MASTERING SECOND HEART SOUND, S_2

DR. NAJEEB LECTURE NOTES

BY FATIMA HAIDER

KGMC

http://koracademy.com/

GENESIS OF S_2

S_2 is produced due to closure of semilunar valves and associated cardiohemic reverberation.

The closing of valves is due to the reversal of blood from major arteries to the ventricles when the ventricular pressure falls down.

Factors causing closure of valves (or reversal of blood from major arteries to ventricles)

1. Drop in ventricular pressure (onset of ventricular diastole)
2. Impedence (resistance of arterioles to blood flow)

If resistance to forward flow is high, blood will reverse back easily.

The closure of semilunar valves cause onset of vibration which begins in valve leaflets and move on to blood in ventricles and major arteries and then to ventricular valves. This vibration is called cardiohemic reverberation. This reverberation when comes in contact with chest wall produce S_2.

S_1 produced at beginning of ventricular systole

S_2 produced at end of ventricular systole

COMPONENTS OF S_2

S_2 has two components

1. Aortic component, A_2
2. Pulmonary component, P_2

AUSCULTATION

Aortic component of S_2 is best heard in 2^{nd} intercostal space in right parasternal area.

Pulmonary component of S_2 is best heard in 2^{nd} intercostal space in left parasternal area.

These are not the exact areas where semilunar valves are placed. These are the areas where the vibrations produced by closure of valves are best transmitted and heard.

Practically A_2 component is heard in all auscultatory areas as A_2 is stronger than P_2. P_2 is only heard in 2^{nd} intercostal space in left parasternal area.

To hear splitting of S_2, we auscultate at pulmonary area.

NORMAL SPLITTING OF S_2

Pulmonary component of S_2 is slightly delayed than aortic component.

Pulmonary circulation is low pressure and low impedence (resistance to forward flow) system.

Systemic circulation is high pressure and high impedence system.

When both ventricular systole ends, pressure in ventricles fall down and become less than the root of major arteries (i.e. aorta and pulmonary artery). Blood needs to reverse back from major arteries towards falling pressure in ventricles. Where the impedence is more, blood will reverse rapidly. Hence blood will reverse rapidly from aorta than from pulmonary artery. Due to rapid reversal, aortic valve closes earlier than pulmonary valve.

The time duration between the end of ventricular systole and closure of valves is called **Hangout interval.** Hangout interval is shorter for aortic valve closure than pulmonary valve closure.

S_2 DURING INSPIRATION AND EXPIRATION

During expiration, aortic component and pulmonary component are very close and can't be distinguished when auscultated.

During inspiration, aortic and pulmonary component interval increases and can be heard as separate units.

WHY PULMONARY COMPONENT IS DELAYED DURING INSPIRATION?

Expansion of chest cavity leads to decreased intra-thoracic pressure and venous return to right ventricle increases. Increased right ventricular filling leads to increased contraction of right ventricle and lead to prolonged ejection time due to which the closure of pulmonary valve is delayed P_2 component of S_2 is delayed.

During inspiration, venous return to left ventricle decrease due to expansion of vascular supply to lungs. Blood can stay in these vessels and hence less blood can return to left ventricle. As left ventricle take more time to be filled, its ejection time will be slowed.

So in inspiration aortic valve will close relatively earlier while pulmonary valve will close relatively late.

During inspiration as lungs and vascular beds in the lungs are expanding, impedence (resistance to forward flow) is thereby decreasing for pulmonary circulation. Due to decreased impedence, blood will take longer time to reverse and close the pulmonary valve. Hence hangout time will increase.

In older age, the physiological splitting of S_2 during inspiration is not pronounced due to stiff chest walls and lungs.

WIDE S_2 SPLITTING

- $A_2 – P_2$ component is pathologically widened
- Either P_2 is excessively delayed or A_2 is earlier
- This interval is widened further in inspiration

CAUSES OF DELAYED A_2

1. RIGHT BUNDLE BRANCH BLOCK

- Left ventricle depolarizes normally
- Right ventricular depolarization is delayed
- Pulmonary valve closure is delayed
- Wide S_2 split is observed

2. DYSFUNCTIONAL RIGHT VENTRICLE MYOCARDIUM

- Poor contraction of right ventricle
- To eject normal amount of blood, longer time is taken
- Delayed P_2 closure

3. PULMONARY VALVE STENOSIS

- Excessive resistance to blood outflow into pulmonary artery
- Prolonged contraction as blood flow is through narrow aperture

CAUSES OF EARLY A_2

The early A_2 is due to closure of aortic valve pre-maturely.

1. MITRAL VALVE REGURGITATION

When ventricle starts contraction, some blood moves backward through mitral valve. As a result, lesser blood goes towards aorta and ventricular systole lasts for shorter duration. Hence aortic valve closes earlier.

2. VENTRICULAR SEPTAL DEFECT

When ventricles start contraction, blood is shunted from left to right due to septal defect. As a result, lesser blood goes towards aorta and ventricular systole lasts for shorter duration. Hence aortic valve closes earlier.

3. WOLFF PARKINSON'S WHITE SYNDROME

- Right ventricle is contracted first
- Right ventricular depolarization is delayed
- A_2 is closed early

FIXED S_2 SPLITTING

In fixed S_2 splitting, the interval between A_2 and P_2 is widened but this interval is fixed during inspiration as well as expiration.

CAUSES OF FIXED S_2 SPLITTING

1. ATRIAL SEPTAL DEFECT

- Left atrium has slightly more pressure than right atrium so left to right shunting takes place and additional blood comes to right heart
- Additional blood in right ventricle leads to prolonged systole and P_2 is pathologically delayed which leads to splitting of S_2

There is no difference in degree of splitting during inspiration and expiration.

During inspiration, blood is pushed more towards the left atrium.

In patient with atrial septal defect, the shunting blood (from left to right atrium) adjusts itself. When during expiration, venous return to right atrium decrease and blood is pushed through the lungs (pulmonary circulation) more towards the left ventricle. As more blood is coming towards left atrium so shunting of blood increase from left to right atrium.

During inspiration, venous return to right atrium increase from systemic circulation but shunting from left to right decrease.

Shunted blood adjusts itself:

- During expiration when venous return is less, shunted blood is increased
- During inspiration, when venous return is increased, shunted blood is decreased.

PARADOXICAL/ REVERSE S_2 SPLITTING

This is reverse of normal S_2 splitting.

During expiration, S_2 splitting is observed and A_2 is heard after P_2. Normally A_2 is heard after P_2 (learn as mnemonic Associate Professor), but in paradoxical splitting aortic valve closure is delayed during expiration and A_2 is heard after P_2.

During inspiration, single sound is heard. A_2 is pathologically delayed and P_2 is normally delayed. Hence A_2 will fuse with P_2 and single sound is heard on auscultation.

CAUSES OF PARADOXICAL SPLITTING

1. AORTIC STENOSIS

- Aortic valve is narrow and exerts excessive resistance to outflow
- Left ventricle contracts for longer duration and hence A_2 is delayed

2. HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY

- Asymmetrical pathological hypertrophy of ventricular septum
- During left ventricular depolarization, the hypertrophic portion acts as outflow obstruction due to which outflow is delayed, systole is prolonged and A_2 is delayed.

3. LEFT BUNDLE BRANCH BLOCK

Left bundle depolarization is significantly delayed and A_2 is delayed

4. DYSFUNCTIONING OF LEFT VENTRICULAR MYOCARDIUM

Left ventricle contracts poorly, but for prolonged time and A_2 is delayed.

INTENSITY OF S_2

Whenever pressure in roots of major arteries is low, valves will close slowly.

Whenever pressure in roots of major arteries is high, valves will close loudly.

- **SOFT P_2 COMPONENT**
  It is mainly due to **Pulmonary stenosis.** Due to pulmonary stenosis, pulmonary valve exerts high resistance due to which pressure in ventricles become high while pressure in pulmonary artery is low. Hence the valve will close softly.

- **SOFT A_2 COMPONENT**
  Due to:
  1. Aortic valve stenosis
  2. Aortic Regurgitation
  3. Mitral Regurgitation

- **LOUD P_2 COMPONENT**

It is mainly due to **Pulmonary hypertension.** High pressure in pulmonary artery will cause the valve to close loudly.
P_2 is normally heard only at pulmonary area so if P_2 is heard at other oscultatory areas, it signifies loud P_2.

- **LOUD A2 COMPONENT**
  It is mainly due to **aortic hypertension.** High pressure in aortic artery will cause the valve to close loudly.