

# MASTERING SECOND HEART SOUND, S<sub>2</sub>

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## GENESIS OF S<sub>2</sub>

S<sub>2</sub> 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<sub>2</sub>.

S<sub>1</sub> produced at beginning of ventricular systole

S<sub>2</sub> produced at end of ventricular systole

## COMPONENTS OF S<sub>2</sub>

S<sub>2</sub> has two components

1. Aortic component, A<sub>2</sub>
2. Pulmonary component, P<sub>2</sub>

## AUSCULTATION

Aortic component of S<sub>2</sub> is best heard in 2<sup>nd</sup> intercostal space in right parasternal area.

Pulmonary component of S<sub>2</sub> is best heard in 2<sup>nd</sup> 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<sub>2</sub> component is heard in all auscultatory areas as A<sub>2</sub> is stronger than P<sub>2</sub>. P<sub>2</sub> is only heard in 2<sup>nd</sup> intercostal space in left parasternal area.

To hear splitting of S<sub>2</sub>, we auscultate at pulmonary area.

## NORMAL SPLITTING OF S<sub>2</sub>

Pulmonary component of S<sub>2</sub> is slightly delayed than aortic component.

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

Systemic circulation is high pressure and high impedance 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 impedance 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<sub>2</sub> 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<sub>2</sub> component of S<sub>2</sub> 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, impedance (resistance to forward flow) is thereby decreasing for pulmonary circulation. Due to decreased impedance, 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<sub>2</sub> during inspiration is not pronounced due to stiff chest walls and lungs.

## WIDE S<sub>2</sub> SPLITTING

- A<sub>2</sub> – P<sub>2</sub> component is pathologically widened
- Either P<sub>2</sub> is excessively delayed or A<sub>2</sub> is earlier
- This interval is widened further in inspiration

### CAUSES OF DELAYED A<sub>2</sub>

#### 1. RIGHT BUNDLE BRANCH BLOCK

- Left ventricle depolarizes normally
- Right ventricular depolarization is delayed
- Pulmonary valve closure is delayed
- Wide S<sub>2</sub> 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<sub>2</sub> 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<sub>2</sub>

The early A<sub>2</sub> 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<sub>2</sub> is closed early

## **FIXED S<sub>2</sub> SPLITTING**

In fixed S<sub>2</sub> splitting, the interval between A<sub>2</sub> and P<sub>2</sub> is widened but this interval is fixed during inspiration as well as expiration.

### **CAUSES OF FIXED S<sub>2</sub> 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<sub>2</sub> is pathologically delayed which leads to splitting of S<sub>2</sub>

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<sub>2</sub> SPLITTING**

This is reverse of normal S<sub>2</sub> splitting.

During expiration, S<sub>2</sub> splitting is observed and A<sub>2</sub> is heard after P<sub>2</sub>. Normally A<sub>2</sub> is heard after P<sub>2</sub> (learn as mnemonic Associate Professor), but in paradoxical splitting aortic valve closure is delayed during expiration and A<sub>2</sub> is heard after P<sub>2</sub>.

During inspiration, single sound is heard. A<sub>2</sub> is pathologically delayed and P<sub>2</sub> is normally delayed. Hence A<sub>2</sub> will fuse with P<sub>2</sub> 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 auscultatory areas, it signifies loud  $P_2$ .

- **LOUD  $A_2$  COMPONENT**

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