Mechanisms of Fixed Splitting of the Second Heart Sound

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Proper clinical analysis of the second heart sound has only recently been emphasized.1 An understanding of the normal second heart sound has increased the diagnostic usefulness of its variations in congenital and acquired heart disease. In atrial septal defect, as emphasized by Wood,9 the aortic and pulmonic components of the second heart sound are not only widely separated, but the split remains fixed throughout the respiratory cycle. The following study was designed to investigate the physiology of fixed splitting of the second heart sound in atrial septal defect and in 3 other categories in which this phenomenon has been observed—right and left bundle-branch block with right ventricular failure, and mitral insufficiency with right ventricular failure.

Materials and Methods

A total of 69 cases were studied. They consisted of 25 normal controls, 13 patients with atrial septal defect, 10 with pure mitral insufficiency, 11 with right bundle-branch block, and 10 with left bundle-branch block. The material included patients in the Georgetown University Medical Center and in the Clinic of Surgery, National Heart Institute, Bethesda. All patients had electrocardiograms and chest fluoroscopy. In the group with atrial septal defect the diagnoses were established by right heart catheterization. The group with pure mitral insufficiency had left heart catheterization. The preoperative diagnosis of atrioventricularis communis was established by the additional use of dye dilution curves with the injections made into the left atrium and left ventricle.2

The patients with right and left bundle-branch block had congenital conduction defects (right), acquired conduction defects associated with arteriosclerotic heart disease, and one acquired (left) after closure of a large patent ductus arteriosus. The diagnoses of complete right and left bundle-branch block were made on the basis of the criteria of Wilson.3 The criteria for the diagnosis of right ventricular failure were clinical and consisted of the presence of systemic venous hypertension, pitting peripheral edema, and congestive hepatomegaly.

The phonocardiograms were logarithmic4 recordings at paper speeds of 50-75 mm, second, taken with either a standard twin-beam Sanborn, or a specially built Cambridge with the same frequency response characteristics. Indirect carotid pulses were obtained with a light tambour applied to the neck. The interval between aortic valve closure and the insertion of the dicrotic notch of the carotid pulse varied 0.02 to 0.04 second, depending on where in the neck the carotid pick-up was located. The intracardiac pulses were, in certain instances, taken on a cathode ray photographic recorder. It is to be emphasized that all recorded events were appreciated by auscultation, and hence have direct clinical application.

Results

Normal Controls. The majority were young adults, although the ages ranged from 5 to 50 years. During relaxed respiration the second heart sound varied from expiratory synchrony to inspiratory separation averaging 0.04 to 0.05 second. The younger subjects differed somewhat in that the second sound more often tended to remain slightly split during expiration (average 0.02 seconds) and tended to split more widely during inspiration. This expiratory asynchrony would often disappear if the subject expired more completely. When the respiratory excursions were increased in magnitude, splitting tended to become more pronounced in all ages, but especially in the younger subjects in whom the inspiratory asynchrony occasionally reached 0.08 to 0.09 seconds. When the records were taken during held expiration, held inspiration, or in the respiratory mid-position, the two components of the second sound tended to drift apart to varying degrees. It should be emphasized, therefore, that analysis of the second heart sound must be made during active respiration.
**Atrial Septal Defect.** Thirteen patients were studied before and after surgical closure of the defect. In each instance the surgeon was satisfied with the technical result of the procedure. Ten of the patients had the RSR'-V1 pattern in the electrocardiogram and one patient had the pattern of complete right bundle-branch block. The QRS configuration of all 13 tracings remained unchanged at the time the postoperative study was done. Eight of these patients were recatheterized postoperatively. By this evidence the defect had been closed in all.

In the preoperative phonocardiograms the splitting of the second heart sound remained fixed throughout respiration. In one patient not operated upon because the shunt was small (less than 1 L. per minute) and the right ventricle normal in size, the split widened 0.02 to 0.025 seconds during inspiration, but never achieved expiratory synchrony. Two patients had arrhythmias causing variations in cycle length, i.e., a wandering pacemaker in the sinoatrial node and episodes of sinus arrest. The split second sound widened after the longer cycle lengths and narrowed after the shorter cycle lengths.

In 11 of the 13 patients the second sound on the postoperative phonocardiogram split normally during inspiration and became single during expiration. In 1 of the 13, however, the second sound did not normalize. Preoperatively, the split had been 0.08 second. Postoperatively, the split was 0.06 second (comparing complexes of the same cycle length) and during inspiration the pulmonic component moved less than 0.02 second. There was no delay in the onset of the right ventricular pressure pulse in the postoperative catheterization study. There was no clinical evidence of right ventricular failure. Postoperative catheterization proved the defect to be closed. The explanation for the findings in this case is not apparent.

In the 1 patient with complete right bundle-branch block, the postoperative split of the second sound increased from an expiratory separation of 0.03 second to an inspiratory separation of 0.06 second. This can be considered a normal inspiratory delay in pulmonic valve closure. The relationship of the two components of the second sound is typical of complete right bundle-branch block. This is of further interest since the onset of the right ventricular pressure pulse was not delayed, occurring 0.06 second after the onset of the QRS of the electrocardiogram.

**Complete Right Bundle-Branch Block.** Eleven patients were studied. In the 8 with compensated right ventricles the split second sound widened on inspiration and narrowed on expiration but never became single. In the 3 patients with right ventricular failure, the split remained fixed throughout the respiratory cycle.

**Complete Left Bundle-Branch Block.** Ten patients were studied. In the 6 with compensated right ventricles, the second heart sound split during expiration and became
Single during inspiration. In the 4 patients with right ventricular failure, the second sound remained constantly split throughout the respiratory cycle.

_Pure Mitrail Insufficiency._ Ten patients were studied. In the 6 with compensated right ventricles, the split second sound widened with inspiration and narrowed with expiration, but never became single. In the 4 patients with right ventricular failure, the split remained fixed. The width of the split varied from 0.04 to 0.10 second. None of these patients had the electrocardiographic pattern of right bundle-branch block.

**Discussion**

As early as 1866, Potain\(^1\) was aware that the second heart sound (S\(_2\)) split during the inspiratory phase of respiration. In 1950, Barber et al.\(^6\) emphasized this finding in children. Leatham and Towers\(^7\) subsequently confirmed its occurrence in the majority of healthy adults. The first component of the split is recorded at all valve areas and is synchronous with the dicrotic notch of the carotid pulse (fig. 1), a feature identifying it as the sound of aortic valve closure (A\(_2\)). The second component is normally confined to the pulmonary area or the immediately subjacent left sternal edge and is synchronous with the dicrotic notch of the pulmonary arterial pulse (fig. 1), a feature identifying it as the sound of pulmonary valve closure (P\(_2\)). During inspiration the **effective pulmonary venous filling pressure remains unchanged, since pulmonary veins, left atrium, and left ventricle share equally the inspiratory fall in intrathoracic pressure.** However, the **effective systemic venous filling pressure increases with inspiration since the inspiratory pressure in the right heart falls below that of the extrathoracic great veins.** This results in selective inspiratory augmentation of right-sided filling. The right ventricle takes longer to eject this increased volume and its prolonged ejection time is reflected in delayed pulmonary valve closure, giving rise to the normal inspiratory splitting of the second heart sound (fig. 2). It is to be emphasized that only P\(_2\) moves with respiration, the timing of A\(_2\) remaining unchanged.*

The phenomenon of fixed splitting of the second heart sound in atrial septal defect has been well documented.\(^8\) Our observations confirm the findings that even with deep inspiration and expiration the interval between aortic and pulmonic components of the second heart sound in atrial septal defect remains constant or moves very slightly (less than 0.02 second), the sounds never becoming single (fig. 3A). In 1 patient with a proven ostium

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*Ed.: Compare contradictory findings of Boyer and Chisholm in the succeeding paper.*
primum defect and normal mitral and tricuspid valves, and in 1 patient with ostium primum type of atrial septal defect with mitral incompetence, fixed splitting of the second sound occurred. Pulmonary hypertensive atrial defects are exceptions to this rule and are not included in this study.

A basic hemodynamic expression of atrial septal defect is constant right-sided diastolic hypervolemia. The right ventricle apparently

Fig. 3. A. Proven atrial septal defect, preoperative. Note that the interval between the aortic (A2) and pulmonic (P2) components of the second sound remains fixed throughout the respiratory cycle. E, pulmonic ejection sound. B. Same patient after surgery. The atrial septal defect was proven to be closed by postoperative cardiac catheterization. Note that now the aortic (A2) and pulmonic (P2) components of the second sound are synchronous during expiration and split normally during inspiration.
takes longer to eject this increased volume, hence, its stroke time is prolonged and the pulmonic valve closes later than the aortic (fig. 3A). Because the overfilled state is present throughout the respiratory cycle, the right ventricular volume during expiration may not diminish. Conversely, as a result of its already excessive diastolic inflow, the right ventricle apparently does not accept the additional inspiratory augmentation of filling, and its stroke volume thus does not increase during inspiration. The result is that aortic and pulmonic valve closures remain constantly separated, their interval being affected little, if at all, by respiration (fig. 3A).

That the asynchrony of semilunar valve closure is not a reflection of the electrocardiographic pattern of "incomplete right bundle-branch block"—perhaps better termed the RSR'-V₁ pattern⁹—was demonstrated by studying 12 patients with the ostium secundum type of atrial septal defect, and 1 with the ostium primum type before and after surgical repair of these lesions. As soon as the second sound could be analyzed (often the day after surgery), it was found to split normally during inspiration, and close during expiration (fig. 3B). In no instance was there any change in the QRS pattern when the postoperative study was made. One patient had complete right bundle-branch block and a fixed split before surgery. Postoperatively, the split widened with inspiration and narrowed with expiration, but never became single, reflecting the usual finding in complete right bundle-branch block (see below). A residual left-to-right shunt through anomalous pulmonary veins may result in a failure of the second sound to normalize postoperatively, even though the defect in the septum is closed.

The following observations suggest that, although the split second sound in atrial septal defect remains fixed relative to respiration, it may vary with changes in cycle length. Figure 4 illustrates an increase in the A₂-P₂ interval from 0.04 to 0.08 second coincident with an increase in cycle length caused by periods of sinus arrest. It can be seen that the interval between S₁ and A₂ remains constant, hence the increase in A₂-P₂ interval must have been due to further delay in pul-

Fig. 4. Proven atrial septal defect, preoperative, with variations in cycle length due to episodes of sinus arrest. Note that after longer cycle lengths the interval between aortic (A₂) and pulmonic (P₂) components of the second sound increases. After shorter cycle lengths this interval decreases.
monic valve closure, reflecting a prolongation of right ventricular stroke time. Thus, after a long cycle length the right ventricular stroke time selectively lengthens. The same phenomenon was observed in another patient with an atrial septal defect, in which the cycle length varied because of a wandering pacemaker in the sinoatrial node. These events might be explained in the following fashion. When there is a defect in the atrial septum all 4 cardiac chambers are in common communication during diastole, but the rate of flow into the right ventricle far exceeds the rate of flow into the left ventricle. An increase in the diastolic filling period should therefore contribute disproportionately to right rather than to left ventricular filling. With longer cycle lengths it is postulated that the right ventricle may be the principle, if not the exclusive recipient of the added increment of diastolic filling. This should reflect itself in selective prolongation of right ventricular stroke time, delay in pulmonic valve closure and wider separation of the A2-P2 interval during the longer cycle lengths.

Fixed splitting of the second heart sound

Fig. 5. A. Complete right bundle-branch block in a 60-year-old female with arteriosclerotic heart disease. Note that during inspiration the interval between aortic \((A_2)\) and pulmonic \((P_2)\) components of the second sound increases and during expiration the interval decreases, but the two sounds never become single. \(X\), systolic cardiorespiratory click. B. Complete right bundle-branch block in a 58-year-old male with hemochromatosis and right ventricular failure. Note that the interval between aortic \((A_2)\) and pulmonic \((P_2)\) components of the second sound remains constant throughout the respiratory cycle. The first sound \((S_1)\) is soft because of a prolonged P-R interval.
is, therefore, a valuable diagnostic sign of atrial septal defect, and possibly of the atrio-ventricularis communis type of anomaly. This phenomenon is probably a manifestation of the right-sided diastolic hypervolemia, which prevents the right ventricle from undergoing its normal respiratory changes in stroke volume. The rather prompt return to the normal state after the defect is closed supports this concept.

**Other Observations**

Although heretofore fixed splitting of the second heart sound has been described only in atrial septal defect, the following considerations led us to postulate its occurrence in other conditions. If the right ventricle in a state of failure were operating on the plateaus of its Starling curve, it should not be able to convert the inspiratory augmentation of filling into increased stroke volume.\(^{10}\)
Hence, in this state of failure, the right ventricle might be expected to have a constant ejection time throughout the respiratory cycle, resulting in a disappearance of the normal inspiratory delay in pulmonic valve closure. This thesis was tested in 3 categories of patients with sufficient splitting of the second heart sound to allow detailed analysis.

The first group consisted of patients with complete right bundle-branch block. A commonly observed mechanical sequel of this delay in right ventricular depolarization is a delay in pulmonic valve closure. This reflects itself in abnormally wide splitting of the second heart sound,¹ which fails to become single during expiration. During inspiration the normal augmentation of right-sided filling still occurs with the associated delay in pulmonic valve closure. One finds accordingly that although the aortic and pulmonic components of the second sound widen their interval during inspiration and narrow it during expiration, they fail to become synchronous during the expiratory phase of respiration (fig. 5A). However, when right bundle-branch block occurs in the presence of right ventricular failure, the splitting of the second sound remains fixed (fig. 5B). This is believed due to the inability of the failing right ventricle to convert its inspiratory augmentation of filling into increased stroke volume. As a consequence of this inability, the inspiratory delay in pulmonary valve closure should
not occur. The $A_2$-$P_2$ interval would, therefore, remain unchanged.

The second group consisted of patients with left bundle-branch block. It appears that a mechanical sequel of this delay in left ventricular depolarization is an associated delay in aortic valve closure. The delay is usually sufficient to cause the aortic valve to close after the pulmonic, thus reversing the normal closing sequence of the semilunar valves. This is demonstrated by timing $A_2$ with the dicrotic notch of the carotid pulse (fig. 6A). The result of this "paradoxical splitting" of the second heart sound is expiratory separation of its two components. During inspiration the timing of pulmonary valve closure moves toward aortic closure and the second sound thus becomes single (fig. 6A). However, in the presence of right ventricular failure, $S_2$ does not become single during inspiration because the inspiratory delay in pulmonic valve closure does not occur (fig. 6B). This results in fixed splitting of the second heart sound in left bundle-branch block.

In one case this method of analysis was of particular interest. The patient, a 57-year-
old male with hypertensive-arteriosclerotic heart disease had an electrocardiogram which resembled left bundle-branch block in the limb leads, and right bundle-branch block in the precordial leads. The vectorcardiogram using the Schmidt-Simonson system\textsuperscript{12} suggested biventricular conduction defects. This apparent electrocardiographic paradox has been variously interpreted. Some investigators\textsuperscript{13} consider these tracings examples of right bundle-branch block, emphasizing the unreliability of the limb leads in distinguishing the side of the block. Others\textsuperscript{14} feel that in some cases this pattern should be regarded as left bundle-branch block with extensive posterolateral myocardial infarction. Auscultatory examination of the patient disclosed a widely split second heart sound, but since marked right ventricular failure was present the split remained fixed, and therefore, could not be used to distinguish right from left bundle-branch block. The phonocardiogram with synchronous carotid arterial pulse (fig. 7) revealed a normal sequence of aortic-pulmonic valve closures, suggesting in this instance the mechanical asynchrony of a right bundle-branch block. These observations further illustrate that in the presence of right ventricular failure, one cannot distinguish right from left bundle-branch block by auscultation.

The third group consisted of patients with pure mitral insufficiency associated with wide splitting of the second sound.\textsuperscript{4} Unusually wide splitting of the second sound has been observed in this lesion\textsuperscript{15} and attributed to premature aortic valve closure. It is suggested that if the left ventricle can expel its contents not only into the systemic circulation, but also into the left atrium, then its ejection time might be shortened, resulting in early aortic valve closure. If this is sufficiently pronounced, aortic and pulmonic valve closures may remain constantly separated (fig. 8A). It is to be noted, however, that—as in normals—inspiratory prolongation of right ventricular ejection time still occurs, resulting in inspiratory delay of pulmonic valve closure and increased splitting of the second heart sound (fig. 8A). It was found, however, that when the right ventricle decompensates in the presence of mitral insufficiency with wide splitting of the second heart sound, there is no inspiratory delay in pulmonic valve closure, hence, the split becomes fixed (fig. 8B).

These data do not permit conclusions regarding the state of decompensation at which the inspiratory delay in pulmonic valve closure would fail to occur. The severity of right ventricular failure will vary from time to time and from case to case. Even if the failing ventricle is operating on a depressed function curve, it still may be able to increase its stroke work if it has not yet reached the plateau of the curve.\textsuperscript{10} It might be anticipated, therefore, that not all patients in whom right ventricular failure coexists with complete bundle-branch block or mitral insufficiency will illustrate the phenomenon of fixed splitting of the second heart sound.

**Summary**

Forty-four patients with wide splitting of the second heart sound and 25 normal controls were studied with fast speed logarithmic phonocardiograms.

In the control group, inspiratory augmentation of right heart filling increased the right ventricular stroke volume, prolonged the right ventricular stroke time and delayed pulmonic valve closure, thus altering the second sound from expiratory synchrony to inspiratory separation of its 2 components.

In the group with atrial septal defects, the second sound normalized postoperatively, reflecting the ability of the right ventricle to undergo its normal inspiratory increase and expiratory decrease in stroke volume when the defect was closed. Before closure, the right ventricular stroke time neither shortened with expiration nor lengthened with inspiration because of the constant diastolic right ventricular hypervolemia. Hence, preoperatively the interval between aortic and pulmonic valve closures remained characteristically wide and fixed. That the preoperative delay in pulmonic valve closure could
not have been a reflection of "incomplete right bundle-branch block" was illustrated by the postoperative normalization of the second sound without change in the QRS pattern of the electrocardiogram. Although the split of the second sound was not altered by respiration, it was altered by change in cycle length, widening after longer cycles and narrowing after shorter cycles.

Wide splitting of the second heart sound was found in mitral insufficiency because of early aortic valve closure, in complete right bundle-branch block because of delayed pulmonic valve closure, and in complete left bundle-branch block because of a reversed sequence of aortic-pulmonic valve closure. When right ventricular failure coexisted with these lesions, the decompensated chamber did not convert its inspiratory augmentation of filling into increased stroke volume. The inspiratory increase in right ventricular stroke time and the inspiratory delay in pulmonic valve closure therefore could not occur so the split of the second sound remained fixed throughout the respiratory cycle.

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SUMMARIO IN INTERLINGUA

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REFERENCES

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