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?

- A) Normal physiological splitting.
- B) Wide mobile splitting.
- C) Wide fixed splitting.
- D) Narrow splitting.
- E) Paradoxical (reversed) splitting.
- F) 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?

- A) 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

- B) 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.

- C) 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.

- D) 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 hang-out 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) inaudible.
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 S₁ and S₂ may be sounding distant with decreased intensity in those with severe myocardial dysfunction, cardiogenic shock, AMI, pericardial effusion, and constrictive pericarditis.

M) A₂ is always best heard in the second right intercostal space.

P₂ and S₂ Split are Best Heard in 2ND Left Intercostal Space (Applicable and True in People with No Cardiac Malpositions)

N) S₂ split audible at apex area is always abnormal and indicates ASD, PAH, large dilated RV.

O) Intensity of S₂, A₂, and P₂ will depend on pressures in the corresponding great vessel, mobility of the corresponding semilunar valves, and transvalvular flow.

P) For S₂ 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.