Tricuspid component of first heart sound

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Right and left atrial pressures with a simultaneous external phonocardiogram and standard lead II electrocardiogram were recorded during routine cardiac catheterization in 15 patients. With one exception, the onset of the second (T₁) major component of the first sound coincided with the peak of the right atrial c wave just as the major left-sided component (M₁), previously studied in this laboratory, coincides with the peak of the left atrial c wave. Right and left atrial c waves were synchronous in the exception and in that instance separate major components were not identified. These observations provide further evidence that T₁ arises at the tricuspid valve. Analogous to the mode of production of M₁, it is believed that after tricuspid valve closure the apposed leaflets are billowed into the right atrial cavity resulting in right atrial c waves, at the peak of which tension on the chordae tendineae and leaflets themselves produces T₁. Some factors affecting the timing and intensity of M₁ and T₁ are mentioned.

The mechanism of production and factors influencing the timing and intensity of the major left-sided component (M₁) of the first heart sound have been studied and discussed in recent publications from this laboratory (Lakier et al., 1972a, b, c). It was concluded that the mitral leaflets appose and the valve ‘closes’ at the point of crossover of the left ventricular and left atrial pressures but that M₁, which is almost always the earlier of the two major first sound components, occurs after the valve has closed. M₁ is caused by maximal tension on the coapted leaflets and the chordae tendineae at the peak of the ascent of the leaflets into the left atrial cavity. The billowing of the apposed leaflets produces the left atrial c wave, at the peak of which vibrations of M₁ can be seen to begin on a simultaneously recorded phonocardiogram. There are several important factors which affect the timing and intensity of M₁ as well as the amplitude of the left atrial c wave (Lakier et al., 1972a). These include mitral leaflet size and the mobility of the valve mechanism. M₁ and the peak of the left atrial c will be delayed, with M₁ louder and the left atrial c of larger amplitude, when the leaflets are relatively more voluminous or the valve mechanism more mobile (Lakier et al., 1972b).

Although it is widely believed that the other major component of the first sound, which nearly always follows M₁, is produced at the tricuspid valve (Leatham, 1954, 1970; Heintzen, 1961; Haber and Leatham, 1965; Rees, Farru, and Rodriguez, 1972), this concept has been disputed (Di Bartolo et al., 1961; Shah et al., 1963; Delman, 1967; Luisada et al., 1971). The present study was, therefore, undertaken to ascertain whether this second major component (T₁) has the same time relation to the peak of the right atrial c wave as does M₁ to the left atrial c wave. Factors affecting the timing and intensity of T₁, as well as the amplitude of the right atrial c, are briefly discussed.

Subjects and methods

Using methods similar to those previously described from this laboratory (Lakier et al., 1972a, c), an electrocardiogram, external phonocardiogram, and simultaneous left atrial and left ventricular pressures were recorded during routine cardiac catheterization in 10 patients, 2 with aortic and 8 with mitral valve disease (Table). In addition, the right atrial pressure was recorded through the Brockenbrough catheter immediately before perforation of the atrial septum. Consecutive right atrial, right ventricular, and left atrial pressures were recorded through a 7F Rodriguez Alvarez catheter in 4 patients with an atrial septal defect of the secundum type. Simultaneous left and right atrial pressures were obtained through two such catheters in one subject in whom cardiac catheterization revealed a diagnosis of ‘straight back syndrome’ (Rawlings, 1966; de Leon et al., 1965) with an associated patent foramen ovale but an otherwise normal heart.
In all instances, phonocardiograms were recorded at a site between the left sternal border and apex at which both major components of the first sound could be demonstrated. Delay between the phonocardiographic and pressure recordings has previously been shown to be negligible (Lakier et al., 1972a) and can be ignored.

Results
With one exception (Case 8, Table), both major components of the first sound were demonstrated phonocardiographically and the onset of T₁ coincided with the peak of right atrial c. The interval between Q of the simultaneous electrocardiogram and T₁ (or right atrial c) ranged from 0·07 to 0·10 sec (Table). In the subject with the straight back syndrome and normal heart (Fig. 1), the 4 patients with an atrial septal defect, and 7 of the 10 with valvular disease, the Q-T₁ (right atrial c) interval exceeded that of Q-M₁ (left atrial c). However, in

![Figure 1](image1)

**FIG. 1** Simultaneous electrocardiogram, phonocardiogram, left atrial (LA), and right atrial (RA) pressure tracings in the patient (Case 15, Table) with a normal heart and the straight back syndrome. M₁ and the peak of left atrial c precede the peak of right atrial c and T₁ by 0·02 sec. See text. Time lines 0·04 sec.

![Figure 2](image2)

**FIG. 2** Reversal of the major components of the first sound in a patient (Case 9, Table) with very tight mitral stenosis. It can be seen in the top tracing, in which the phonocardiogram is recorded at the apex, that M₁ and the synchronous left atrial c peak occur almost immediately after the crossover (PCO) of LA and LV pressures. On the lower tracing, with the phonocardiogram recorded inside the apex, the vibration T₁ which starts at the peak of the right atrial c is shown to precede M₁.

In all 4 patients with an atrial septal defect left atrial c was of smaller amplitude than right atrial c and the intensity of M₁ was less than that of T₁ (Fig. 3).

Discussion
Good evidence for a tricuspid origin of the second major first sound component (T₁) was provided by
Heintzen’s observations (1961) on its movement and changes in intensity during respiration and after the Valsalva manoeuvre. Haber and Leatham’s (1965) findings in asynchronous ventricular contraction associated with right bundle-branch block, ectopic beats, and cardiac pacing supported this concept. However, the audibility of a right-sided component has been refuted (di Bartolo et al., 1961; Shah et al., 1963; Luisada et al., 1967; Delman, 1967; Van Bogaert, 1968; Sainani et al., 1968), and Luisada et al. apparently still believe (1971) that the major first sound components arise in the left ventricle and aorta.

Just as the onset of $M_1$ was shown to coincide with the peak of the left atrial c in our studies of the mitral components of the first heart sound (Lakier et al., 1972a, b, c), so a similar time relation between $T_1$ and right atrial c has been confirmed in this investigation. One of the arguments against the second major component arising on the right side of the heart is based on evidence (di Bartolo et al., 1961; Shah et al., 1963; Luisada et al., 1971) that tricuspid valve closure and the onset of right ventricular contraction precede this vibration by a variable time interval. However, and analogous to the mechanism of production of $M_1$, it should be emphasized that it is not tricuspid ‘closure’ which produces $T_1$ but the maximal tension on the apposed leaflets, and their chordae, at the limit of their ascent into the right atrial cavity. It is theoretically possible that a true tricuspid ‘closure’ sound precedes $T_1$, but this would usually be obscured by vibrations arising at the mitral valve.

As was expected, $M_1$ preceded $T_1$ in the majority

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TABLE Summary of data from 15 patients

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Diagnosis</th>
<th>Time intervals (sec)</th>
<th>$Q-M_1$ (LAC)</th>
<th>$Q-T_1$ (RAC)</th>
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<tr>
<td>1</td>
<td>Aortic stenosis</td>
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<td>0·09</td>
<td></td>
</tr>
<tr>
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</tr>
<tr>
<td>3</td>
<td>Mitral stenosis</td>
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<td>0·09</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Mitral stenosis</td>
<td>0·07</td>
<td>0·08</td>
<td></td>
</tr>
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<td>7</td>
<td>Mitral incompetence</td>
<td>0·06</td>
<td>0·09</td>
<td></td>
</tr>
<tr>
<td>8*</td>
<td>Mitral stenosis and incompetence</td>
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<td></td>
</tr>
<tr>
<td>9</td>
<td>Tight mitral stenosis</td>
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<td>0·07</td>
<td></td>
</tr>
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<tr>
<td>15</td>
<td>'Straight back syndrome'</td>
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<td>0·09</td>
<td></td>
</tr>
</tbody>
</table>

* Separate major components were not identified but left atrial c (LAC) and right atrial c (RAC) were synchronous—see text.

FIG. 3 A patient (Case 13, Table) with an atrial septal defect. (a) The relatively loud $T_1$ is synchronous with the peak of the right atrial c. (b) $M_1$ is soft and the left atrial c of small amplitude. For explanation, see text.
of patients in this study (Table). However, the first sound was single in one instance and the peaks of left atrial and right atrial c were then synchronous. In the 2 patients with very tight mitral stenosis, left atrial c and M1 were delayed to 0-10 sec and occurred after the right atrial c and T1. The components of the first sound were thus reversed, a feature that has previously been recognized in tight mitral stenosis (Leatham, 1954; Hultgren and Leo, 1958; Leatham, 1970) and left atrial myxoma (Goodwin et al., 1962).

In their investigation of sound and pressure events in mitral stenosis, Wooley et al. (1968) observed that M1 and the left atrial c coincided with the onset of an increased rate of rise of left ventricular pressure. This feature was readily apparent to us during our previous studies (Lakier et al., 1972a, c) and is invariably present when the mitral leaflets are voluminous and the valve mechanism mobile. A similar observation (Fig. 4) can often be made on the right side of the heart. It is understandable that the rate of ventricular pressure rise should increase during the isovolumetric contraction phase from the time that the slack in the atroventricular valve has been taken up. The point at which this change in ventricular pressure rise occurs is thus contributory in identifying the relevant major component of the first sound. Pocock, Tucker, and Barlow (1969), for example, noted that the increased rate of rise of right ventricular pressure in Ebstein’s anomaly coincided with a loud early systolic sound and postulated that the sound arose at the tricuspid valve, an hypothesis confirmed by both Fontana and Wooley (1972) and Crews et al. (1972).

The association of a soft M1 and loud T1 in atrial septal defect has previously been recognized (Leatham and Gray, 1956; McKusick, 1958; Lopez, Linn, and Shaffer, 1962), and Rees et al. (1972) have recently claimed that there is a correlation between the delay in T1 and the magnitude of the shunt. We were impressed by the relatively small amplitude of the left atrial c compared to that of the right atrial c in our 4 patients with atrial septal defect. Though the original explanation for the soft M1 and loud T1 provided by Leatham and Gray (1956) and McKusick (1958) was based on the premise that these sounds were caused by valve closure, the haemodynamic and functional anatomical events outlined by those authors remain relevant. Because of the increased volume of blood flowing into the right ventricle, the tricuspid valve remains wide open throughout diastole. With the onset of ventricular systole, the rate at which the tricuspid leaflets appose and subsequently billow into the right atrium is rapid and a relatively large right atrial c and loud T1 result. On the other hand, left atrial c is small and M1 relatively soft since left ventricular inflow is virtually completed before that ventricle contracts and thus the mitral leaflets would already be moving towards apposition.

The recognition of T1 by its synchronous occurrence with the peak of the right atrial c should prove useful in differentiating systolic clicks which, whether ejection or non-ejection, may occupy a similar position (Barlow, 1965; Hutter et al., 1971) in the cardiac cycle. T1 is delayed in cases of atrial septal defect (Rees et al., 1972) and it has been observed (Pocock and Barlow, 1971; McDonald et al., 1971) that non-ejection systolic clicks are not uncommonly associated with that congenital cardiac malformation. Should such a click occur early in systole, its time relation to the peak of right atrial c
would serve to distinguish it from T₁. It was in this way that we were recently able to identify an early non-ejection click associated with a submitral left ventricular aneurysm (Kanarek et al., 1973).

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