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The Mechanism of S2 (Second Heart Sound) Splitting in Normal Physiology and Congenital Heart Defects

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Abstract

Splitting of the second heart sound is a difficult concept which is stated, rather than explained, in many medical texts. The splitting into the pulmonary valve and aortic valve components occurs in physiology during inspiration, which is due to later closure of the pulmonary valve as well as earlier closure of the aortic valve, determined by changes in intrathoracic pressure and the capacitance of the pulmonary vasculature. Fixed splitting of the second heart sound describes a situation where the second heart sound is split in both inspiration and expiration and is classically described in an atrial septal defect. The evidence behind these mechanisms is discussed in the following article.

Keywords: Blood; Pulmonary; Pediatrics; Cardiology; Heart; Hypertension

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Introduction

There is a great deal of confusion and complexity surrounding the second heart sound and how it "splits" in normal individuals and in patients with congenital heart defects, such as atrial septal defect and ventricular septal defect. It is well understood that the second heart sound "splits" during the inspiratory phase of respiration [1], but the mechanism behind this splitting is not clearly documented in popular pediatric medical textbooks. Similarly, the presence of second heart sound splitting is widely reported as being a clinical feature of congenital heart defects [2].

The first heart sound is formed from the closing of the mitral and tricuspid valves, whereas the second heart sound is formed from the closing of the aortic and pulmonary valves [3]. During inspiration, and throughout the entire respiratory cycle in some congenital heart defects, the pulmonary valve may close after the aortic valve, creating a "split" second heart sound. When the second heart sound is split during inspiration, but not during expiration, this is termed "physiological splitting".

When the second heart sound is split throughout both expiration and inspiration, this is termed a "fixed and split" second heart sound. The mechanism underlying the two separate events of physiological and pathological splitting is outlined below.

By discussing the evidence behind the mechanism of the splitting of the second heart sound in normal physiological instances, and congenital heart defects, we hope to ease how medical students and training pediatricians learn and understand the clinical findings in the pediatric cardiovascular examination.

Physiologically split-second heart sound

As discussed above, the second heart sound (S2) is physiologically "split" during inspiration but not during expiration. Studies have demonstrated that the aortic valve shuts first, producing the first component of the second heart sound and the pulmonary valve the second component [2].

In a study of 162 patients of ages 1-80 years, by Harris and Sutton [1], 84% of patients had a split-second heart sound on inspiration with an interval of >0.02 seconds between the aortic and pulmonary valve component. 90% of these patients had a single second heart sound during expiration. These findings were based on the requirement for the second heart sound to be split by >0.02 seconds before it is considered audible on auscultation [4]. Traditionally it has been thought that the mechanism of S2 splitting is due to delayed closure of the pulmonary valve during inspiration, although it is also believed that earlier closure of the aortic valve during inspiration also contributes.

Later closure of pulmonary valve during inspiration

A study by Aygen and Braunwald demonstrated that in 51 patients aged from 4 to 49 years, the pulmonary valve closed on average 32.6 milliseconds later during inspiration [5]. This data was replicated in studies by Castle and Jones on 80 normal children aged from 5 to 15 years, which showed an average later closure of the pulmonary valve of 13 milliseconds during inspiration [6].

The mechanism of the delayed closure of the pulmonary valve is due to increased ejection time of the right ventricle, caused by an increase in the venous return to the right atrium because of decreased intrathoracic pressure during inspiration [2,7,8]. There is also a decrease in the pressure of the pulmonary artery during inspiration, which may contribute to later closure of the pulmonary valve.

Earlier closure of aortic valve during inspiration compared to expiration

Historically, it was considered that the inspiratory splitting of the second heart sound was due to the pulmonary valve changes as discussed above. However, Harris and Sutton showed that the aortic valve closed earlier during inspiration, compared to expiration, contributing between 21-36% of the time of the 'split' [1]. This was also demonstrated by Castle and Jones who showed that the change in the aortic valve closure timing contributed, on average, 35% to S2 splitting [6].

A suggested mechanism for this is that the blood from the augmented right ventricular output during inspiration reaches the left ventricle after passing through the vascular bed, and therefore does not reach the left ventricle for a few heartbeats, by which point the patient will be in expiration. As a result, this augmented volume increases left ventricular ejection time during the following expiratory phase. Therefore, the closure of the aortic valve is comparatively earlier during inspiration than expiration [9].

Atrial septal defect and S2 splitting

Atrial septal defects are a common congenital heart defect, affecting 6 in 10,000 live births [10], whereby there is a communication between the two atria (ASD) or the atria and ventricles (AVSD). In ASD, there is fixed splitting of the second heart sound, meaning that the second heart sound is split throughout both expiration and inspiration.

There are several proposed theories for the mechanism of fixed splitting of the second heart sound in atrial septal defect. These include: Right bundle branch block leading to augmented right ventricular systole, left to right shunt through the septal defect leading to increased right ventricular filling and delayed emptying, and dilatation of the pulmonary vasculature leading to delayed pulmonary valve closure [11,12]. Below, we will discuss the evidence for each of the proposed mechanisms.

Right bundle branch block: The presence of right bundle branch block (RBBB) is a well-known conduction problem in patients with ASD. In a study by Barber et al. [13], the authors noted 52 of 62 cases of ASD had a widely split-second heart sound. They reasoned that the "mean pressure in the right ventricle and pulmonary artery may be normal or little above normal in the presence of splitting" and therefore attributed the splitting of the second heart sound to RBBB, which 95% of patients demonstrated on their ECG, or to delayed emptying of the "over-filled" right ventricle (see section 2: left to right shunt, below). Their explanation was a prolonged right ventricular ejection time due to the conduction defect in RBBB.

However, hemodynamic studies of patients with ASD [14] demonstrate that there is no prolongation of time interval between the onset of ventricular depolarization and of right ventricular contraction, which contradicts the above reasoning.

Left to right shunt: Historically, the proposed mechanism of S2 splitting in ASD was that a left-to-right shunt caused increased loading on the right ventricle, leading to delayed emptying and therefore delayed closure of the pulmonary valve. As both sides of the circulation are linked, there is no differential effect on either ventricle by respiration, and therefore splitting of the second heart sound remains constant throughout the respiratory cycle. As stated above, several studies have demonstrated that surgical correction of the atrial septal defect leads to reversal of S2 splitting. Perloff and Harvey [8] showed that 11 of the 13 patients with atrial septal defects undergoing repair of the defect had a split-second heart sound on inspiration, and a single second heart sound on expiration after surgery.

Their pre-operative phonocardiograms had shown fixed splitting throughout the respiratory cycle. The authors suggested that fixed S2 splitting in ASD was "a manifestation of right-sided diastolic hypervolemia" due to left to right shunting. They hypothesized that the persistence of S2 splitting in the other cases may have been due to persistent shunting between the pulmonary vasculature.

This evidence was supported by Leatham and Gray [15], who demonstrated by phonocardiography that the duration of right ventricular systole was 0.02 seconds greater than left ventricular systole throughout the entirety of the respiratory cycle. Further evidence for the importance of shunting came from studies by Aygen and Braunwald [5] of 118 patients with ASD. In 31 patients undergoing operative closure of their septal defect, 29 of these patients had a "completely abolished" left-to-right shunt, with the other 2 patients' shunts being "reduced" but "not completely eliminated". In 29 of the 31 patients, there was a decrease in the difference between closure of the aortic and pulmonary valve during expiration, the only exceptions being a patient that did not exhibit S2 splitting before their operation, and a patient diagnosed with pulmonary hypertension.

They argue that the shunt is greatest during expiration, evidenced by higher oxygen saturations of the pulmonary artery during expiration, whereas during inspiration right ventricular systole is lengthened by increased systemic return in a similar mechanism to normal physiology.

Dilated pulmonary vasculature: In 1971, Kumar et al. [11] studied 52 patients with ASD (and 4 normal controls). In those with ASD the average values for the time between the onset of depolarization and contraction of the left and right ventricle were identical to normal controls. This suggests that there was no

increase in the length of right ventricular systole, and therefore the increased RV volume and emptying time did not explain the split S2 in this instance.

The authors argue that P2 (the audible component of the pulmonary valve closure) occurs at the timing of the upstroke in pressure in the pulmonary artery (known as the 'incisura'), and that this is delayed in ASD due to changes in the capacitance of the pulmonary vasculature, which causes later audible closure of the pulmonary valve. This evidence is further supported by O'Toole et al. [12] who argue that there is documented persistence of S2 splitting after surgical ASD repair, and that there is no significant correlation between the amount of shunting and the width of S2 splitting, suggesting that the cause is not due to left-to-right shunting through the ASD.

By investigating 27 patients undergoing diagnostic cardiac catheterization, the authors found that in all patients with normotensive ASD, all patients had wide, fixed S2 splitting. In these patients, there was not an increased duration of right ventricular systole. Instead, as above, the authors hypothesize that the ASD leads to a chronic dilated pulmonary artery, leading to delayed P2. The authors commented that patients with idiopathic dilatation of the pulmonary artery also have fixed, splitting of the second heart sound, despite there being no shunt in the cardiovascular system, strengthening their argument that the dilated pulmonary vasculature was the cause of the fixed, and split, S2.

Ventricular septal defect

Ventricular septal defects (VSDs) are one of the most common congenital heart defects, occurring in 2.6-5 per 1000 live births, and occur in almost 50 percent of all patients with congenital heart disease [16,17]. Clinical manifestations depend on the size of the defect, and most patients present in the neonatal period with symptoms ranging from an isolated murmur to severe heart failure [18].

It had initially been demonstrated that in patients with VSD, the second heart sound followed that of normal individuals (in that there is physiological splitting). In 1960, Shafter used a phonocardiogram to show that splitting in 6 patients with VSD was no different to splitting in normal subjects [9].

However, there have been numerous studies which contradict these findings. Leatham and Segal [4] used phonocardiography from different sites in 23 patients with isolated ventricular septal defects. They noted that splitting was abnormally wide (from 0.03 to 0.066 s) in expiration in 9 of 13 subjects with small VSDs and from 0.034 to 0.075 s in 8 of 10 patients with large VSDs.

This was corroborated by Harris et al. [19] who investigated 11 cases of VSD (all of which had left to right shunt confirmed by cardiac catheter and no ASD or pulmonary hypertension) with phonocardiography during, and after, held expiration and inspiration, and during a prolonged period of exaggerated respiration. Mean interval on expiration was 0.035 s *vs.* 0.051 s on inspiration, and therefore could be considered to some degree fixed. This led the authors to conclude that fixed second heart sounds are therefore not specific for ASDs but can also be found in VSDs.

They hypothesized that in expiration there is an increase in return of pulmonary flow to the left heart and increase in shunt flow. This causes an increase in the intensity in the pan systolic murmur which 'spills through' A2 [19]. The increase in enveloping noise masks A2, leaving P2 to be perceived as a single, or narrowly split, 2nd sound.

In inspiration, there is decreased return to the left heart and so reduction in shunt flow and reduction in murmur intensity. Therefore, there is a more sharply defined A2 which can be easily distinguished from P2. Thus, it had been assumed that A2-P2 moves normally with respiration when this may be a product of 'masking' of the sound by the loud murmur.

Discussion and Conclusion

Although evidence behind our understanding of the mechanism of the "splitting" of the second heart sound has improved, most of the data for this is from studies of the 1950s and 1960s and there has been little change in our understanding. With the introduction of echocardiography, the anatomical classification of congenital heart defects can be characterized, although the strong relevance of clinical examination and findings continues in modern practice.

The second heart sound is comprised of closure of the aortic and pulmonary valve, and these can occur at different times, resulting in a 'split' second heart sound [3]. This can occur physiologically during inspiration but can also occur in various pathological states.

Physiological S2 splitting is thought to occur due to changes in the timing of the closure of the pulmonary and aortic valves during the respiratory cycle. During inspiration there is increased venous return to the right side of the heart and decreased pressure in the pulmonary artery, causing lengthening of the duration of right ventricular output and leading to later closure of the pulmonary valve.

This "inspiratory splitting" is augmented by earlier closure of the aortic valve due to decreased length of left ventricular systole, the mechanism for which is not completely understood. In expiration, however, it is believed that the pulmonary vasculature recoils. This enables increased venous return and an increased left ventricular stroke volume. The increased volume delays the closure of the aortic valve, thus bringing it in line with the pulmonary valve closure and therefore creating a single second heart sound [1,6,9].

The "fixed and split" S2 that occurs in atrial septal defect patients was originally thought to be due to the equalization of pressures between the left and right ventricles due to shunting through the ASD [15,19]. However, there is data to suggest that fixed and split-second heart sound persists after surgical closure and is due to increased capacitance of the pulmonary vascular bed due to chronic change in blood flow across the defect [12].

Typically, it is described in cardiology texts that the second heart sound is not split in VSD patients [9]. However, evidence suggests that there is a split, fixed S2, but this is perceived as physiological splitting due to masking of the closure of the aortic valve by a pan systolic murmur which is loudest in expiration [4,19].

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