Non-invasive observations on initial low frequency vibrations of the first heart sound – correlation with the ‘presystolic’ murmur in mitral stenosis¹

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SUMMARY The initial low frequency component of the first heart sound, ‘M’, has been studied in normal subjects, and in patients with various prosthetic mitral valves and with mitral stenosis, using simultaneous low frequency phonocardiography, echocardiography, and apex cardiography. These techniques showed ‘M’ to have a constant morphology in preisovolumic systole. In mitral stenosis, ‘M’ and the preisovolumic ‘presystolic’ murmur appear to be the same phonocardiographic phenomenon. While ‘M’ was present in sinus rhythm, augmentation of this normal vibration occurred particularly during the short cycles of atrial fibrillation. Leaflet coaption and movement of the ventricular wall as detected echocardiographically do not appear to play a role in its pathogenesis but the sound could emanate from the ventricular wall as it tautens and decreases its compliance at the onset of systole.

The initial low frequency component of the first heart sound has been studied for more than 150 years (Orias, 1936; Eckstein, 1937; Smith *et al.*, 1941; Lakier *et al.*, 1970; Luisada and Argano, 1971; Lakier *et al.*, 1972a, b; Armstrong and Gotsman, 1973). We hope to show that the sound is more than just an abstruse phonocardiographic finding but has clinical significance in that it is responsible for the short crescendo, so-called ‘presystolic’, murmur of mitral stenosis in atrial fibrillation, as suggested by Tavel and Bonner (Bonner *et al.*, 1976; Tavel and Bonner, 1976). This sound, which occurs near the peak of the R wave of the electrocardiogram and at the crossover point of the left atrial and ventricular pressures, has been termed ‘M’ by Lakier *et al.* (1970) and ‘O’ by Luisada and Argano (1971) (Fig. 1). In contrast, the later higher frequency component of the first heart sound (M₁) occurs at the time of the left atrial C wave (Lakier *et al.*, 1972a, b).

The cause of ‘M’ remains controversial. The sound has been attributed to initial coaption of the mitral valve leaflets and the sound ‘M₁’ to subsequent tensing of the mitral cusps and chordae

(Lakier *et al.*, 1970, 1972a, b). Other studies have suggested that muscular contraction producing sudden deceleration of blood flow across the atrio-ventricular orifice is more likely to produce the sound. The advent of strip-chart echocardiography has provided the potential to investigate further the role of muscular contraction as the cause of ‘M’ and also the relation of this sound to the ‘presystolic’ murmur in atrial fibrillation. This paper reports our findings using simultaneous echocardiography, phonocardiography, and apex cardiography in normal subjects, patients with mitral stenosis, and patients with various prosthetic mitral valves.

Subjects and methods

The material comprised 12 normal subjects, 5 patients with Starr-Edwards, 5 with Beall, 3 with Lillehei-Kaster, and 2 with Björk-Shiley mitral valve prostheses. In addition, there were 36 patients with mitral stenosis (8 in atrial fibrillation); 14 of these were studied by kinetocardiography and phonocardiography only. The equipment used in the first 4 cases was a standard Ekoline echocardiograph with polaroid camera, gated to record ball movement on a Sanborn 2 channel photographic recorder, with a stethoscopic apical phonocardi-

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Fig. 1 Simultaneous electrocardiogram (ECG) (A), phonocardiogram (PCG) (B) recorded at 50 to 20 000 cycles per second at the mitral area, and kinetocardiogram (KCG) (C) recorded from the apex. Normal youth, aged 16, left lateral position. 'M' is distinguishable from S4 and the high frequency 'M₁', and is synchronous with the onset of upstroke (outward movement) of the KCG. The deflection 'M' occurs after Q and its peak near the R wave of the ECG. RFW and SFW = rapid and slow filling waves. Paper speed 100 mm/s.

gram and kinetocardiogram as reference traces. Kinetocardiography was used in most of the earlier cases and in the left oblique position, if a satisfactory apical impulse could not be obtained in the supine position. These phonocardiograms were either recorded on the standard 'stethoscopic' range with an electrocardiogram as reference or, when kinetocardiograms and simultaneous sounds from the apex were needed, with a Hewlett Packard 21050BT Transducer and Heart Sound Amplifier 1506A with a cut-off frequency of 50 Hz linked to the Sanborn electrocardiographic channel. It should be emphasised that the stethoscopic range is very sensitive to low frequencies. The remaining studies were performed with a Picker echocardiograph linked to a Cambridge 6-channel physiological recorder which inscribed, as needed, an electrocardiogram, a low frequency (60-600 Hz) phonocardiogram, an apex cardiogram, and an echocardiogram of ball, disc, or mitral valve movement. Patients investigated with this equipment had apex cardiograms as opposed to kinetocardiograms as a matter of convenience. Though in general, apex cardiograms and kinetocardiograms cannot be regarded as interchangeable, there is no difference in relation to this paper as it is only the earliest onset of the upstroke (onset of preisovolumic systole) which is relevant. Tracings recorded with a 'thick' line often show the morphological pattern of 'M' more clearly than 'thin' line recordings.

Echoes were recorded just below the mitral valve simultaneously with the aforementioned variables.

Results

(i) NORMALS

'M' was recorded in all 12 subjects and was usually of small amplitude. It was not distinguishable on auscultation from other low frequency sounds.

(ii) PROSTHETIC MITRAL VALVES

'M' could be clearly heard using the bell of the stethoscope at the apex by two observers in all patients. The echocardiogram proved to be very sensitive and enabled the exact moment of the start and completion of closure of ball, disc, or mitral valve to be defined and correlated with the electrocardiogram and phonocardiogram (Fig. 2 and 3). 'M' was recorded and defined in every case. It was often biphasic and had more than one peak, as had been seen among normals and in patients with mitral valve disease. In many patients with prosthetic valves the sound was of greater amplitude than in normal persons or in patients with mitral valve disease and facilitated the accurate timing of 'M' in relation to ball or disc movement. This movement served as a useful reference point for the crossover of left atrial and ventricular pressures. It was very noticeable that in atrial fibrillation 'M' often varied inversely in size with the preceding

Fig. 2 Simultaneous echocardiogram (A), PCG (B), and ECG (C) in a patient with a Björk-Shiley mitral prosthesis. The CC (closing click) coincides with the apposition of the echoes from the disc and prosthetic ring. 'M' is loud and occurs at a time when the disc is still open. OC = opening click. Paper speed 100 mm/s. Time lines 40 ms.

RR interval—a short RR interval producing a larger than usual 'M' (Fig. 3).

The onset of 'M' vibrations always occurred after the Q wave of the electrocardiogram and just before the initial rise of the apex cardiogram. The echocardiogram showed this point to be approximately 30 to 40 ms before the start of ball closure (Fig. 2, 3). This movement has two phases: a slow phase resembling a shoulder, followed by a fast phase lasting about 40 ms which ends abruptly as the ball strikes the valve seat. At this moment the high frequency and high amplitude closing click is clearly and separately recorded. The peak of 'M' occurs at the very onset of closure, at the shoulder of the echo of ball movement, presumably when the

pressure in the left ventricle exceeds that in the left atrium. This shoulder is an excellent reference point for identifying 'M'. There is, therefore, a gap between the onset of 'M' at which time the ball is stationary, and the peak of 'M' when it just begins to move. 'M' may have more than one wave, and at times the initial wave may show a very low frequency of 25 to 50 Hz; its onset just precedes the initial movement of the apex cardiogram.

(iii) MITRAL STENOSIS

The timing of 'M' was the same in those patients with mitral stenosis, where the configuration of the leaflet echoes was very similar to those of a ball or disc. It was noticeable, however, that at the

Fig. 3 Simultaneous echocardiogram (A), apex cardiogram (ACG) (B), PCG (C), and ECG (D) in a patient with a Starr-Edwards mitral prosthesis. The onset of 'M' just precedes the initial rise of the ACG, close to the R wave of the ECG at which time the echocardiograph shows a stationary ball. Note that 'M' is louder in the first two short cycles than in the third cycle after a long diastole. The major vibration of 'M' is marked. Paper speed 100 mm/s. Time lines 40 ms.

Fig. 4 Simultaneous echocardiogram (A) phonocardiogram (B) and electrocardiogram in mitral stenosis. At the time of 'M' the anterior and posterior mitral leaflets (AL+PL, respectively) are separate. 'M₁' occurs at the most posterior point of movement of both leaflets. In the second cycle where diastole is shorter, 'M' is much augmented and produces pre-systolic accentuation of the mid-diastolic murmur (MDM). A₂=aortic component of the second heart sound. OS=opening snap. Paper speed 100 mm/s. Time lines 40 ms.

Fig. 5 Simultaneous echocardiogram (A), PCG (B), and ECG (C) in a normal subject. The echocardiogram shows that 'M₁' occurs at the point of apposition of the anterior (AL) and posterior (PL) leaflets at the onset of isovolumic ventricular systole. 'M' precedes this point and the leaflets of the mitral valve are clearly separated, but beginning to close. Paper speed 100 mm/s. Time lines 40 ms.

Fig. 6 Top tracing: simultaneous high frequency (logarithmic) phonocardiogram (A) and electrocardiogram (B) showing a mid-diastolic murmur (MDM) followed by a 'presystolic' murmur (arrow). Lower tracing: same patient with simultaneous low frequency (stethoscopic) phonocardiogram (A) and kinetocardiogram (B) which identifies the sound in pre-systolic systole as 'M'. Paper speed 75 mm/s. Time lines 40 ms.

shoulder of the mitral valve echogram the anterior and posterior leaflets were still separated (Fig. 4). Leaflet separation was also evident at the time of 'M' in normal subjects in whom leaflet apposition occurred at the time of 'M₁' (Fig. 5).

'M' frequently showed a constant morphology from beat to beat, and, once identified by a reference trace, could often be clearly recognised in subse-

quent beats by its shape alone. In 32 of our 36 patients with mitral stenosis the sound was identified on low frequency tracings where it appeared to constitute the crescendo portion of the audible 'presystolic' murmur both in sinus rhythm and in atrial fibrillation (Fig. 4, 6, 7). The sound was particularly augmented by the short cycles during atrial fibrillation (Fig. 4).

Fig. 7 Simultaneous echocardiogram of mitral valve (A), phonocardiogram (B), and electrocardiogram (C) in mitral stenosis. The presystolic vibrations after atrial systole are interrupted by the typical morphology of 'M' forming the preisovolumic systolic murmur. Paper speed 100 mm/s. Time lines 49 ms.

Fig. 8 Simultaneous echocardiogram (showing septal echo (A), chordal echo (B), endocardial echo (C), phonocardiogram (D), and electrocardiogram (E) in a normal subject. At the time of 'M' the endocardial echo is straight and moves anteriorly 80 ms after 'M₁'. Paper speed 75 mm/s. Time lines 40 ms.

Fig. 9 Simultaneous logarithmic phonocardiogram (log) in mitral area (MA) and tricuspid area (TA) in a patient with Beall mitral and tricuspid valve prostheses. Though the calibration is less sensitive in the MA, 'M' is of similar amplitude to the second sound (2) and is absent in the TA. Both mitral and tricuspid closure sounds are clearly shown and are distinct from the preceding 'M'.

(iv) LEFT VENTRICULAR WALL MOVEMENT

The echocardiogram of the endocardium of the posterior left ventricular wall readily records forward movement in systole and a straight line or backward movement in diastole. The peak of 'M' with high paper speeds of 100 mm/s was seen to occur well before any detectable anterior systolic movement (Fig. 8).

Discussion

'M' is not merely a recordable vibration but may at times be an audible sound localised to the apex. In the past most authorities have regarded the initial low frequency vibration as a strictly phonocardiographic phenomenon. In Fig. 2, 3, 7, and 9 it will be noted that the size and frequency of 'M' is similar to that of aortic valve closure. We have found that 'M' is easily detected in most patients with the Starr-Edwards mitral prosthesis when the bell of the stethoscope is placed precisely over the apex. While 'M' may be recorded by high and medium frequency phonocardiograms, it is seen much more readily and more often with a clearly recognisable morphology in the low frequency range.

The crescendo 'presystolic' murmur of mitral stenosis is generally accepted to be a result of increased presystolic flow as a consequence of atrial systole. Crescendo presystolic murmurs have, however, also been heard and recorded in the presence of atrial fibrillation. These have clearly been shown to occur late, after the onset of preisovolumic systole and are, therefore, systolic and not 'presystolic'. They have been attributed to an increased velocity, but diminishing volume of flow in the face of apposing mitral leaflets and a diminishing pressure gradient before valve closure (Criley *et al.*, 1971; Criley and Hermer, 1971; Lakier *et al.*,

1972b). While agreement exists that these vibrations occur in early systole, serious reservations have recently been expressed by Tavel and Bonner that increasing velocity of flow across a closing mitral valve is the actual mechanism (Bonner *et al.*, 1976; Tavel and Bonner, 1976). These authors have propounded the thesis that early systolic accentuation of the initial low frequency component of the first heart sound may be responsible for the so-called 'presystolic' murmur during the short cycles of atrial fibrillation. Two simultaneous events were considered necessary to produce or enhance the sound: (1) active flow of blood into the ventricle; (2) an abrupt change in ventricular compliance of the left ventricle at the onset of systole which decelerates flow.

Stimulated by these observations we have carefully scrutinised our low frequency phonocardiographic tracings and by the timing of 'M' in relation to the electrocardiogram, echocardiogram, and apex cardiogram have found that the major vibrations of 'M' also occur at the very onset of preisovolumic systole. The sound has a clearly recognisable typical morphology identifiable from beat to beat. In atrial fibrillation the so-called 'presystolic' accentuation of the diastolic murmur of mitral stenosis could be accounted for by the presence of 'M'. In sinus rhythm 'M' constituted that portion of the presystolic murmur which fell in isovolumic systole (Fig. 6, 7). Examination of the low frequency tracings and simultaneous echocardiograms from the paper by Toutouzas *et al.* (1974) shows the same findings.

We have not made any studies relating to flow across the mitral valve. It is interesting to note, however, that our non-invasive parameters show the ball of a Starr-Edwards prosthesis to be stationary or just beginning to move at the peak of

'M'. It is, therefore, difficult to conceive how augmentation of 'M' could be accounted for by postulating an increase in the velocity of flow through a diminishing orifice when there has been insignificant travel of the ball from the apex to the base of the prosthesis. Using a transseptal Doppler ultrasound technique, Kalmanson *et al.* (1975) have shown that the velocity of blood flow across the mitral valve is decreasing at this time.

The initial low frequency component of the first heart sound has been attributed to either muscular activity, tricuspid valve closure, or coaption of the leaflets of the mitral valve by various authors (Eckstein, 1937; Smith *et al.*, 1941; Lakier *et al.*, 1972a, b; Kalmanson *et al.*, 1975). Lakier *et al.* (1970) showed an excellent correlation in time between the point of crossover of the left atrial and ventricular pressures and 'M'; they believed, therefore, that the leaflets of the mitral valve coapted to produce the vibration 'M' once the pressure in the left ventricle exceeded that in the left atrium. Subsequently, the apposed leaflets billow into the left atrium at the time of the left atrial 'C' wave and cause the mitral sound 'M₁' (Lakier *et al.*, 1970, 1972a, b). The demonstration that 'M' may occur after mitral valve replacement casts serious doubt upon this explanation (Armstrong and Gotsman, 1973). In support of their concept, however, is the phonocardiographic recording of a mitral closure sound during ventricular diastole in some cases of severe aortic regurgitation (Wigle and Labrosse, 1965). This could suggest that leaflet coaption might at least partially contribute to the production of 'M'. Our studies show this to be incorrect because 'M' occurs well before the echocardiographic closing movement of a ball or disc. In addition, echoes recorded from both normal anterior and posterior leaflets and in mitral stenosis show the leaflets to be well separated at the time of 'M'. Leaflet apposition occurs later, at the time of 'M₁' or the closing click of a prosthesis. Tricuspid closure, postulated by Hultgren and Hubis (1965) as a cause of 'M' is unacceptable, since we have previously shown (Armstrong and Gotsman, 1973) that 'M' is present after combined mitral and tricuspid valve replacement and in any event tricuspid closure occurs far too late to be responsible for 'M' (Fig. 9) (Waider and Craige, 1975).

Smith *et al.* (1941) thought that the initial low frequency vibrations 'M' were caused by muscular activity because they were not abolished in the dog heart by occluding the venae cavae, by tensing a ligature around the atrioventricular sulcus, or by inflating balloons in the ventricular cavities. Sounds were recorded as long as the vigour of myocardial contraction was maintained. Eckstein (1937) pro-

duced experimental evidence favouring muscular activity as the cause of this sound but emphasised that a critical amount of shortening was required for its production. In addition, Wiggers (1923) showed that contraction of ventricular muscle of cats produced a sound and thought that the sound resulted from sudden tautening of muscle fibres.

Echocardiographically it is possible to detect anterior movement of the posterior endocardium of the left ventricle during systole. Our tracings indicate that though the first inscription of 'M' coincides with the initial upstroke of the apex cardiogram, which signals the onset of systole (Manolas *et al.*, 1975), it precedes any forward movement of the endocardial echo by 60 ms. Forward movement of the endocardial echo is detected shortly after 'M₁' or the closing click of a prosthesis. These observations are in keeping with the concept of isovolumic systole. As detected by the endocardial echo we are, therefore, unable to correlate 'M' with actual movement of the ventricular wall and retract our previous belief that 'M' is caused by muscular movement (Armstrong and Gotsman, 1973). It seems that echocardiographic movement of the posterior wall reflects volume changes in the left ventricle during the ejection phase rather than changes in configuration during isovolumic systole. Because of the complex array of longitudinal and circular muscle fibres within the left ventricle, the earliest motion in isovolumic systole is transferred to the apex which moves downward and outward, while the posterior wall is held stationary.

It appears from our non-invasive correlates that 'M' and the preisovolumic vibrations ('presystolic murmur') which occur in sinus rhythm or atrial fibrillation with mitral stenosis are the same phonocardiographic phenomenon. The exact cause of the sound is obscure in view of the lack of correlation with echocardiographically detectable wall movement. It could be that 'M', like the third heart sound, emanates from the ventricular wall and is produced by systolic tautening and decreased compliance of the left ventricle as diastolic filling is terminated. While the origin of the sound is uncertain, we consider that 'M' constitutes that portion of the 'presystolic' murmur which occurs during isovolumic systole in mitral stenosis.

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