Mitral components of the first heart sound^{1 2}

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Simultaneous left atrial and left ventricular pressure tracings, standard lead II of the electrocardiogram, and an external phonocardiogram were studied in 41 patients, all but 5 of whom had mitral valve disease. A soft low pitched vibration (M) was frequently observed at the point of cross-over (PCO) of left ventricular and left atrial pressures. We postulate that apposition of the mitral leaflets causes M and that this sound M, or the PCO point, marks the onset of the left ventricular isovolumetric contraction phase. The first major component of the first sound (M1) occurs later and at the same time as the peak of the 'c' wave (LAC) of the left atrial pressure tracing. We believe that the apposed mitral leaflets billow into the left atrium resulting in LAC, at the peak of which tension on the chordae tendineae and on the leaflets themselves produces M1. The well-known phonocardiographic measurement of the Q-M1 interval is thus the same as the Q-LAC interval. Furthermore, this interval Q-M1 (LAC) can be subdivided into Q-M (PCO) and M (PCO)-M1 (LAC) intervals. Evidence is provided that measurement of these intervals and of the height of LAC are contributory in the assessment of mitral valve pathology and function.

The previously recognized variations in the timing and intensity of M1, which may be associated with mitral valve pathology, myocardial decompensation, and with disturbances of conduction and rhythm, are briefly discussed and explained.

Though it is generally accepted that the two components of the second heart sound depend on aortic and pulmonary valve closure (Leatham, 1954; Delman, 1967), the number of components of the first sound and their mechanism of production remain disputed and ill understood (Dock, 1965; Delman, 1967). It has been shown by phonocardiography (Rappaport and Sprague, 1942; Counihan *et al.*, 1951; Dayem and Raftery, 1966; Rushmer, 1970) that the normal first heart sound has at least 4 vibrations or groups of vibrations (Fig. 1). These comprise initial low frequency vibrations, two higher pitched 'major vibrations', and then low frequency 'after vibrations.' Though the early low frequency vibrations are clinically audible in some instances (Kincaid-Smith and Barlow, 1959a), it is the two major components only which are heard and assessed by most clinicians. The 'after vibrations' are probably never audible.

The initial low frequency components have been variously attributed to 'residual vibrations of the auricular sound' (Rappaport and Sprague, 1942), to 'vibrations due to isometric contraction of the ventricles' (Luisada, Mendoza, and Alimurung, 1949), to muscular factors (Counihan *et al.*, 1951; Nazzi, Rico, and Meda, 1954), and to 'a combination of muscular factors with auricular components' (Orias, 1949). Kincaid-Smith and Barlow (1959a) observed that one of the initial low pitched vibrations, which coincides with or occurs just after the Q wave of the simultaneous electrocardiogram, must depend on atrial contraction for its production since it disappears with atrial fibrillation or nodal rhythm. They called this the 'atrial component or the first heart sound' and stated that it was sometimes clinically audible.

In 1941, Smith, Gilson, and Kountz postulated that the first sound resulted from muscular vibrations of the left ventricle, and apparently Luisada and co-workers (Luisada, Gagnon, and Ikeda, 1970) still consider that the major components are largely 'of left ventricular origin'. Most workers, however, believe that they are related to valvular events,

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FIG. 1 Diagrammatic representation of the four groups of vibrations of the first heart sound as recorded phonocardiographically. The initial low frequency atrial component of the first sound ACF occurs with, or just after, the Q wave of the simultaneous electrocardiogram. A second low frequency vibration M occurs after Q, at the time of the peak of the R wave, and is followed by two higher pitched vibrations M₁ and T₁, which comprise the major components of the first heart sound. Lastly, the so-called 'after vibrations' are depicted.

though opinions differ as to the mechanism of production. Some consider that the opening of the semilunar valves causes the second major component (Rappaport and Sprague, 1942), while others ascribe the sounds to either the closure of the mitral valve alone (Dayem and Raftery, 1966), or to the closure of both the mitral and tricuspid valves (Wolferth and Margolies, 1930; Leatham, 1954; Braunwald and Morrow, 1958; Hultgren and Leo, 1958). Dock (1965) explains the vibrations on the basis of tensing of the mitral and tricuspid leaflets and chordae *after* closure. Heintzen's observations (1961) on the behaviour of the two major components during and after the Valsalva manoeuvre have provided good evidence that both the mitral and tricuspid valves are involved in their production.

In this investigation an early low frequency vibration (M), which occurs near the peak of the R wave of the simultaneous electrocardiogram and therefore after the so-called atrial component (Kincaid-Smith and Barlow, 1959a), and the first major component of the first heart sound (M₁) have been studied. Evidence is provided that M is caused by apposition of the mitral leaflets and M₁ by tension on the chordae and leaflets after the mitral valve is 'closed'. In addition, we show

that the intensity of M₁, the size of the left atrial 'c' wave, and the timing of M and M₁ are useful in the assessment of the function and pathology of the mitral valve.

Material and methods

Simultaneous left ventricular and left atrial pressure tracings, an external phonocardiogram, and standard lead II of the electrocardiogram, recorded during routine cardiac catheterization, were available for study in 41 patients. There were 9 patients with tight mitral stenosis, 18 with pure mitral incompetence of more than moderate severity in all but one, 9 with haemodynamically significant mitral incompetence and stenosis, 2 with hypertrophic obstructive cardiomyopathy, and 1 patient had a congestive cardiomyopathy. Two patients, catheterized because of features suggestive of a left atrial myxoma, had normal hearts. Twenty-one of the patients were in sinus rhythm and 20 in atrial fibrillation (Fig. 2).

The left atrium was entered by the transseptal route using a 69 cm 8.5 bore USCI Teflon catheter. The catheter was positioned so that the tip lay freely above the mitral valve. Retrograde left ventricular catheterization, by the Seldinger technique, was achieved using a KIFA or USCI 125 cm 7 bore polyethylene catheter. An Electronics for Medicine DR-12 recorder was used in all instances. Simultaneous left atrial and left ventricular pressures were recorded through Statham P23Db strain gauge type transducers at paper speeds of 75, 100, and 150 mm/sec on sensitivities of 40 and 100 mmHg. An external phonocardiogram, using a frequency response range of 40–2,000 cycles/sec and an Electronics for Medicine Ps-1 displacement microphone, was recorded during held expiration at the site of maximum intensity of the first heart sound. Measurements were taken from Q of the simultaneous electrocardiogram to the early vibration M and the first major component M₁ of the first heart sound. In addition, the distances between Q and the crossover of left ventricular and left atrial pressures (PCO) and from PCO to the apex of the left atrial 'c' wave (LAC) were measured. The height of the left atrial 'c' wave was measured from the point PCO to its peak with the attenua-

FIG. 2 Diagnosis in the 41 patients studied, 20 of whom had atrial fibrillation.

tion set so that 3 mm = 1 mmHg. In one patient with mitral incompetence the height of the 'c' wave could not be assessed as the left atrial tracing was overdamped.

Possible delay between phonocardiographic and pressure tracings was assessed by a method similar to that described by Wood (1950). The tip of a USCI 125 cm 7 bore polyethylene catheter was placed in close proximity to the balloon of a single lumen Rashkind catheter within a container of water. An Electronics for Medicine Ps-1 displacement microphone was attached to the outside of the container, close to the catheter tip, and the balloon of the Rashkind was then burst. The recorded delay between pressure and sound measurement was less than 0.002 sec and could therefore be ignored.

Results

The soft vibration M was recorded in 36 cases and coincided with the crossover (PCO) of

FIG. 3 Diagrammatic representation of simultaneous standard lead II of the electrocardiogram (STD2), external phonocardiogram (PCG), left atrial (LA), and left ventricular (LV) pressures. The early vibration M coincides with the point of crossover (PCO) of LV and LA pressures. M₁ occurs after PCO at the time of the peak of the left atrial 'c' wave (LAC). The Q-M₁ interval can thus be subdivided into Q-M plus M-M₁ which is the same as Q-PCO plus PCO-LAC. a = left atrial 'a' wave.

FIG. 4 Diagrammatic representation of the Q-M₁ intervals, separated into Q-M (PCO) (cross-hatched) and M (PCO)-M₁ (LAC) intervals, in the 41 patients. In the patients with combined mitral stenosis (MS) and incompetence (MI) large letters indicate the lesion which predominates. For details see text.

FIG. 5 Simultaneous electrocardiogram, phonocardiogram, left atrial, and left ventricular pressures in a patient with tight mitral stenosis in sinus rhythm. The Q-M₁ (LAC) interval is prolonged to 0.08 sec, the major cause of the prolongation being a Q-PCO (M) interval of 0.07 sec. M₁ (LAC) occurs 0.01 sec after PCO. The vibration M is obscured by the presystolic murmur. LAC is of small amplitude indicating a rigid valve mechanism. Paper speed 100 mm/sec. In this and subsequent tracings time lines measure 0.04 sec.

left ventricular and left atrial pressures. It could not be identified in the 5 patients with tight mitral stenosis in sinus rhythm as it was obscured by the presystolic murmur. The component M₁ occurred after PCO and either coincided with, or was within 0.01 sec of, the peak of the left atrial 'c' wave (LAC) in all instances. The well-known phonocardiographic measurement of Q-M₁ can thus be subdivided into Q-M plus M-M₁ intervals which are the same as Q-PCO plus PCO-LAC (Fig. 3). In the patients with atrial fibrillation the Q-M₁ (LAC) interval was inversely related to the length of the preceding diastolic pause but the variation did not exceed 0.01 sec. In these cases the average measurement of all intervals over six successive cardiac cycles was taken.

The Q-M₁ (LAC) interval fell within the normal range of 0.06 sec (Barlow and Kincaid-Smith, 1960; Dack *et al.*, 1960) in the 2 patients with normal hearts and the 3 with a cardiomyopathy. The Q-M (PCO) and M (PCO)-M₁ (LAC) intervals were equal. The Q-M₁ (LAC) interval was 0.06 sec in 1 of the 9 patients with tight mitral stenosis but was abnormally prolonged (0.065–0.10 sec) in the remaining 8 (Fig. 4). The prolongation was due mainly to an increase in the Q-M (PCO) interval (Fig. 4 and 5), whereas the measurement M (PCO)-M₁ (LAC) was short and was never more than 0.025 sec. In one instance PCO coincided with M₁ (LAC). In the 18 patients with mitral incompetence, the Q-M₁ (LAC) interval was also prolonged and ranged between 0.065 and 0.09 sec (Fig 4 and 6). The Q-M (PCO) interval was 0.03 sec in 4 cases and 0.035–0.04 sec in 12. In contrast to the patients with mitral stenosis, the prolongation of Q-M₁ (LAC) resulted largely from a lengthened M (PCO)-M₁ (LAC) interval, which ranged from 0.03 to 0.05 sec (Fig. 4). A similar trend was noted in the 9 patients with mixed mitral incompetence and stenosis. Thus, the 2 patients in whom incompetence was the more significant lesion had a M (PCO)-M₁ (LAC) interval prolonged to 0.04 sec, whereas in all cases with dominant stenosis it was the Q-M (PCO) interval which was relatively prolonged (Fig. 4).

Good correlation was found between the mobility of the mitral valve, assessed either by cineangiography, at operation, or both, and the height of LAC (Fig. 7). A relatively large amplitude LAC, greater than 8 mm, was associated with a mobile valve, whereas a LAC of small amplitude generally indicated impaired mobility of the mitral valve mechanism.

FIG. 6 Simultaneous electrocardiogram, phonocardiogram, left atrial, and left ventricular pressures in a patient with pure mitral incompetence and voluminous leaflets. The Q-M₁ (LAC) interval is prolonged to 0.08 sec with M (PCO)-M₁ (LAC) of 0.04 sec. Both LAC and M₁ are of good amplitude, indicating a pliable valve mechanism. Paper speed 75 mm/sec.

Discussion

Investigators have failed to agree on the actual time of closure of the mitral valve. Closure has been regarded as occurring at the point of crossover of left ventricular and left atrial pressures (Braunwald and Morrow, 1958; Di Bartolo *et al.*, 1961) or after pressure crossover at a time related to the 'c' wave of the left atrial tracing (Rich, 1959; Brockman, 1966; van Bogaert, 1968). It appears to us that once the pressure in the left ventricle exceeds that in the left atrium the mitral leaflets must be in apposition, the valve mechanism must then be 'closed', and the point of pressure crossover thus indicates the onset of the isovolumetric ventricular contraction phase. Because of the excellent correlation in time between the crossover of left ventricular and left atrial pressures and the M component of the first heart sound, we believe that M is caused by the apposition of the leaflets (Lakier *et al.*, 1970). Supportive evidence that M arises at the valve is provided by the phonocardiographic recording of a mitral 'closure' sound during ventricular diastole in some cases of

FIG. 7 Correlation between the mobility of the mitral valve, as assessed by cineangiography and at operation (22 patients), and the amplitude of LAC. It can be seen that an LAC greater than 8 mm is associated with a pliable valve mechanism. Furthermore, a correlation is shown ($P < 0.05$) in the cases of pure mitral incompetence between the height of LAC and the length of the M (PCO)-M1 (LAC) interval. For full explanation, see text.

Solid symbols indicate a rigid, semi-rigid, and open symbols a pliable valve mechanism.

severe aortic incompetence (Meadows *et al.*, 1963; Rees *et al.*, 1964; Wigle and Labrosse, 1965). Such a sound must be unrelated to myocardial contraction since it occurs well before the onset of ventricular systole. We therefore suggest that the mitral valve closes at PCO and the early, low frequency vibration M results from leaflet apposition, that the apposed leaflets then billow back into the left atrium producing LAC, at the peak of which the chordae and leaflets come under maximal tension and cause the sound M1. This postulate is compatible with the observations of Rich (1959) and Wooley *et al.* (1968, 1970) that M1 occurs at the peak of the 'c' wave of the left atrial tracing and also with the observations of both McCall and Price (1967) and

Thompson *et al.* (1970) who showed, by means of simultaneous phonocardiography and left ventricular cineangiography, that M1 coincides with the maximal ascent of the mitral leaflets into the left atrium.

It is well known that the intensity of the first heart sound alters with the changing PR interval of complete atrioventricular dissociation (Wolferth and Margolies, 1930; Reinhold and Rudhe, 1957) and with the varying length of the preceding diastolic pause in atrial fibrillation (Rytand, 1949; Tavel, Feigenbaum, and Campbell, 1965). These observations have been explained on the basis that the leaflets of a valve which is relatively wide open at the onset of ventricular contraction have a longer excursion before apposing or 'closing', thus producing a louder sound. The fact that the major component M1 occurs *after* valve closure is quite compatible with these observations since the degree of tension in the chordae and leaflets at the peak of the billow would be greater where the leaflets billow back from an original wide open as compared to a semi-closed position and this would be an important factor influencing the intensity of M1. Another factor which must affect tension on leaflets and chordae, and hence the amplitude of M1, is the rate of rise of left ventricular pressure. It would be logical to expect that the more rapid the rise of pressure the greater would be the force by which the leaflets and chordae are put 'on stretch', hence there would be increased tension on these structures resulting in a louder M1. In an experimental study using dogs, Sakamoto *et al.* (1965) showed a good correlation between the rate of rise of left ventricular pressure and the intensity of the first sound. Though these workers concluded that the first sound resulted from 'vibration of all left ventricular structures', their observations are, in fact, compatible with, or even confirmatory of, a valvular origin alone of M1. Dock (1959), a leading protagonist of the valvular origin of heart sounds, has shown experimentally that, whereas very large tensing forces are necessary to produce audible vibrations from ventricular muscle, relatively little force is required to evoke sound when leaflets or chordae are drawn taut. As a corollary it may be inferred that only small alterations in the amount of tension on leaflets or chordae may significantly affect the intensity of the sound produced.

The position of the mitral leaflets at the onset of left ventricular contraction and the rate of pressure rise must both contribute to the very loud M1 in cases of mitral stenosis with a pliable valve mechanism (Leatham,

1970; Thompson *et al.*, 1970). In such instances, the leaflets are maintained in the 'open' position by the high left atrial pressure until the delayed PCO point, which thus occurs when the left ventricular pressure is rising steeply (Fig. 5). As a consequence M₁ is loud and also, as is well recognized, delayed (Wooley *et al.*, 1968). The prolongation of the Q-M₁ (LAC) interval in tight mitral stenosis is thus derived predominantly or entirely from a long Q-M (PCO) interval (Fig. 4 and 5). The delay in crossover of left ventricular and left atrial pressures has previously been noted to be a major factor in the prolonged Q-M₁ interval by Messer *et al.* (1951) and Wooley *et al.* (1968, 1970). Analysis of our results reveals that the Q-M₁ interval is also prolonged in cases of mitral incompetence with voluminous leaflets and mobile, elongated chordae, thus confirming the findings of Günther and Münster (1968). The PCO point and the sound M occur relatively early in mitral regurgitation (Fig. 4 and 6) since the left atrial end-diastolic pressure is usually normal or only slightly raised and the prolonged Q-M₁ is due mainly to an increased M (PCO)-M₁ (LAC) interval (Fig. 4). We believe that the prolongation is due to the increased time taken by the mobile voluminous leaflets to reach the apex of their ascent into the left atrium at which point they would come under tension. Tensing of the leaflets and their chordae at the apex of a relatively long ascent into the left atrium, analogous to a sail snapping taut in the wind, may be a factor contributing to the loud M₁ in these cases. In either mitral incompetence or stenosis, if the valve mechanism is mobile the leaflets are able to billow into the left atrial cavity resulting in a LAC of large amplitude (Fig. 7). If the leaflets are rigid with shortened, thick chordae, little ascent or billow of such leaflets into the left atrium is possible (Nichols *et al.*, 1956) and consequently LAC is of small amplitude (Fig. 7) and M₁ is usually reduced in intensity. However, a small group of patients with rigid valve leaflets but relatively mobile chordae tendineae do have a loud M₁ (Bramwell-Jones *et al.*, 1970). In such patients it is suggested that some ascent of the leaflets is possible and the resultant tension on the chordae is responsible for the production of the loud M₁.

A correlation ($P < 0.05$) has been shown in cases of pure mitral incompetence between the M (PCO)-M₁ (LAC) interval and LAC, in that the longer the M (PCO)-M₁ (LAC) interval the taller is LAC (Fig. 7). This observation is compatible with our belief that, provided the rate of left ventricular pressure rise

is similar, prolongation of the M (PCO)-M₁ (LAC) interval will be greatest in those patients with the longest chordae and most voluminous leaflets and it is in such cases that a tall LAC would be anticipated. We suspect that a similar correlation could be shown in mitral stenosis and in combined mitral stenosis and incompetence if the number of cases was statistically significant. In those instances, however, the M (PCO)-M₁ (LAC) intervals would always be shorter than in pure mitral incompetence because of the shorter chordae and less voluminous, sometimes rigid leaflets and also because the left ventricular pressure rise is relatively more rapid at the time of the higher PCO point.

Finally, we suggest that our concept of the factors affecting the intensity and timing of M₁ elucidate two other observations on the first sound not hitherto explained. Leonard, Weissler, and Warren (1958) showed that the Q-M₁ interval was longer and Kincaid-Smith and Barlow (1959b) noted that M₁ was softer when associated with an early atrial sound than when the atrial sound moved later. It is generally accepted that an atrial sound occurs later with improvement in myocardial function and vice versa (Kincaid-Smith and Barlow, 1959b; Tabatznik, 1964) and that the rate of rise of left ventricular pressure is more rapid with such improvement (Gleason and Braunwald, 1962). We thus submit that it is this more rapid rise in left ventricular pressure which results in M₁ becoming earlier and louder as the atrial sound migrates to a later position.

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