Heart sounds and murmurs

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Heart sounds: First and second heart sounds

Genesis of the first and second heart sounds:

The first heart sound occurs **at the onset of ventricular systole**, when both the left and right ventricles begin contracting simultaneously. As contraction starts, intraventricular pressure begins to rise. As the ventricular pressure rises, the mitral leaflets move toward each other and close, but the pressure also tends to bulge the leaflets backward into the left atrium. To counteract this, **the papillary muscles** contract simultaneously with the ventricular myocardium, **tightening the chordae tendineae** and preventing excessive prolapse of the valve leaflets into the atrium. This tug-of-war between the blood pressure pushing the valve leaflets upward and the chordae tendineae pulling them downward generates **vibrations** in the leaflets, chordae, and adjacent myocardial tissue. These vibrations resonate through the cardiac structures and produce the audible **first heart sound (S1)**. Thus, the genesis of S1 is a combination of **valve closure and the dynamic vibration of the valve apparatus, particularly the leaflets, chordae tendineae, and papillary muscles, as they respond to the rising pressure of ventricular systole.** After the atrioventricular (AV) valves (mitral and tricuspid) close, the ventricles continue contracting, **and intraventricular pressure rises (isovolumetric contraction)**. Once the pressure in the ventricles exceeds that in the aorta and pulmonary artery, the aortic and pulmonary (semilunar) valves open silently, initiating the **rapid ejection phase**, during which a large volume of blood is forcefully ejected into the major arteries. This is followed by the **slow ejection phase**, during which the remaining portion of the stroke volume is expelled more gradually. After ejection, the **ventricles begin to relax**, marking the end of systole and the beginning of ventricular diastole. As the **intraventricular pressure rapidly falls** below the pressure in the major arteries, blood in the aorta and pulmonary artery attempts to flow back into the ventricles. This backward flow **forces the semilunar valves to close**, which prevents regurgitation. The closure of the semilunar valves—specifically the aortic and pulmonary valves—marks the end of systole and **produces the second heart sound (S2)**. This sound is not due to the valves simply shutting, but to the **elastic recoil and vibration of the valves, arterial walls (aorta and pulmonary artery roots), and surrounding cardiac structures** as the valve leaflets rebound toward the arterial side. These vibrations collectively generate the second heart sound.

Note: **In a healthy heart, the valves open silently without producing any sound, and if so, we call it a "click in systole" or "snap in diastole "and it marks an underlying pathology**.

Features of first and second heart sounds:

The first heart sound is low-pitched (low-frequency) with longer duration, while the second heart sound is high-pitched (high-frequency) with shorter duration.

Auscultation of the first heart sound:

The first heart sound has mitral and tricuspid components. Mitral component is best heard at the apex, and tricuspid component is best heard at the left lower sternal border (specifically at the fourth intercostal space)

Factors affecting the intensity of the first heart sound :

The primary factor influencing the intensity of the first heart sound is the **degree of separation between the valve leaflets (mitral and tricuspid) at the onset of ventricular systole.**

Normal Physiology:

• During **ventricular diastole**, blood flows from the atria into the ventricles, passing through open atrioventricular (AV) valves (mitral on the left, tricuspid on the right).

• In **early diastole**, the AV valves open widely due to rapid ventricular filling.

• As ventricular filling continues, the flow slows down and the **valve leaflets begin to float passively toward each other**, aided by the rising volume and pressure in the ventricles.

• In late diastole, atrial contraction (atrial systole) pushes the last bit of blood into the ventricles, further approximating the valve leaflets.

• By the **start of ventricular systole**, the AV valve cusps are already **close together**, and ventricular contraction quickly shuts them. This closure, followed by their vibration, produces a **normal first heart sound (S1).**

Pathological Conditions:

• If the AV valves are **abnormally wide open** at the onset of ventricular systole—due to disease or altered hemodynamics—the leaflets will have to travel a longer distance before closing.

• This results in a more forceful closure and **greater vibration**, producing a **louder S1.**

1. **PR Interval**: A Short PR interval (Wolff-Parkinson's syndrome) makes S1 louder. This is because ventricular systole will begin soon after atrial contraction, which means that the valve leaflets will still be widely open, producing a louder S1 sound. In contrast, a long PR interval (first-degree heart block) will make S1 sound softer. The PR interval is the most important determinant.

2. **Abnormal heart rate**: Tachycardia makes S1 louder as it reduces diastolic time and thus ventricular filling. Bradycardia makes S1 softer

3. **Velocity of ventricular contraction:** The strength and velocity of ventricular contraction directly influence how forcefully the AV valves close. Stronger and faster contractions (hyperdynamic states: Exercise, fever, anxiety, positive ionotropic drugs) cause a loud S1, while weaker and slower contractions (hypodynamic states: ischemic heart disease, post MI, HF, negative ionotropic drugs) lead to a soft S1.

4. **Valvular pathologies:**

    - **Early/Moderate Mitral Stenosis → Loud S1**

In early or moderate mitral stenosis (most commonly due to rheumatic heart disease), the valve becomes thickened and fibrotic but **remains mobile**. These changes create resistance to blood flow from the left atrium to the left ventricle, especially during diastole, and as a result, atrial pressure increases, and the atrium takes a longer time to empty. The mitral valve remains **widely open** at the onset of ventricular systole because the leaflets fail to drift close together, and the **forceful ventricular contraction abruptly closes the widely separated leaflets, producing a loud S1**.

- **Advanced Mitral Stenosis → Soft S1**

In advanced stages, the mitral valve becomes **heavily calcified and immobile**, and the chordae tendineae may also be thickened and retracted. These stiff, non-mobile **leaflets cannot come together or move effectively** in response to ventricular contraction, so **the mitral component of S1 will be diminished or absent.**

- **Tricuspid stenosis:**

Although less common than mitral stenosis, tricuspid stenosis behaves similarly as the valve remains more **widely open** at the onset of **right ventricular systole,** and if the valve leaflets **are still mobile**, they close **forcefully and rapidly, producing a loud S1.**

- **Mitral and tricuspid regurgitation:**

In **regurgitant lesions**, the valves fail to close properly during ventricular systole due to **structural defects** in the valve apparatus (ie, fibrotic chorda tendineae). The valve **does not close fully as a result**, leading to regurgitation of blood back into the atria. This will **make S1 diminished or absent**

- **Aortic regurgitation:**

Though the aortic valve is not directly involved in S1, **aortic regurgitation** can cause a soft S1 indirectly: During diastole, **regurgitant blood from the aorta flows backward** into the left ventricle. This abnormal flow strikes the anterior mitral leaflet, **prematurely pushing it toward closure**. Additionally, volume overload from both atrial and aortic sources leads to overfilling, bringing the **mitral leaflets closer together** passively. Thus, when ventricular systole begins, valves are already nearly closed, **resulting in a softer S1**

5. **Sound conductivity to the chest wall:**

The intensity of the first heart sound is not only determined by **how it is produced**, but also by how well it is **transmitted through intervening tissues to the stethoscope**. Conditions that **add air, fluid, or mass** between the heart and chest wall typically result in a **soft S1, while minimal barriers lead to a louder S1.**

Conditions causing variable intensity of the first heart sound:

Physiological splitting of the second heart sound:

Although both ventricles contract and relax almost simultaneously, the **aortic valve closes slightly earlier than the pulmonary valve, resulting in the physiological splitting of the second heart sound**. This difference arises from the varying resistance or impedance—each circulation offers to the forward flow of blood. The **systemic circulation**, supplied by the left ventricle through the aorta, is a **high-resistance, high-pressure system** characterized by narrower arterioles and greater smooth muscle tone. In contrast, **the pulmonary circulation**, supplied by the right ventricle via the pulmonary artery, is **a low-resistance, low-pressure system** with wider vascular channels. In the **systemic circulation**, where impedance is high, the **forward flow is quickly halted as soon as ventricular pressure drops**. The final portion of the ejected blood column promptly **reverses and strikes the aortic valve cusps**, leading to their **earlier closure**. Conversely, in **the pulmonary circulation**, the lower impedance allows the blood to continue **moving forward for a longer duration** even after the right ventricular pressure falls. This delays the reversal of flow and consequently delays the closure of the pulmonary valve.

Relation of physiological splitting to the respiratory cycle :

During inspiration, the chest cavity expands, leading to a **decrease in intrathoracic pressure**. This negative pressure not only allows air to enter the lungs but also **draws more venous blood into the thoracic cavity**. As a result, the systemic venous return to the right atrium and right ventricle increases. The **right ventricle** becomes more filled—**overloaded compared to baseline**, and consequently, when it contracts, it takes a **longer time to eject this larger volume of blood**. This leads to a **prolonged right ventricular systole and a delayed closure of the pulmonary valve**.

At the same time, the expansion of the lungs causes **the pulmonary vasculature to expand as well**, increasing its capacity to hold blood. This temporary sequestration of blood within the pulmonary circulation **reduces the amount of blood returning to the left atrium and ventricle**. As a result, **the left ventricle receives less blood, contracts with a smaller volume, and completes its ejection more quickl**y. This leads to an earlier closure of the aortic valve. The combined effect of a delayed pulmonary valve closure and earlier aortic valve closure during inspiration causes the two components of the second heart sound—A2 and P2—to move further apart. This separation is known as **physiological splitting** of the second heart sound, and it is a **normal auscultatory finding during inspiration**.

Auscultation of physiological splitting :

To hear the inspiratory physiological splitting, put your diaphragm on the right parasternal area, second intercostal space to hear the aortic component, and the left second intercostal space to hear the pulmonary component. However, **if you were to choose one area where you can hear both components, choose the pulmonary area (left parasternal area, second intercostal space).**

Types of splitting of the second heart sound:

1. **Wide splitting:** A2-P2 interval is prolonged, audible during expiration, and further widens during inspiration **(wide and variable).** It happens as a result of conditions that cause **early closure of the aortic valve or delayed closure of the pulmonary valve**

Delayed closure of the pulmonary valve:

- **Right bundle branch block (RBBB):** In RBBB, the electrical impulse reaches the right ventricle through a **slower**, muscle-to-muscle conduction from the left ventricle, **delaying** the onset of **right ventricular contraction**. As a result, the **pulmonary valve closes later** than normal, widening the A2–P2 interval. RBBB is one of the most important and common causes of wide S2 splitting.
- **Right Ventricular Dysfunction:** When the right ventricular myocardium is weakened—due to ischemia or other conditions—it **contracts more slowly and inefficiently**. This **prolongs right ventricular systole**, requiring more time to eject blood, which leads to a **delayed closure of the pulmonary valve.**
- **Pulmonary Stenosis:** In this condition, the pulmonary valve is narrowed due to fibrosis or thickening**, increasing resistance to right ventricular outflow**. The right ventricle needs more time to overcome this resistance and eject blood, **prolonging systole and delaying P2**.

Early closure of the aortic valve:

- **mitral regurgitation (MR):** the mitral valve is **incompetent**, allowing a portion of the **stroke volume to leak backward** into the left atrium during systole. As a result, less blood is

ejected into the aorta. With **reduced forward output**, the left ventricle finishes ejecting its smaller stroke volume more quickly, leading to **premature aortic valve closure** and hence, early A2.

- **Ventricular septal defect (VSD):** With a left-to-right shunt, there is an abnormal communication between the left and right ventricles. When the left ventricle contracts, **some of the blood is shunted into the right ventricle instead of being ejected into the aorta**. This again reduces the volume of blood passing through the aortic valve, **shortens the duration of systole, and causes early A2.**
- **Electrical conduction abnormalities,** such as in Wolff-Parkinson-White (WPW) syndrome type A, can also result in early A2. In this condition, there is an **accessory conduction pathway** that **prematurely activates the left ventricle** (pre-excitation), while the right ventricle is activated through the normal delayed conduction. The left ventricle contracts and relaxes earlier, leading to **earlier aortic valve closure**, while the right ventricle contracts and relaxes later, potentially delaying P2. This results in a wide A2–P2 interval due to both early A2 and possibly delayed P2

2. **Fixed splitting:** A2-P2 interval is prolonged but remains constant across the respiratory cycle **(wide and fixed).** Fixed splitting of the second heart sound (S2) is a hallmark finding in patients with **atrial septal defect (ASD)**. In ASD, there is a persistent **left-to-right shunt** due to a hole in the interatrial septum. Because the left atrial pressure is higher, blood continuously flows into the right atrium, adding to the normal systemic venous return. This chronic volume overload of the right heart **prolongs right ventricular systole and delays P2 pathologically**. The key point is that this delay in P2 remains constant throughout the respiratory cycle. This happens because **the amount of shunted blood dynamically adjusts**: during expiration (when systemic venous return is lower), more blood is shunted from left to right, and during inspiration (when systemic venous return is higher), less blood is shunted. These adjustments **result in a consistently elevated right-sided volume load, maintaining a fixed delay of P2 regardless of the respiratory phase**. Consequently, S2 remains split during both inspiration and expiration, and the degree of splitting does not vary.

3. **Paradoxical (reversed splitting):** Paradoxical splitting is characterized by a **reversed order of valve closure** (P2 before A2, because the **aortic valve closure is pathologically delayed**), audible splitting of S2 during expiration, and fusion or narrowing of the split during inspiration, which is the opposite of what happens in normal physiological splitting. The main mechanism behind paradoxical splitting is **delayed left ventricular systole**, which leads to late closure of the aortic valve (A2). Several cardiac conditions can cause this:
    - **Aortic stenosis**: The narrowed aortic valve increases afterload, prolonging left ventricular ejection time and thus delaying A2.
    - **Hypertrophic obstructive cardiomyopathy (HOCM**): Asymmetric septal hypertrophy obstructs the left ventricular outflow tract, delaying ejection and A2.

- **Left bundle branch block (LBBB)**: Electrical conduction to the left ventricle is delayed, postponing systolic contraction and A2.
- **Left ventricular systolic dysfunction**: Ischemia or myocardial damage reduces contractility, causing the ventricle to contract sluggishly and for a longer duration, also delaying A2.

Third heart sound

During the early phase of ventricular diastole, the mitral (or tricuspid) valve opens, allowing a large volume of blood to rapidly enter the ventricle—a stage known as the **rapid ventricular filling phase**. As blood enters, the ventricular walls stretch, leading to ventricular distension. By the end of this rapid inflow, the **ventricular walls reach their elastic limit and become taut**—they can no longer expand easily. If rapid blood flow continues despite the walls being fully stretched, **the incoming blood causes the tense ventricular walls to vibrate**. This **vibration generates the third heart sound (S3).** Thus, S3 arises due to rapid ventricular filling into a distended, non-compliant (taut) ventricle. Clinically, S3 is a low-pitched, low-frequency, and soft sound, **best heard with the bell of the stethoscope, typically over the apex in the left lateral decubitus position**. It reflects a **physiological response in young individuals or a pathological state in older adults**, such as heart failure, where ventricular compliance is reduced.

Fourth heart sound

caused by forceful atrial contraction against a non-compliant or stiff ventricle, most often with left ventricular hypertrophy due to hypertension, aortic stenosis, or hypertrophic cardiomyopathy. It cannot occur when there is atrial fibrillation**.** Best heard with the bell at the apex.

Murmurs

Heart murmurs are **audible vibrations** produced by **turbulent blood flow** through the heart or great vessels. While normal blood flow may have mild turbulence, it is typically inaudible. When turbulence becomes significant **due to accelerated or abnormal flow**, it becomes detectable through auscultation as a murmur.

Causes of Turbulent Flow Leading to Murmurs

1. **Within cardiac chambers**: e.g., abnormal flow between atria or ventricles due to structural defects.
2. **Across valves**: e.g., forward flow obstruction (e.g., aortic stenosis) or regurgitation (e.g., mitral regurgitation).
3. **Through septal defects**: e.g., ventricular septal defect (VSD) allows abnormal flow between ventricles.
4. **From ventricles to great arteries**: e.g., left ventricle to aorta or right ventricle to pulmonary artery.
5. **Between great arteries:**e.g., patent ductus arteriosus (PDA).

Classification Based on Timing in the Cardiac Cycle

1. Systolic Murmurs

- Occur between S1 (first heart sound) and S2 (second heart sound).
- May start with S1 or shortly after and end before or with S2.
- Represented by blood flow during ventricular systole.

2. Diastolic Murmurs

- Occur between S2 and the following S1.
- May start with or after S2 and terminate before or at the next S1.
- Represent blood flow during ventricular diastole.

3. Continuous Murmurs

- Span both systole and diastole, enveloping the S2.
- Begin in systole, persist through S2, and continue into diastole.
- Characteristic of abnormal connections, e.g., PDA, and are not restricted to one phase of the cycle.

Systolic Sounds :

Systolic clicks (pathological sounds upon the opening of aortic and/or pulmonary valves) can be divided into Early (ejection clicks) and Mid-systolic (Non-ejection clicks). While auscultating them, palpate the carotid artery and feel the pulse, as early clicks correspond to the carotid pulse upstroke, while mid-systolic clicks can be auscultated after the upstroke of the carotid pulse.

1. Aortic (left-sided) ejection clicks: These can be caused either because of problems in the valve itself (Bicuspid congenital aortic valve / Aortic stenosis) or in the root of the aorta (Dilated root of aorta). Being able to hear the clicks means that the leaflets are still flexible (to some extent). As time passes, the clicks may disappear, indicating that the valve is heavily calcified and immobile.
2. Pulmonary (right-sided) ejection clicks: These can be caused either because of problems in the valve itself (pulmonary stenosis) or in the root of the aorta (Dilated root of aorta). Being able to hear the clicks means that the leaflets are still flexible (to some extent). As time passes, the clicks may disappear, indicating that the valve is heavily calcified and immobile.

Notes :

- The aortic valve calcifies more heavily and rapidly than the pulmonary valve
- Murmurs of the right side of the heart are all best heard during inspiration except the pulmonary ejection click. that is because of the high EDP that will doom the valve leaflets even before systole starts
- In mitral valve prolapse, one or both leaflets will project into the left atrial cavity during systole. Problems in the annulus, leaflet itself, chordae tendineae, or papillary muscles might be the cause. Mitral valve prolapse produces mid-systolic non-ejection click. Why mid-systole? Let's say that the ventricle has filled with a large end-diastolic volume of blood. As soon as it starts contracting, the blood volume will decrease progressively, and the ventricle will get smaller in size. With the presence of a problem in the mitral valve apparatus, the leaflet won't stay in its position, and will prolapse causing the chordae tendineae to tense suddenly, vibrate, producing the click.

Opening snap :

Opening snap is a **high-pitched** sound heard during **early diastole**, when a **stenotic** AV valve, typically **the mitral**, opens abnormally. In healthy heart, the valve leaflets are thin, flexible, and open smoothly, allowing blood to flow freely from atrium to the ventricle. However, in conditions such as **mitral stenosis**, usually caused by **rheumatic heart disease**, the valve leaflets become thickened, fibrosed, and stuffed due to chronic inflammation and scarring. These changes reduce the mobility of the valve and lead to valve opening narrowing. As a result, the **left atrial pressure increases** in order to push blood through the narrowed valve. Because the valve is partially pliable, it starts to open rapidly, but due to fibrosis and adhesions of the valve leaflets, there is a sudden halt in the opening motion. This will eventually lead to vibration of the valve apparatus, producing the characteristic opening snap.

Note:the timing of the opening snap relative to S2, known as S2-OS interval, shortens as the severity of stenosis increases. When the valve becomes heavily calcified and stenosed, the opening snap disappears.

Early-diastolic murmur:

An early diastolic murmur occurs at the very beginning of diastole, immediately following the **second heart sound (S2).** This murmur is indicative of a problem with the **aortic or pulmonary valve**, the **semilunar valves** responsible for preventing the backflow of blood from the great arteries into the ventricles once systole ends and diastole begins. Under normal conditions, after the ventricles contract and eject blood into the aorta and pulmonary artery, these vessels elastically recoil. The aortic and pulmonary valves close to prevent retrograde flow, producing the S2 sound.

However, if the aortic or pulmonary valve is incompetent, meaning it fails to close properly due to structural damage such as from infective endocarditis, rheumatic disease, or aortic root dilation, the valve leaflets fail to close, and regurgitant blood flows backward into the ventricles during early diastole. The result is turbulent backward flow, which produces an early diastolic murmur that begins immediately after S2.

This murmur is **decrescendo**, meaning it begins with high intensity and gradually decreases. This pattern reflects the **declining pressure gradient** between the aorta (or pulmonary artery) and the corresponding ventricle.

Mid-diastolic murmur:

Moving further into diastole, the next physiological event is the opening of the atrioventricular (AV) valves (mitral and tricuspid) once ventricular pressure drops below atrial pressure. Normally, this causes passive ventricular filling without turbulence. However, if there is an obstruction to forward flow (such as mitral or tricuspid stenosis caused by rheumatic heart disease) or if there is increased flow across the valve (as in high-output states), this phase becomes turbulent and produces a mid-diastolic murmur. This murmur begins after S2, not with it, because it results from turbulent flow during the rapid filling phase, rather than from semilunar valve incompetence. Therefore, early diastolic murmurs are usually due to regurgitation through the aortic or pulmonary valves. In contrast, mid-diastolic murmurs are caused by stenosis or high flow across the mitral or tricuspid valves during ventricular filling.

Late-diastolic murmur (pre-systolic murmur) :

In the final phase of diastole, after the period of slow ventricular filling, the atria contract to actively push the remaining blood into the ventricles. If the atrioventricular (AV) valves, particularly the **mitral or**

tricuspid valve, are narrowed due to **stenosis**, the atria must contract more forcefully to overcome the obstruction. This increased pressure generates a jet of blood through the stenotic valve, causing **turbulent** flow, which results in a **late diastolic murmur**, also known as a **pre-systolic murmur**. This murmur occurs just before the onset of the next ventricular systole and is most classically associated with mitral stenosis, where elevated left atrial pressure and strong atrial contraction produce the characteristic turbulent flow across the narrowed mitral orifice.

What is the difference between late-diastolic murmur and S4? While both are caused by atrial contraction, they differ in their **mechanisms. S4** is produced when the atria contract **against a stiff, non-compliant ventricle**, such as in left ventricular hypertrophy or acute ischemia, causing a low-pitched sound due to the sudden deceleration of blood. In contrast, the **late diastolic murmur** arises when atrial contraction forces blood through a **partially obstructed atrioventricular valve**, typically the mitral or tricuspid valve, as seen in valvar stenosis. This creates a turbulent flow that produces a murmur just before S1.

Note:

Clinically, this sequence of sounds can be appreciated during auscultation. A patient with mitral stenosis may exhibit:

- **A loud S1**. As the disease advances, leaflet calcification worsens. This causes S1 to become softer.
- **An opening snap shortly after S2**. It will disappear as the disease progresses.
- **A mid-diastolic murmur,** as the disease progresses, will become **longer in duration** as the pressure gradient between the left atrium and ventricle persists further into diastole. However, **murmur intensity does not necessarily correlate with severity**. In very advanced disease, the orifice may be so narrow that only a small volume of blood passes through, reducing turbulence to the point where the murmur becomes faint or even inaudible. Therefore, in late-stage mitral stenosis, the **murmur may paradoxically disappear despite severe obstruction and low cardiac output.**
- **A late diastolic murmur (pre-systolic murmur), often culminating in pre-systolic accentuation,** if atrial fibrillation (AF) develops—a common complication due to atrial dilation and high pressures, atrial contraction is lost, and **pre-systolic accentuation disappears.**

The overall murmur thus has a **crescendo** shape: it starts softly after the opening snap, becomes louder during the middle of diastole, dips slightly, then intensifies again just before S1 due to atrial contraction. This dynamic change helps differentiate it from other murmurs and assess the severity and progression of mitral stenosis.