The Contribution of Tricuspid Valve Closure to the First Heart Sound
An Intracardiac Micromanometer Study

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SUMMARY The sound-pressure correlates of the second high frequency component of a split first heart sound (S₂) were investigated in 27 patients. An external phonocardiogram was recorded with high fidelity sound and pressure from the left and right atria in 21 patients, from the pulmonary artery in 14 of these, and from the central aorta in 11. In the remaining six patients, high fidelity recordings from the central aorta and right-sided chambers were obtained with an external phonocardiogram. The external component of S₂ that coincided with a left atrial C wave and "internal sound" was defined as M₁. In those cases where the left atrial pressure was not recorded, this component could be identified by a low frequency transient in the central aortic pressure trace. The other external high frequency component of S₂ that was synchronous with a separate right atrial C wave and "internal sound" was defined as T₁; with two exceptions, M₁ preceded T₁. The two exceptions which caused reversal of this order, so that T₁ preceded M₁, were due to chronic left bundle branch block and mitral stenosis. In both cases, T₁ was shown to be distinctly separated from the upstroke of pressure rise in the central aorta. This finding was also demonstrated in three cases of right bundle branch block and one case with aortic valvular disease. The usual asynchrony of ventricular contraction was altered by induction of ventricular premature systoles; the separation of externally identifiable M₁ and T₁ components and their internal markers was predictably altered by this maneuver. The occurrence of T₁ was variable in relation to the upstroke of the pulmonary artery pressure, which suggests that it is not related to pulmonic ejection. It is concluded that micromanometrically recorded right and left atrial C waves can serve as markers for externally recordable M₁ and T₁ components of the first heart sound. In addition, T₁ is frequently an externally recordable and audible event.

SINCE THE CLASSIC PAPER on the nature of heart sounds by Wiggers and Dean,¹ considerable interest has centered around the genesis of the two high frequency components of the first heart sound (S₁). While there is partial agreement on the physiologic basis for the first component,²-¹⁰ the hemodynamic basis of the second component remains controversial. At present, two theories have been proposed: the valvular theory which attributes the two components to mitral and tricuspid valve closing sounds,¹⁰-¹⁵ and the exclusive left-sided theory which holds that tricuspid valve closing sound is silent and attributes the two components to deceleration of a column of blood occurring as a result of pressure rise in the left ventricle followed by ejection of blood into the aorta giving rise to an ejection sound (AES).¹⁶-²⁶

The valvular theory has found support from phonocardiographic studies by Leatham¹² and others,¹³,¹⁴ and more recently, by echocardiographic studies.¹⁰,²⁷,²⁸ Experimental support for this theory is based on the canine experiments of Smith et al.² who noted a less distinct first heart sound following excision of the mitral valve and a barely audible first heart sound when this procedure was followed by tricuspid valve excision.

Others hold that the two components of S₁ are entirely due to left-sided events. The data from phonocardiographic,¹⁷,¹⁸ hemodynamic,¹⁹,²⁰ and left-sided micromanometer studies²¹-²⁷ have been interpreted to mean both components have a left-sided basis, thus excluding any contribution of right-sided events. Experimental support for excluding a tricuspid valve origin has been obtained from canine experiments where the right ventricle was completely bypassed without changing the first heart sound complex recorded from the left ventricle.²⁹ In a second experiment where left ventricular contractility was markedly impaired, the intervention was associated with loss of the first heart sound recorded from the right ventricle.³⁰

The normally close temporal association of right ventricular isometric contraction events and the onset of left ventricular ejection provides a distinct problem in utilizing hemodynamic correlates to determine the origin of the two high frequency components of S₁. The present study undertook to resolve this problem by using sound and pressure recordings from high fidelity micromanometer catheters in situations where alterations in this close temporal association might be expected.

Materials and Methods

Twenty-seven patients, ranging in age from 17 to 65 years, were chosen for this study. In each case, specific permission was granted to perform high fidelity intracardiac sound and pressure measurements during the course of diagnostic catheterization. The patients and their diagnoses are listed in table 1. In 21 cases simultaneous micromanometric sound and pressure were recorded from both atria. Six cases did not undergo transseptal catheterization and had only central aortic and right-sided sound and pressure determinations. Of the 27 patients, micromanometric sound and pressure were recorded from the pulmonary artery in 19 and central...
aorta in 17. One patient had chronic left bundle branch block; four had chronic right bundle branch block. In two cases, right and left ventricular premature beats were produced by catheter stimulation of the appropriate ventricle to alter the sequence of right and left-sided mechanical events. In one patient, isorhythmic A-V dissociation was produced by right ventricular pacing.

In one patient, events.

These pressures were measured with micromanometric catheters.* These transducers allow recording of high fidelity intracardiac pressures free of contour distortion and without time lag. Access to the left atrium was obtained by passing a #5 French micromanometer catheter through a #9 French Ross transeptal catheter or passing across an atrial septal defect. A #8 French micromanometer catheter was used to record central aortic pressure just above the valve. Right-sided pressures were obtained using either a single or triple-tipped pressure transducer catheter, the latter one allowing for simultaneous recording from right atrium, right ventricle and pulmonary artery.

Intracardiac sound was obtained from the micromanometer transducer. The audio-circuitry had a flat response from 70 to 2,000 Hz with a roll-off of 12 decibels per octave below 70 Hz. Internal high frequency vibrations referred to as “internal sound” were recorded from the left atrium; it occurred simultaneous with a left atrial C wave. This left sided “internal sound” was also well recorded in the left ventricle and central aorta. The micromanometer catheter had to be in close proximity to the tricuspid valve in order to record the right atrial C wave and simultaneous high frequency vibrations subsequently referred to as “internal sound.” In each case special attention was exercised to avoid artifacts due to catheter impact against walls of the heart or valve cusps. The right-sided internal sound was usually not seen in the right atrium at any appreciable distance from the tricuspid valve; it was seen in the right ventricle as a low amplitude vibration only in cases of atrial septal defect.

At all times, intracardiac sound was recorded together with external sound; the latter was recorded using an Electronics for Medicine microphone and band pass filters set at 120 and 500 Hz on a DR 12 recorder. The microphone was placed on the chest wall where the two components of the first heart sound were loudest, generally along the left sternal border a few centimeters to the left of the sternal. A simultaneous electrocardiogram was also recorded for timing purposes and in several of the cases respirations were recorded by means of a nasal thermistor. Unless otherwise indicated, recordings were made at a paper speed of 100 mm/sec with time line markers of 0.020 sec.

Results

All cases in this series had two high frequency components of the first heart sound which could be identified on the external phonocardiogram. The external component coincident with a left atrial C wave and internal sound was labeled M1. The external component synchronous with a right atrial C wave and internal sound was labeled T1. In the six cases where the left atrium was not entered, T1 was identified and the remaining component then was labeled M1. The iden-

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<tr>
<th>Table 1. Summary of Data on 27 Patients</th>
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<tr>
<td>Diagnosis</td>
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<tr>
<td>Chest pain with normal coronaries</td>
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<td>Atrial septal defects (seundum type)</td>
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<td>Coronary artery disease</td>
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<td>Asymmetric septal hypertrophy</td>
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<td>Idiopathic pulmonary hypertension</td>
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Abbreviations: RBBB = right bundle branch block; LBBB = left bundle branch block.

FIGURE 1. This figure shows simultaneously recorded right atrial (RA) and left atrial (LA) "sound" and pressure and external phonocardiogram in a 59-year-old female with asymmetric septal hypertrophy without obstruction. The first heart sound on the external phonocardiogram consists of two narrowly separated high frequency components. The first component is coincident with a high frequency vibration (internal sound) recorded from the left atrium and the downstroke of the left atrial C wave. It is labeled M1. The second component is coincident with a high frequency vibration (internal sound) recorded from the right atrium and the downstroke of the right atrial C wave and it is labeled T1. Time lines in this and subsequent illustrations correspond to 0.020 sec.

*Millar Mikro-tip pressure transducer, Houston, Texas.
The external sound vibrations following the wave and flow recorded left atrial sound and internal sound recorded from the right atrium. The larger sound and C wave recorded from the right atrium. The vibrations following T1 on the external phonocardiogram are due to a systolic flow murmur.

These two pressure events were significantly separated in a patient with chronic left bundle branch block and a Bjork-Shiley aortic valve prosthesis. Figure 3 shows T1 preceding both M1 and onset of pressure rise in the central aorta. A similar finding was produced when a left bundle branch block pattern was induced by stimulating the right ventricular cavity with a catheter, producing a run of four successive premature beats as shown in figure 4. The top panel represents the control state and shows wide splitting of S1 on the external phonocardiogram; the second component is coincident with a right atrial C wave and internal sound and also with the upstroke of the central aortic pressure. The lower panel depicts premature beats produced by stimulating the right ventricle. The usual order has been reversed: T1 coincides with the right atrial C wave and internal sound from this chamber and precedes M1, which coincides with a low frequency transient seen on the central aortic pressure curve. Both sounds significantly precede the upstroke of the abbreviated aortic pressure curve rendering it unlikely that these sounds originated from ejection of blood into the aortic root.

The four cases with chronic right bundle branch block demonstrated wide splitting of the first heart sound. The T1
component occurred after the pressure rise in the central aorta in two subjects, was coincident with the aortic upstroke in one and preceded the aortic upstroke in one (fig. 5). Thus, it was shown for our four cases with right bundle branch block that the external component labeled T₁ bore no constant relationship to the upstroke of pressure rise in the central aorta.

In one patient with severe mitral stenosis and pulmonary hypertension (mean 50 mm Hg) the usual sequence was reversed, that is, T₁ preceded M₁ (fig. 6). This order was normalized when a left-sided premature contraction was produced by catheter stimulation of the left ventricle.

The two components of S₁ were studied in cases of aortic valvular disease where there was no conduction disturbance and a valvular or prosthetic click could be easily recorded from the aortic root. The findings in a 26-year-old male with a Starr-Edwards aortic prosthesis are demonstrated in figure 7. Two high frequency components of S₁ (M₁ and T₁) are synchronous with C waves and internal sounds recorded from the left and right atria, respectively. Both of these sounds precede the upstroke of the central aortic pressure curve. The aortic upstroke is synchronous with a small external deflection that may represent an aortic root sound which occurs an appreciable interval before the opening click of the aortic prosthesis. This patient is representative of the entire spectrum of sound that may be recorded around the time of S₁. Five additional patients with aortic valvular disease demonstrated similar findings in that the second component coincided with a right atrial C wave and internal sound.

The external component labeled T₁ did not bear a constant relationship to the onset of pressure rise in the pulmonary artery. The T₁ component occurred prior to this onset in the four cases with chronic right bundle branch block and in four additional cases with pulmonary hypertension. In the remaining cases with normal pulmonary artery pressures, the external component labeled T₁ occurred synchronous with or after the onset of pressure rise in the pulmonary artery. In those cases where T₁ occurred after the onset of pressure rise in the pulmonary artery, no tricuspid regurgitation could be demonstrated. This was true even though the micromanometer catheters were in proximity to the tricuspid valve and the murmur of regurgitation was carefully sought.

The results of A-V dissociation produced by isorhythmic pacing of the right ventricle are shown in figure 8. The first paced beat initiates ventricular contraction before atrial contraction and the first heart sound is almost nondiscernible externally. The second paced beat is preceded by an “a” wave and a loud single S₁ lines up with a right atrial C wave.

Figure 4. The data recorded in a 61-year-old male with underlying atherosclerotic heart disease. The top panel shows high fidelity right atrial and central aortic sound and pressure recorded simultaneously with an external phonocardiogram. There is wide splitting of S₁ with a second high frequency component coincident with a right atrial C wave and internal sound as well as the upstroke of the central aortic pressure trace. In the lower panel, premature ventricular contractions have been produced by stimulating the right ventricle. This caused reversal of the usual sequence in this patient with T₁ preceding M₁. In each case, the external component labeled T₁ coincides with a right atrial C wave and internal sound from this chamber. The external component labeled M₁ is coincident with an internal sound recorded in the central aorta and a low frequency transient seen on the aortic pressure curve. Both components occur a considerable interval prior to pressure rise in the central aorta demonstrating that they are not related to aortic ejection.

Figure 5. Simultaneous right atrial and central aortic sound and pressure with an external phonocardiogram in a 58-year-old man with chronic right bundle branch block and severe atherosclerotic heart disease. The second high frequency component is synchronous with a right atrial C wave and internal sound and occurs 0.014 sec before pressure rise in the central aorta. There is a soft third component of S₁ which coincides with aortic upstroke and a transient recorded in the aortic root. This may represent an aortic root or ejection sound.
and internal sound. The third normally conducted beat then shows an externally split first sound; both components have significantly increased in intensity as compared to the first ventricular paced beat. The second component is simultaneous with internal sound and the aortic upstroke by 0.010 sec. Thus, the intensity of both high frequency components of \( S_1 \) and \( S_2 \) behaves similarly during the induction of isorhythmic A-V dissociation.

**Discussion**

Different hemodynamic explanations have been given for the genesis of the two high frequency components of the first heart sound. The first component, with a few exceptions to be discussed subsequently, is a left-sided event. This sound has been attributed either to checking action on a column of blood by the mitral valve\(^9\) or to intraventricular pressure rise.\(^{19, 20, 23, 24}\) Our data show that this sound always occurs synchronously with a sharp left atrial C wave and/or a discernible transient on the central aortic pressure curve. In keeping with the established nomenclature and consistent with our data, this component has been labeled \( M_1 \), implying a mitral valve basis for its generation.

The main thrust of this report is to clarify the controversial issue concerning the hemodynamic basis of the second high frequency component of the first heart sound. Many previous reports have noted that this sound coincides with the onset of pressure rise in the central aortic\(^8\) or with peak acceleration of blood into this great vessel.\(^9, 21\) Thus, the origin of this second component has been related to the sudden acceleration of blood into the aortic root and it has been referred to as an aortic ejection sound (AES).\(^{28}\) Recently, Luisada has stated that the second component is not related in any way to closure of the tricuspid valve.\(^{24, 25}\) Others ascribe the origin of the second component to a right-sided atrioventricular valvular mechanism. They attribute the second high frequency component to checking action on a column of blood by the tricuspid valve. Echocardiographic studies have demonstrated synchronous occurrence between echocardiographic tricuspid valve closure and an externally recordable sound in Ebstein's anomaly\(^{28}\) and in patients with atrial septal defects.\(^{19, 21}\) Recently, Lakier et al.,\(^{32}\) using pressures recorded through fluid-filled catheters, reported separate left and right atrial C waves lining up with the two high frequency components of \( S_1 \) that he called \( M_1 \) and \( T_1 \), respectively. Thus, evidence has been obtained to support two widely divergent theories regarding the genesis of the second component.

The basis for the controversy regarding the second com-
ponent lies in the fact that it occurs in close temporal relationship to both inscription of the right atrial C wave and onset of pressure rise in the central aorta. In order to determine whether the second high frequency component of S1 was related to aortic ejection or tricuspid closure, it was necessary to investigate situations where greater temporal separation of these two events might be expected. Manipulations of this component by ventricular pacing or production of ventricular extrasystoles tended to show maintenance of synchrony with the right atrial C wave while tending to exclude a relationship to aortic ejection. Hence, it appeared the right atrial C wave was a proper hemodynamic marker for T1.

It was also expected this synchrony would be maintained in stable situations where an altered relationship between aortic ejection and the right atrial C wave might occur such as bundle branch blocks. Previous studies on S1 in the presence of left bundle branch block have been inconclusive with regard to the presence or absence of the T1 component. Oravetz et al., in an external phonocardiographic study of the bundle branch blocks, showed left bundle branch block caused consistent delay in the onset of the first heart sound. The absence of T1, clearly preceding a three component S1 complex in any case was taken by these authors as evidence that tricuspid valve closure does not contribute to the generation of the first heart sound. This assumption, however, is not warranted because delay in the Q-M1 interval in the left bundle branch block is inconsistent and usually not marked and would not be expected to cause reversal of the usual sequence. That this does occur in cases of significant left-sided electromechanical delay is illustrated in figures 3 and 4.

Several studies have cited the wide splitting of the first heart sound in right bundle branch block as evidence that the second component results from delayed tricuspid valve closure secondary to delayed right ventricular activation. Oravetz et al. did not find abnormally wide splitting in this condition and phonocardiographically demonstrated that the second high frequency component was synchronous with the carotid artery upstroke. The absence of wide splitting of S1 in the presence of electrocardiographic evidence of right bundle branch block may be explained by the finding that delayed right ventricular activation cannot be predicted reliably by this electrocardiographic pattern. The four cases of chronic right bundle branch block in this series all had wide splitting of the first heart sound and the second, or T1, component coincided with the right atrial C wave. In these four subjects, T1 demonstrated a variable relationship to the central aortic pressure upstroke. A delay in right ventricular mechanical activation was also produced by catheter stimulation of the left ventricle leading to a delayed T1 component as shown in figure 6.

Previous work reported on the relationship between P-R interval and the intensity of the first heart sound lends further support to the concept that in some cases the two components of the first heart sound result from A-V valve checking action. In patients with high grade atrioventricular block, Shah et al. noted the first heart sound was markedly diminished in intensity when the P-R interval was greater than 0.020 sec. This correlated with preclosure of the mitral valve on echocardiogram. These findings were interpreted to mean the intensity of the M1 component was related to mitral valve leaflet position at the time of ventricular systole. If the leaflets are widely open, the resulting sound will be loud and, conversely, if there is preclosure of the leaflets, the resulting sound will be soft or absent. If the R-R interval remained constant in these cases, it is easy to understand why the intensity of the M1 component of the first heart sound varied inversely with the P-R interval but is not clear why the intensity of an aortic ejection sound would always change in a similar direction. However, if the second component is related to tricuspid valve closure, its behavior would be expected to parallel that of the mitral valve. Support for this valvular concept is shown in figure 8 where right ventricular pacing was performed. When isorhythmic A-V dissociation was produced by ventricular pacing, the intensity of the second high frequency component of S1 was similarly related to P-R interval.

The canine experiments interpreted as showing the absence of T1 do not conclusively prove the absence of this sound in humans. The fact that T1 was not recordable with micromanometers from either ventricle at rest or after alteration of right or left ventricular contractility is not inconsistent with the data from this study. Except for cases of atrial septal defects where a loud T1 was faintly recorded.

Figure 8. The results of A-V dissociation produced by isorhythmic pacing from the right ventricle. High fidelity sound and pressures are recorded from the right atrium and central aorta. Note that a high right atrial pressure curve does not show distinct C waves. There is a very soft external S1 with the initial paced beat where ventricular depolarization occurs before atrial contraction. As 'a' waves reappear in the second and third beats, a split S1 reappears and its second component is synchronous with a right atrial C wave and internal sound. This shows that the intensity of the two high frequency components of S1 in this case behave in a parallel manner suggesting a similar hemodynamic basis. While the aortic ejection time increases with each successive beat there is no significant increase in an aortic ejection sound.
in the right ventricle, this second component could not be recorded in the right ventricle, even though it was carefully sought. The reason that T1 is not as widely transmitted internally as M1, is not explained by this study, but perhaps is related to the difference in energy imparted to the respective atrioventricular valves by ventricular systole.

In this series of patients, there was a marked variability between the onset of T1 and the onset of ejection into the pulmonary artery. This component occurred prior to pulmonary artery upstroke in cases of pulmonary hypertension (4 cases) and chronic right bundle branch block (4 cases) and synchronous or well after this upstroke in the remaining cases. Moreover, no internal sound was recorded in the pulmonary artery at the time of T1. The foregoing findings suggest that this component does not represent a pulmonary artery ejection sound.

This present study has shown two high frequency components of the first heart sound are frequently synchronous with separate high frequency vibrations (sound) and low frequency pressure transients recorded in the respective atrial chambers. There were minor differences in duration and frequency content between internal and external phonocardiograms. These differences may be related to the transmission and resonating characteristics of the chest wall. M1 and T1 were shown to uniformly occur on the downstroke of respective C waves anywhere from the peak to the nadir. Although the same acceleratory and/or deceleratory forces might give rise to both low frequency pressure changes as C waves and high frequency vibrations (sound), the precise relationship to each other may vary depending upon the resonating characteristics of the cardiohemic system. However, maintenance of synchrony between T1 and the right atrial C wave irrespective of pathological states or the order of ventricular contraction suggests the tricuspid valve checking action, at least in some patients, contributes to the audible and recordable events of the first heart sound.

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References

The contribution of tricuspid valve closure to the first heart sound. An intracardiac micromanometer study.

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