulsus paradoxus is a hallmark sign and often correlates with the severity of hemodynamic compromise.
- **Severe Pulmonary Disease:** Acute severe **Asthma** or **Chronic Obstructive Pulmonary Disease (COPD)**. In these cases, the mechanism is completely different: extreme swings in negative intrathoracic pressure directly transmit to the aorta, mimicking a drop in stroke volume.
- **Constrictive Pericarditis:** Less common than in tamponade, occurring in about one-third of cases (typically the effusive-constrictive variant).
- **Other Causes:** Tension pneumothorax, massive pulmonary embolism, or severe hypovolemic shock.

IX TAMPONADE WITHOUT PULSUS PARADOXUS (THE EXAM CAVEAT)

- **Exam Question:** A classic examiner question asks when a patient can have true Cardiac Tamponade but completely lack a pulsus paradoxus. This occurs when ventricular interdependence is bypassed or impossible:
- **Severe Aortic Regurgitation:** The left ventricle fills from the aorta during diastole, so it does not care about the septal shift compromising left atrial inflow.
- **Atrial Septal Defect:** The septal defect perfectly balances the filling pressures between the right and left sides, neutralizing the paradox.
- **Severe Left Ventricular Hypertrophy or Dysfunction:** A stiff, non-compliant left ventricle will not allow the right ventricle to push the septum over.