Tricuspid component of first heart sound¹

J. B. Lakier, K. R. Bloom, W. A. Pocock, and J. B. Barlow

From the Cardio-Vascular Research Unit, Department of Medicine, University of the Witwatersrand; and the Cardiac Clinic, General Hospital, Johannesburg, South Africa

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 (T_1) major component of the first sound coincided with the peak of the right atrial c wave just as the major left-sided component (M_1), 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 T_1 arises at the tricuspid valve. Analogous to the mode of production of M_1 , 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 T_1 . Some factors affecting the timing and intensity of M_1 and T_1 are mentioned.

The mechanism of production and factors influencing the timing and intensity of the major left-sided component (M_1) 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 M_1 , which is almost always the earlier of the two major first sound components, occurs after the valve has closed. M_1 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 M_1 can be seen to begin on a simultaneously recorded phonocardiogram. There are several important factors which affect the timing and intensity of M_1 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. M_1 and the peak of the left atrial c will be delayed, with M_1 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 M_1 , 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 (T_1) has the same time relation to the peak of the right atrial c wave as does M_1 to the left atrial c wave. Factors affecting the timing and intensity of T_1 , 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, 1960; de Leon *et al.*, 1965) with an associated patent foramen ovale but an otherwise normal heart.

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TABLE Summary of data from 15 patients

Case No.	Diagnosis	Time intervals (sec)	
		Q-M ₁ (LAC)	Q-T ₁ (RAC)
1	Aortic stenosis	0.065	0.09
2	Aortic stenosis	0.065	0.10
3	Mitral stenosis	0.07	0.09
4	Mitral stenosis	0.07	0.08
5	Mitral stenosis	0.07	0.10
6	Mitral incompetence	0.06	0.07
7	Mitral incompetence	0.06	0.09
8*	Mitral stenosis and incompetence	0.07	0.07
9	Tight mitral stenosis	0.10	0.07
10	Tight mitral stenosis	0.10	0.09
11	Atrial septal defect	0.07	0.10
12	Atrial septal defect	0.05	0.08
13	Atrial septal defect	0.06	0.08
14	Atrial septal defect	0.05	0.08
15	'Straight back syndrome'	0.07	0.09

* 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.

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

FIG. 4 Right ventricular pressure (RV) of the same patient (Case 13, Table) as in Fig. 2. T_1 coincides with the change (see arrows) in the rate of rise of the pressure.

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 M_1 were delayed to 0.10 sec and occurred after the right atrial c and T_1 . 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 M_1 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 atrioventricular 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 M_1 and loud T_1 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 T_1 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 M_1 and loud T_1 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 T_1 result. On the other hand, left atrial c is small and M_1 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 T_1 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. T_1 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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Requests for reprints to Professor J. B. Barlow, Department of Medicine, General Hospital, Johannesburg, South Africa.