

# Student's Corner—4

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## 1. How to differentiate volume overload and pressure overload of right ventricle from left parasternal heave?

Answer: Left parasternal movements (pulsations or heave) could originate from right ventricle (RV), left atrium (LA), (or) descending thoracic aorta.

**A.** The RV is located anteriorly and left parasternal pulsations are produced by its enlargement. This abnormal RV pulsation could be due to volume overload (for e.g., atrial septal defect (ASD), partial anomalous pulmonary venous connection [PAPVC] or total anomalous pulmonary venous connection [TAPVC], tricuspid regurgitation, pulmonary regurgitation). It could also be due to pressure overload (for e.g., pulmonary stenosis [PS] or pulmonary arterial hypertension [PAH]).

1. The following features can identify volume overload RV pulsation:

- RV impulse upstroke is not rapid.
- Early and midsystolic.
- There is no sustained peak.
- Early descent of the impulse.
- Easily obliterated by mild pressure with the palm of the examiner.

2. Pressure overload RV pulsation may have the following features:

- RV impulse upstroke is rapid.
- Early late systole.
- Sustained peak longer than half of systole.
- Not easily obliterated by palm pressure, the degree of pressure needed to obliterate the heave can approximately guess intraventricular pressure of RV.
- Accompanying collateral signs may give clues to RV pressure load like the presence of diastolic shock (palpable  $P_2$  in 2nd left intercostal space), constant ejection click, and large “a” waves in jugular venous pulse. In the earlier years before echo was available, RV systolic time

intervals, RV apex cardiogram, and cardiokymography were useful.

- B.** Severe mitral regurgitation (particularly anteriorly directed) can produce left parasternal “heave” (or) pulsation due to systolic expansion of the LA, which is in the central chest, located posteriorly. This is a transmitted expansile pulsation and can be differentiated from RV heave, as it is late systolic in timing.
- C.** Occasionally, the expansile pulsation of large aneurysm of descending thoracic aorta may be transmitted to the left parasternal space.
- D.** In corrected transposition of great arteries in the left-sided heart, the anteriorly located ascending aorta pulsations may also be palpable in the 2nd left and 3rd left intercostal spaces.
- E.** The ability to appreciate the left parasternal heave and interpret will also depend on chest wall thickness, abnormalities, lung status, pericardial disease, the sensitivity of the palm of the observers, and their clinical experience.

Comment:

To be noted and understood is that there indeed is significant subjectivity in appreciating the distinguishing clinical features.

## 2. What is the concept of hangout interval and its influence on second heart sound ( $S_2$ ) in physiological and pathological status?

This question is going to be answered under the following question-answer format:

1. What is hangout interval?
2. Are there differences between the right and left sides of the heart?
3. Why?
4. What are the normal hangout intervals?

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5. How does it affect  $S_2$  in normal individuals?
6. What is the behavior of  $S_2$  in normal individuals with physiological splitting and its relation to hangout interval?
7. What are the types of  $S_2$  splitting?
8. How does the hangout interval influences the various  $S_2$  splitting abnormalities?
9. What decides the loudness of each component of  $S_2$ ?
10. What are the factors influencing our ability to appreciate  $S_2$  variations at the bedside?
11. What are the mimics of  $S_2$  abnormalities?

## Questions/Answers

### 1. What is hangout interval?

It is the time gap between the end of systole of the corresponding ventricle and the closure of the corresponding semilunar valve, that is, RV—pulmonary valve/left ventricle (LV)—aortic valve. It occurs between the pressure curve of the chamber and the related semilunar valve at the level of incisura, when the aortic (or) pulmonary valves close but the actual  $S_2$  occurs milliseconds later, the genesis of which is possibly due to after vibrations originating from the valve, ventricle and great vessel of the corresponding side (→Fig. 1) (hangout interval named by Shaver et al).

### 2. Are there any differences between the right and left sides of the heart?

Answer: Yes

The hangout interval on the right side is longer than the left side of the heart.

### 3. Why?

Answer: The hangout interval is influenced by the vascular resistance (or) impedance in the respective circulations, that is, pulmonary and systemic circulation. The pulmonary vascular bed and system are low resistant, very high capacitance capable, and hence highly compliant and accommodative of the blood volume.

But in the systemic circulation, the vasculature is high-resistant and low-compliant. As a corollary to that, the hangout interval is longer on the right side than the left side, so aortic component of second heart sound ( $A_2$ ) of  $S_2$  comes first and precedes pulmonary component of second heart sound ( $P_2$ ) in the normal circumstances.

*Hangout Interval and Vascular Resistance are Inversely Related*

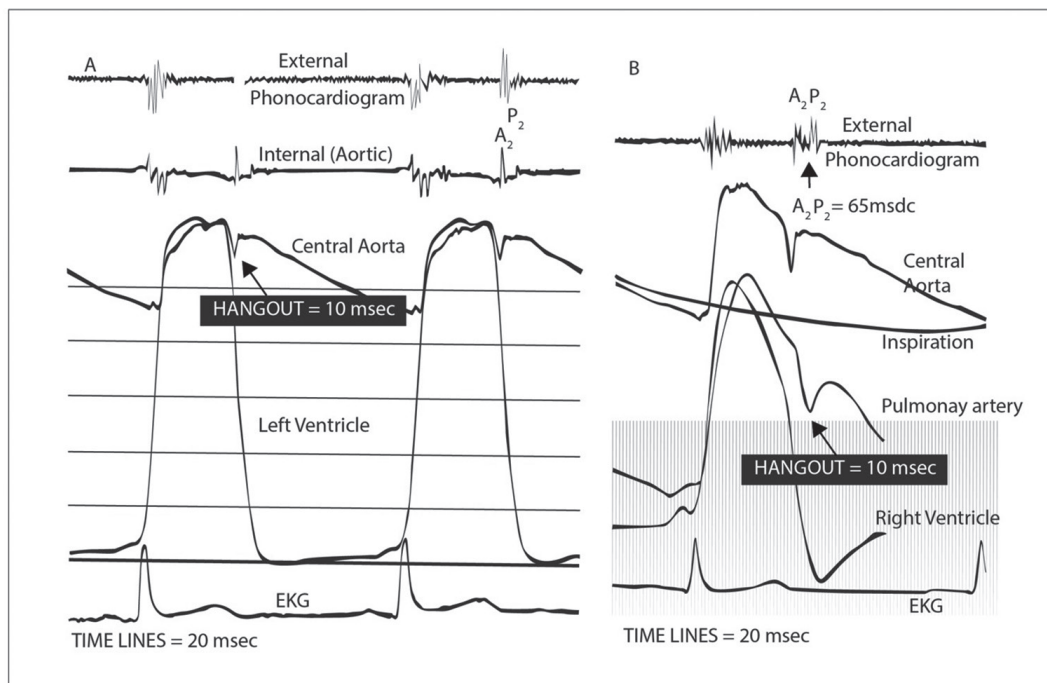
### 4. What are the normal hangout intervals?

Answer: Pulmonary hangout interval is 30 to 120 msec (average around 60 msec). Aortic hangout interval is 5 to 15 msec (average less than 10 msec)

Note: The time intervals quoted may vary from one study to another.

### 5. How does it affect $S_2$ ?

Answer: Hangout interval and vascular resistances of pulmonary and systemic circulation are related inversely, so higher the vascular resistance, shorter is the hangout interval and vice versa. So, obviously the duration of hangout interval has a very significant impact on  $S_2$  splitting and its clinical and phonocardiographic variabilities which are very important in clinical cardiology



**Fig. 1** Left ventricular (LV) aorta and right ventricular (RV) pulmonary artery (PA) pressure tracing overlaps to show the hangout intervals on left and right side of the heart.

## 6. What is the behavior of $S_2$ in normal individuals with physiological splitting and its relation to hangout interval?

Answer: Physiological  $S_2$  splitting is recognized by audibility of split  $S_2$  with both components during inspiration and ability to clinically hear  $S_2$  as a single sound in expiration. This is because of the following events:

Inspiration  $\rightarrow$   $\downarrow$  Pulmonary vascular resistance  $\rightarrow$   $\uparrow$  Hangout interval (RV–pulmonary circulation)  $\rightarrow$  delays pulmonary valve closure  $\rightarrow$  delayed  $P_2$   $\rightarrow$  audible splitting of  $S_2$  ( $A_2 - P_2$  interval widens),  $A_2 - P_2$  interval increases to 40 to 80 msec

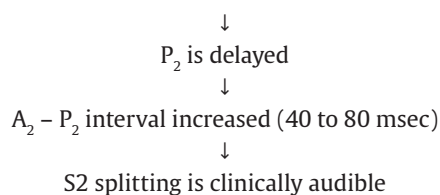
Expiration  $\rightarrow$   $\downarrow$  Hangout interval  $\rightarrow$  Early pulmonary valve closure  $\rightarrow$  Earlier  $P_2$   $\rightarrow$  so  $A_2 - P_2$  interval narrows (about 30 m/sec). Hence,  $S_2$  is heard as a single sound as the  $A_2 - P_2$  interval of less than 30 msec is not clinically appreciable to be heard as two components.

## 7. What are the abnormalities of $S_2$ splitting?

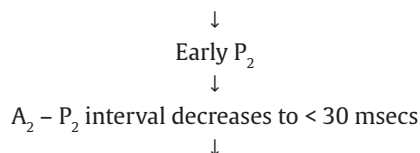
- Normal physiological splitting.
- Wide mobile splitting.
- Wide fixed splitting.
- Narrow splitting.
- Paradoxical (reversed) splitting.
- Single  $S_2$ .
  - Only  $A_2$  audible.
  - Only  $P_2$  audible.
  - Fusion of  $A_2$  and  $P_2$ .

## 8. How does the hangout interval influence the various $S_2$ splitting abnormalities?

- Physiological splitting = Inspiration  $\rightarrow$   $\uparrow$  Hangout interval as the pulmonary vascular resistance falls



Expiration  $\downarrow$  Hangout interval as the pulmonary vascular resistance increase



Clinically  $S_2$  split is not audible; hence,  $S_2$  is audible as a single sound

- $S_2$ —Narrow mobile splitting.
  - $A_2 - P_2$  interval less than 40 msec respiratory variation present.
  - $A_2 - P_2$  appreciable in the supine position, but audible as a single sound on standing.

- As in pulmonary arterial hypertension, the pulmonary vascular resistance is increased; so, hangout interval is abbreviated and  $A_2 - P_2$  interval is narrowed.

In PAH  $\rightarrow$   $\uparrow$  Pulmonary vascular resistance  $\rightarrow$   $\downarrow$  Hangout interval  $\rightarrow$   $A_2 - P_2$  interval narrowed  $\rightarrow$   $S_2$  narrow splitting.

- Wide splitting of  $S_2$ .

Wide fixed  $S_2$  = ASD; PAPVC with ASD; TAPVC; single atrium; severe valvular PS.

Wide mobile  $S_2$  = right bundle branch block (RBBB); PS; PAPVC with no ASD.

- Wide splitting of  $S_2$  in ASD/PAPVC with ASD/TAPVC without PAH is because of decreased pulmonary vascular impedance with high pulmonary blood flow.
- Fixity of the  $S_2$  split is because of the unchanged  $A_2 - P_2$  interval during the respiratory phases, as both the ventricles share a common reservoir that is interconnected atria.
- Wide splitting by definition indicates audible  $S_2$  splitting in both inspiration and expiration.
- The expiratory  $S_2$  splitting in these conditions is due to prolonged hangout interval in both respiratory phases, due to highly compliant pulmonary vascular system along with large pulmonary blood flow.
- Wide but mobile split in RBBB or in Wolff–Parkinson–White (WPW) syndrome (or) LV pacing (or) LV origin of the premature beat is due to the electrical delay between the two ventricles.
- Wide but mobile  $S_2$  split in pulmonary valve stenosis is due to prolonged RV ejection time with preserved phasic respiratory changes.
- Wide but mobile  $S_2$  split in PAPVC with intact atrial septum is due to A) increased pulmonary blood flow  $\rightarrow$  increased pulmonary vascular capacitance  $\rightarrow$   $\downarrow$  Pulmonary vascular resistance  $\rightarrow$   $\uparrow$  Hangout interval. It is mobile split because the atrial septum is intact; hence, there is no common venous reservoir for both the ventricles.

- Eisenmenger ASD and wide fixed  $S_2$  splitting

- $S_2$  split is wide, as it is audible in both phases of respiration.
- $S_2$  split is narrow with severe PAH, because the pulmonary vascular resistance is close to systemic vascular resistance.
- Because of high pulmonary vascular resistance, the hangout interval is significantly reduced, leading to a decrease in  $A_2 - P_2$  interval, so the two components are separated by just 30 to 50 msec
- $A_2 - P_2$  interval fixity is maintained as the common venous reservoir (both atria) is still present.
- It is also relevant to consider that RV dysfunction prolonging the RV ejection time and the electrical delay due to RBBB may also delay  $P_2$  increasing  $A_2 - P_2$  interval; hence, widening the  $S_2$  split.

- E) It is useful to remember that a wide and fixed split may also be audible in those with right heart failure, complete RBBB, and constrictive pericarditis.
- Fixity of split  $S_2$  is due to constant and near-identical RV stroke volume in right heart failure and constrictive pericarditis.
  - Due to RV dysfunction, RV cannot increase its diastolic volume during inspiratory augmentation, and pulmonary vascular resistance remains almost constant as the lungs are fluid loaded and congested due to left heart dysfunction.
- F) Ventricular septal defect (VSD) (isolated).
1. Small VSD with the small left-to-right shunt.
    - $S_2$  has a physiological splitting.
  2. Large VSD with the large left-to-right shunt and no PAH.
    - Leads to increased pulmonary blood flow  $\rightarrow \uparrow$  Pulmonary capacitance.
    - $\rightarrow$  Increased hangout interval.
    - $\rightarrow$  Increased  $A_2 - P_2$  interval.
    - $\rightarrow$  Wide  $S_2$  splitting but mobile.
  4. Large VSD with severe PAH (Eisenmenger syndrome).
    - Leads to increased pulmonary vascular resistance  $\rightarrow$  equaling the systemic vascular resistance  $\rightarrow$  hang-out intervals on both right and left circulations are near identical  $\rightarrow$  hence,  $S_2$  will be single and loud.
    - If one can hear splitting of  $S_2$  in VSD with PAH, it means that there is still left-to-right shunt and it is still operable.
- G) Patent ductus arteriosus (PDA).
1. Small PDA + small left-to-right shunt.
    - $S_2$  is physiologically split.
  2. Large PDA + large left-to-right shunt + no PAH.
    - Increased LV volume leads to delay of  $A_2$  and narrowing of  $A_2 - P_2$  interval. As the shunt is increasingly delayed  $A_2$ ,  $S_2$  may become single due to fusion of  $A_2$  and  $P_2$  and sometimes even reversed split, that is,  $A_2 - P_2$  will become  $P_2 - A_2$  due to prolonged LV ejection time. But this phenomenon may not be appreciated because of the masking loud murmur.
  3. PDA + severe PAH + Eisenmenger syndrome.
    - $S_2$  split will be normal with normal respirophagic changes but  $P_2$  will be loud.
    - $A_2 - P_2$  interval may be 30 msec due to increased hangout interval of right side and so has narrow  $S_2$  splitting but with physiological respirophagic changes.
- H) Paradoxical (reversed) splitting.
- It is due to delay in  $A_2$ , leading to  $A_2 - P_2$  sequence becoming  $P_2 - A_2$ , that is, reversed relation.
    - Due to the above changes, the  $S_2$  split is heard in the expiratory phase, but  $S_2$  becomes single during inspiration, that is, it is the reverse of what happens in physiological splitting.
    - Paradoxical splitting always is indicative of significant cardiac disease and due to prolonged left ventricular ejection time longer than right ventricular ejection time, like in aortic stenosis (AS), hypertrophic obstructive cardiomyopathy (HOCM), acute myocardial infarction (AMI), severe dysfunction of the left ventricle, and also in severe systemic hypertension.
    - It occurs as well in left bundle branch block (LBBB) due to electrical delay.
    - Paradoxical splitting is heard well in LBBB and HOCM but not so well in AS and AMI, as  $A_2$  is muffled in that case scenario.
- I) Single  $S_2$ .
1.  $P_2$  absent (or) inaudible.
  2.  $A_2$  absent (or) in audible.
  3.  $A_2 + P_2$  fusion/simultaneous occurrence.
  4. Single valve—only aortic valve present, only pulmonary valve present, and common truncal valve.
    - $P_2$  absent/inaudible = severe PS/pulmonary atresia.
      - Absent pulmonary valve.
    - Transposition of great arteries (posteriorly located PA)
    - $A_2$  absent/inaudible = severe calcific AS.
      - Aortic valve atresia, hypoplastic left heart syndrome.
    - The absent aortic valve, dysplastic aortic valve.
    - $A_2 + P_2$  Fusion = large VSD with Eisenmenger syndrome.
      - Single ventricle.
      - Double outlet RV/LV.
- Single valve present = pulmonary atresia.
    - Absent pulmonary valve.
    - Aortic atresia.
  - Common valve = truncus arteriosus
- J) Mimics of  $S_2$  splitting abnormalities
- $S_2$  with opening snap.
  - $S_2$  with  $S_3$ .
  - $S_2$  with pericardial knock.
  - $S_2$  with tumor plop.
- K) Mimics of single  $S_2$
- Very soft  $A_2$  (or)  $P_2$ .
  - Hyperinflated lungs.
  - Thick chest wall.
  - Cardiac malpositions.
  - All conditions with paradoxical splitting when the  $A_2 - P_2$  or  $P_2 - A_2$ .
    - Interval is lesser than 20 msec.
  - $A_2$  may be masked by the loud murmur of VSD (or) AS.

L) Both S<sub>1</sub> and S<sub>2</sub> may be sounding distant with decreased intensity in those with severe myocardial dysfunction, cardiogenic shock, AMI, pericardial effusion, and constrictive pericarditis.

M) A<sub>2</sub> is always best heard in the second right intercostal space.

*P<sub>2</sub> and S<sub>2</sub> Split are Best Heard in 2ND Left Intercostal Space (Applicable and True in People with No Cardiac Malpositions)*

N) S<sub>2</sub> split audible at apex area is always abnormal and indicates ASD, PAH, large dilated RV.

O) Intensity of S<sub>2</sub>, A<sub>2</sub>, and P<sub>2</sub> will depend on pressures in the corresponding great vessel, mobility of the corresponding semilunar valves, and transvalvular flow.

P) For S<sub>2</sub> split evaluation, always auscultate in lying down position and also standing.

**Note**

Many of the typical clinical features of the heart sounds may change in those with arrhythmias, postcardiac surgery when on devices like pacemakers, LV/RV assist devices, cardiac transplants, and also in those with a complex combination of structural and functional abnormalities of the heart, either congenital or acquired.