MASTERING FIRST HEART SOUND

DR. NAJEEB LECTURE NOTES

BY FATIMA HAIDER

KGMC

http://koracademy.com/

GENESIS OF S_1

The first heart sound i.e. S_1 is produced at the onset of ventricular systole.

S_1 is associated with closure of mitral valve and tricuspid valve.

When ventricular systole starts, intraventricular pressure begins to rise. As soon as ventricular pressure becomes greater than atrial pressure, blood from ventricular chamber will try to go back to atrial chamber but at that moment atrioventricular valves close, thus preventing backward flow of blood. The ventricular pressure may cause cusps of AV valves to evert into the atrial chambers. This eversion is prevented by contraction of papillary muscles whose tendons (chordae tendinae) are attached to the cusps of AV valves.

Closure of mitral and tricuspid valves will lead to vibrations in ventricular chambers, ventricular walls and the major arteries (aorta and pulmonary artery). This vibration is transmitted through the tissues to the chest wall and can be heard as first heart sound.

The ventricles keep on contracting. When ventricular pressure becomes greater than aortic and pulmonary pressure, the aortic and pulmonary valves open. In healthy heart, valves open silently.

If opening of semilunar valves produce sound, we call it **click** and usually it indicate some pathologic condition.

If opening of AV valves i.e. mitral and tricuspid valves produce sound, it is called **snap** which is heard in pathologic conditions.

When ventricles start relaxing, intraventricular pressure rapidly goes down. As soon as intraventricular pressure becomes lower than major arteries, pulmonary and aortic valves close and produce second heart sound, S_2. So S_2 isat the end of ventricular systole and beginning of ventricular diastole.

Ventricular systole is between S_1 and S_2.

S_1 – Lub

S_2 – Dub

COMPONENTS OF S_1

S_1 has two components; mitral component and tricuspid component.

M_1 – Mitral component

T_1 – Tricuspid component

Pressure in left ventricle rises more rapidly than right ventricle so mitral valve closes slightly before the tricuspid valve and there may be slight separation of S_1 sound. In 50% of normal population, this separation of heart sound may be audible as independent entities.

In first heart sound, mitral component contributes more than tricuspid component i.e. mitral component is louder than tricuspid component.

FEATURES OF S_1

Pitch of S_1 – The pitch of S1 is lower as compared to S2.

As AV valves and ventricular chambers, both are less elastic, so when AV valves close these structures vibrate with less frequency.

The semilunar valves, on the other hand are more elastic, their associated chambers (atria) are also more elastic and they are more taut, so they vibrate with higher frequency.

Duration and Frequency of S_1:

Duration of S_1 – 0.15s

Duration of S_2 – 0.11s

Frequency of S_1 – 25-45 Hz

Frequency of S_2 – 50Hz

So S_1 have long duration and low pitch.

S_2 have short duration and high pitch.

AUSCULTATION OF S_1

To auscultate mitral component of S_1, we have to place stethoscope on chest wall at point where left ventricle is closest to chest wall. Mitral component can be auscultated at apex beat (5^{th} intercostal space in mid clavicular line)

To auscultate tricuspid component of S_1, stethoscope should be placed on chest where right ventricle is closest to chest wall. The tricuspid component can be auscultated at 4^{th} intercostal space at left sternal border.

S_1 is louder than S_2 at mitral and tricuspid area.

S_2 is louder than S_1 at base of the heart (aortic and pulmonary area)

APEX BEAT :

Apex beat is the outermost and lowermost part of the precordium. Precordium is that part of the chest which is in front of the heart.

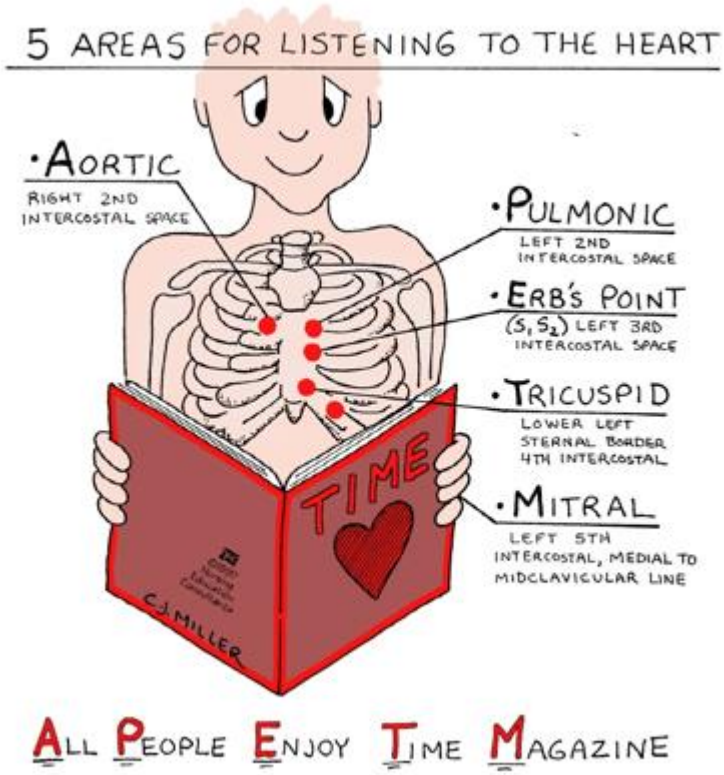
When ventricle contract, it hits the chest wall at the apex beat. At apex beat, a definitive cardiac impulse is palpated.

At apex beat, mitral component of first heart sound can be auscultated. Most commonly, apex beat is palpated at 5^{th} intercostal space in mid clavicular line.

At apex beat, only mitral component of S_1 is heard, while at 4^{th} intercostal space at lower left sternal border, both mitral and tricuspid components of S1 can be heard. So it's preferable to auscultate at this point as splitting of S_1 can be observed at this point.

How to differentiate S_1 from S_2?

To confirm the S_1, we palpate the carotid artery. Because when the ventricles contract, not only it produce the first heart sound, but at almost same time, it produce an upstroke of the carotid pulse. The heart sound which synchronizes with the upstroke of the carotid pulse is S_1.



INTENSITY (Amplitude or Loudness) OF S_1

Intensity of S_1 may be loud or soft. The intensity of S1 is determined by degree of separation of cusps of AV valves when ventricular systole starts.

In some pathologic conditions, just before ventricular systole, valves are kept wide open. In such cases when ventricles will contract, the valves will close from greater distance and loud heart sound will be produced.

While when valves will close from smaller distance, the sound produced will be low and called soft first heart sound.

LOUD S_1

Factors leading to loud S_1

1. Shortened PR Interval
2. Hyperdynamic ventricles
3. Initial stages of mitral stenosis
4. Tricuspid stenosis

- ## SHORTENED PR INTERVAL

PR interval is the time in which the ventricles are filled. PR interval is the most important determinant of intensity of S1.

Whenever PR interval is short, it means atria are filling the ventricles when ventricular contraction begins. As the cusps are still wide open, so the valves will close with a loud sound.

Classical examples of short PR interval are seen in

- Left ventricular pre-mature excitation. It's examples include Wolf Parkinson's White Syndrome and Lown-Ganong-Levine syndrome

Wolf Parkisnon's White Syndrome - WPW is an electrical abnormality in the heart that may be associated with supraventricular tachycardia (fast heart rate originating above the ventricles).

In WPW, along with normal conduction pathway, there is an extra pathway called an accessory pathway. They are normal heart muscle, not specialized electric tissue, and they may Conduct impulses faster than normal or Conduct impulses in both directions

The impulses travel through the extra pathway (short cut) as well as the normal AV-HIS Purkinje system. The impulses can travel around the heart very quickly, in a circular pattern, causing the heart to beat unusually fast.

Lown-Ganong-Levine syndrome - The Lown-Ganong-Levine (LGL) syndrome occurs when an accessory pathway is congenitally present that directly connects the atria to the ventricles, bypassing the AV node similar to the Wolff-Parkinson-White (WPW) **syndrome**.

- Tachycardia - Tachycardia is the medical term for a heart rate over 100 beats per minute. During tachycardia, ventricular filling time is reduced. By the time ventricles start contracting, still atria are pushing the blood and valves are wide open. Hence the valves will close will loud S_1.

- ## HYPERDYNAMIC VENTRICLES

Whenever ventricles are hyperdynamic, they are under positive ionotropic influences. When ventricles contract strongly and rapidly, they will close the cusps with more power and velocity and S_1 observed will be loud.

Conditions producing hyperdynamic ventricles include Hyper androgenic conditions or conditions producing hyper dynamic circulation e.g.

1. Exercise
2. Fever
3. Positive ionotropic drugs
4. Anxiety

- ## PATHOLOGIC CONDITIONS OF AV VALVES

The pathologic AV valves conditions producing loud S1 include Mitral stenosis and tricuspid stenosis

- ## MITRAL STENOSIS

In mitral stenosis, initially S_1 becomes louder and at advanced stage it becomes softer.

In early stages of mitral stenosis, the mitral valve is fibrotic, thickened but still it is not heavily calcified. The valve does not open completely, it only opens poorly and is still mobile. In mitral stenosis, atria take longer time to empty into ventricles. In a stenotic mitral valve, blood is pushed into ventricles under high pressure. Due to these reasons, at beginning of ventricular systole, they are still kept open. So when ventricular contraction begins, the valves will close forcefully produce loud S_1.

At advanced stages of mitral stenosis, mitral valve becomes very narrow and heavily calcified. The chordae tendinae also thickens and shrink. Under these circumstances, when left ventricle contract, the cusps will not meet each other and the valves will not close. S_1 in this case will be produced by tricuspid component only i.e. mitral component of S_1 will be absent and hence S_1 will be soft.

SOFT S_1

Factors leading to soft S_1

1. Prolonged PR interval
2. Hypodynamic ventricles
3. Aortic Regurgitation
4. Late stages of mitral valve stenosis i.e. calcified mitral valve
5. Mitral valve regurgitation
6. Pericardial effusion
7. Emphysema
8. Pneumothorax
9. Pleural effusion

- ## PROLONGED PR INTERVAL

Atria have enough time to fill the ventricles. Before ventricular contraction begins, the cusps have come close to each other so S_1 will be soft.

- ## HYPODYNAMIC VENTRICLES

Whenever ventricles contract poorly i.e. in hypodynamic ventricles, they will close the valve leaflet slowly and S_1 observed will be soft.

Conditions causing hypodynamic ventricles:

1. Ischemic heart disease
2. Post Myocardial Infarction
3. Cardiac output drop
4. Left ventricular dysfunction
5. Negative ionotropic drugs

- ## AORTIC REGURGITATION

During ventricular systole, mitral valve should close and eventually aortic valve should open and blood should be ejected out.

During ventricular diastole, aortic valve should close and mitral valve should open.

If aortic valve is incompetent, and cannot prevent the back flow then during ventricular diastole, the mitral valve will close due to two reasons:

1. The aortic jet of blood will hit the anterior mitral leaflet, causing it to close
2. As ventricle is being filled from both sides, mitral valve will close and S_1 observed will be soft.

- ## MITRAL VALVE REGURGITATION

The mitral valve in certain cases, do not close properly during ventricular systole and backward flow of blood through mitral valve takes place. This condition may be due to shortened, fibrotic chordae tendinae. As function of chordae tendinae is to keep the valves closed, so if chordae tendinae is fibrotic, they won't allow the valve to close and hence blood will flow back through atrium. As valves do not close so S_1 will be soft.

VARIABLE S_1 INTENSITY

In variable S_1 intensity, degree of loud sound may vary from one beat to the next beat.

Causes of variable S_1 intensity include:

1. Ventricular premature beat
2. Third degree heart block
3. Ventricular tachycardia
4. Atrial fibrillation (AFib)

• VENTRICULAR PRE-MATURE BEAT

In ventricular pre-mature beat, some myocardial cells which are pathologically excited may start to dispay abnormal automaticity. These cells induce strong ventricular pre-mature contraction due to which valves will close rapidly and produce loud S_1.

A ventricular premature beat is an extra heartbeat resulting from abnormal electrical activation originating in the ventricles (the lower chambers of the heart) before a normal heartbeat would occur.

• THIRD DEGREE HEART BLOCK

Third degree heart block is also called third degree AV block. In this condition, current from atria cannot go to the ventricles. Normally the only point through which current can go from atria to ventricle is AV node. Due to some diseases AV node cannot conduct electrical current. Under these circumstances, ventricles start their own rhythm i.e. produce their own ectopic pacemaker.

Atria will contract in response to SA node while ventricles will contract in response to ectopic pacemaker so both will contract at different rhythm and atrial contractions will no more be synchronized with ventricular contractions.

Due to this loss of synchronization, ventricles will close AV valves at different beats from different positions and produce different intensity of S_1.

• VENTRICULAR TACHYCARDIA

Abnormal pacemaker within the ventricles, also called ectopic focus, may result in ventricular tachycardia. This pathologic pacemaker starts producing repeated action potentials.

• ATRIAL FIBRILLATION (AFib)

When the SA node is directing the electrical activity of the heart, the rhythm is called “normal sinus rhythm.” The normal heart beats in this type of regular rhythm, about 60 to 100 times per minute at rest.

Atrial fibrillation (AF or AFib) is the most common irregular heart rhythm that starts in the atria. Instead of the SA node (sinus node) directing the electrical rhythm, many different impulses rapidly fire at once, causing a very fast, chaotic rhythm in the atria. Because the electrical impulses are so fast and chaotic, the atria cannot contract and/or squeeze blood effectively into the ventricle.

Instead of the impulse traveling in an orderly fashion through the heart, many impulses begin at the same time and spread through the atria, competing for a chance to travel through the AV node. The AV node limits the number of impulses that travel to the ventricles, but many impulses get through in a fast and disorganized manner. The ventricles contract irregularly, leading to a rapid and irregular heartbeat. The rate of impulses in the atria can range from 300 to 600 beats per minute.

S_1 SPLITTING

S_1 splitting may be:

1. Normal splitting
2. Pathological splitting
3. Pseudo splitting

1. NORMAL SPLITTING OF S_1

Normally the mitral valve closes slightly earlier than the tricuspid valve as left ventricular contraction is stronger than right ventricular contraction. Normally the split is so narrow that it's hardly observed.

During inspiration, intrathoracic pressure is reduced and venous return to the right heart is increased. So during inspiration, right ventricular contraction is delayed i.e. delayed T_1 (tricuspid component of S_1) and splitting of heart sound can be auscultated. This is called **inspiratory accentuation of S_1 splitting**.

During expiration, venous return to right heart decreases and tricuspid heart closes earlier hence, T_1 and M_1 can't be separated.

2. PATHOLOGICAL SPLITTING

If due to any reason, mitral valve is closed earlier while tricuspid valve is closed with undue delay, it will lead to production of wide splitting of S_1.

CONDITIONS LEADING TO EARLY M_1

All conditions which lead to pre-excitation of left ventricle lead to early closure of M_1 and M_1 will be heard earlier. These conditions include **left ventricular pre-excitation syndrome**. Due to some abnormal pathway between atria and ventricles, current reaches left ventricle pre-maturely e.g. in Wolff Parkinson White Syndrome.

CONDITIONS LEADING TO DELAYED T_1

The right bundle branch block may lead to delayed T_1. In this case, right ventricular contraction is pathologically delayed.

Right bundle branch block may be due to:

1. Congenital defect
2. Myocardial Infarction
3. Myocarditis

STENOTIC TRICUSPID VALVE

Most common cause of stenotic tricuspid valve is rheumatic heart disease. Another cause is carcinoid syndrome esp. carcinoid tumor. Carcinoid tumors may secrete hormones that cause thickening of the lining of heart chambers, valves and blood vessels. Chronic exposure of tricuspid and pulmonary valves to these hormones may lead to fibrosis of these valves.

ATRIAL SEPTAL DEFECT

Blood moves pathologically from left atrium to right atrium. So there is overload to the right side of the heart.

As it is overloaded so it will take longer time to pass to the ventricle and the valve will be kept open for longer time. Hence delayed closure of tricuspid valve will take place and wide splitting of M_1 and T_1 is observed.

3. PSEUDO-SPLITTING OF S_1

Normal opening of AV and pulmonary valves produce no sound.

LEFT VENTRICULAR HYPERTROPHY

In left ventricular hypertrophy e.g. in systemic hypertension, atrium has to push the blood with extra force so atria contract strongly against stiffened ventricle and produce a sound at the end of diastole called S_4. Just after S_4, ventricle will contract and produce S_1. Sometimes S_4 can be confused with splitting of S_1. This kind of splitting is called pseudo-splitting.

How to differentiate between S_4 and S_1?

S_4 is generally soft sound and mostly it is generated in left ventricle as left ventricular hypertrophy is more common than right ventricle. So S_4 can only be heard at apex beat.

If we auscultate apex beat and hear splitting of S_1 but when we auscultate tricuspid area and no splitting is observed, then we can conclude that sound at apex beat was due to S_4 as splitting of S_1 is best heard at tricuspid area.

EARLY SYSTOLIC CLICK OR EJACULATION SOUND

Sometimes an additional sound is heard just after S_1. This is due to opening of aortic and pulmonary valves in early systole due to diseased or stiff aortic and pulmonary valves and this sound is called early systolic click or Ejaculation sound.

Early systolic click may be due to aortic valve opening or pulmonary valve opening. If this click is heard just after S_1, this sound might be confused with true splitting of S_1.

Ejection click is either heard at aortic or pulmonary area so ejaculation sounds are best heard at base of the heart.