Echocardiographic studies of genesis of mitral diastolic murmurs

Nicholas J. Fortuin and Ernest Craig

From the Biochemical Research Branch, Division of Effects Research of the Environmental Protection Agency and the C. V. Richardson Laboratory of the Department of Medicine, University of North Carolina, Chapel Hill, North Carolina, U.S.A.

In order to determine the relation of mitral valve motion to the presystolic murmur of mitral stenosis and the mid-diastolic rumble accompanying mitral regurgitation or large left-to-right shunts, we used the combined techniques of mitral valve echocardiography, phonocardiography, and apex cardiography in 32 patients. In patients with mitral stenosis, in sinus rhythm or atrial fibrillation, the crescendo presystolic murmur occurred simultaneously with closing movements of the mitral valve. In these patients, mitral valve closure was effected by ventricular systole. The mid-diastolic rumble of mitral regurgitation occurred after the period of peak left ventricular filling at a time when the mitral valve was closing rapidly. The temporal relation of valve motion to cardiac murmurs suggests that a closing valve in the face of persistent antegrade flow may be of aetiological importance in the genesis of these murmurs through the production of turbulence, increased blood flow velocity, vortex shedding, or other mechanisms.

Recent studies using the techniques of cineangiography and echocardiography have described the relation of mitral valve motion to certain diastolic cardiac murmurs and have challenged traditional concepts of the aetiology and genesis of these murmurs (Criley, Feldman, and Meredith, 1971; Criley and Hermer, 1971; Fortuin and Craig, 1972). By meticulous analysis of cineangiograms, Criley has shown that the crescendo presystolic murmur of mitral stenosis, formerly thought to be due to atrial systole, is temporally related to closing movements of the mitral valve which occur after atrial systole has spent its force. He has postulated that this murmur occurs as a consequence of progressive narrowing of the orifice which results in increasing flow velocity. We have recently suggested a similar mechanism for production of the Austin Flint rumble associated with aortic regurgitation, based on observations of mitral valve motion obtained by echocardiography in conjunction with phonocardiography (Fortuin and Craig, 1972). In the studies reported here, we have used the combined techniques of phonocardiography, mitral valve echocardiography, and apex cardiography to investigate further the relation of valve motion to production of the presystolic murmur in patients with mitral stenosis with sinus rhythm or atrial fibrillation and the mid-diastolic rumble accompanying pure mitral regurgitation or large left-to-right shunts.

Patient selection and methods

Thirty-two patients were studied. All were attending the Cardiac Clinic or were inpatients at North Carolina Memorial Hospital. Twenty patients had mitral stenosis with apical diastolic murmurs recorded by phonocardiography: 10 of these were in normal sinus rhythm and 10 in atrial fibrillation. All had typical physical findings of mitral stenosis, and in all mitral valve echocardiography revealed a diastolic (E to F) slope of less than 30 mm/second. Nine patients had pure mitral regurgitation with recordable mid-diastolic apical rumbling murmurs introduced by a third heart sound. Associated stenosis of the mitral valve was excluded in this group by the mitral valve echogram, which in all cases showed an E to F slope in excess of normal (Edler, 1967). In 3 patients, a large left-to-right shunt was demonstrated by cardiac catheterization; two had ventricular septal defects and one had a sinus of Valsalva aneurysm which had ruptured into the right ventricle. Each of these had an apical mid-diastolic rumbling murmur recorded by phonocardiography.

Received 3 July 1972.

1 This study was supported by Contract RR-46 from the National Institutes of Health and by the Henry A. Foscue Professorship at the University of North Carolina.

2 Present address: Department of Medicine, Johns Hopkins University School of Medicine, Baltimore, Maryland 21205, U.S.A.
Phonocardiograms were recorded on a Cambridge multichannel recorder using Cambridge (Leatham) microphones with variable filters. Apex cardiograms were obtained simultaneously using a Hellige pulse transducer, with a time constant of the transducer-recorder in excess of 3 seconds. Mitral valve echocardiograms were obtained with an Ekoline 20 ultrasonoscope with a 2 cm transducer. An electronic gating circuit was employed to transmit the isolated valve echogram to the multichannel recorder (Effert, Hertz, and Bohme, 1959).

Results

Fig. 1 illustrates the relation of mitral valve motion to electrocardiographic and phonocardiographic events of the cardiac cycle and praecordial movement in a normal subject. Shortly after the second heart sound the mitral valve abruptly begins to open, rapidly reaches its peak opening movement, immediately begins to close, reaches a stable point of near closure, and is reopened again after the onset of atrial systole. The valve comes to a near-closed position as a result of atrial systole alone and before the onset of ventricular systole which is identified by the upstroke of the systolic phase of the apex cardiogram.

In Fig. 2, the characteristic valve echo abnormalities seen in patients with mitral stenosis in normal sinus rhythm are illustrated together with a phonocardiogram. An opening snap occurs simultaneously with the peak of valve opening. The valve does not close rapidly in early or mid-diastole as in the normal situation, but is held in an open position throughout diastole. Atrial systole results in a very small anterior movement of the mitral valve followed by a small movement toward closure, but completion of valve closure does not occur until after the Q wave of the electrocardiogram or after the onset of isometric systole as marked by the simultaneously recorded apex cardiogram in Fig. 3. The presystolic murmur recorded at the cardiac apex has its onset well after the P wave of the electrocardiogram. It begins to manifest a typical crescendo as the mitral valve is closing rapidly after the anterior movement of the valve produced by atrial systole has occurred. The murmur ends in a loud first heart sound at the time of mitral valve closure. We observed this relation of the crescendo presystolic murmur to the closing movement of the mitral valve in all the patients with mitral stenosis in sinus rhythm. The relation of the apex cardiogram to the movements of the mitral valve is shown in Fig. 3. Here it can be seen that valve closure is initiated by atrial systole. The valve, however, is still in a relatively wide open position at the onset of ventricular systole, and it is that event which effects closure.

Of the 10 patients with mitral stenosis and atrial fibrillation, 5 had an intermittent presystolic murmur. An example together with a valve echogram is shown in Fig. 4. The presystolic murmur is seen in conjunction with the shorter cardiac cycles. With longer cycles, as the last complex, no presystolic murmur is recorded. As in the case with normal sinus rhythm, the presystolic murmur in this patient

---

**Fig. 1** Multichannel graphics record from a normal subject to illustrate relation of heart sounds to mitral valve echocardiogram (mitral echo) and apex cardiogram (apex). Note that mitral valve closure is almost completed at the time of the systolic upstroke of the apex cardiogram. PCG = phonocardiogram; PA = pulmonary area; 2 = second heart sound.
Echocardiographic studies of genesis of mitral diastolic murmurs

FIG. 2 Record obtained from a patient with mitral stenosis and 'atrial systolic murmur' (ASM). Note the characteristic deformity of the valve echogram of mitral stenosis. The ASM assumes a crescendo character coincident with closing movement of the mitral valve. AA = aortic area; MA = mitral area; OS = opening snap; SM = systolic murmur; carotid = carotid pulse tracing.

FIG. 3 The same patient as in Fig. 2. The apex cardiogram is included to allow precise timing of the onset of ventricular systole, as signified by the upstroke of the systolic movement. Note that the mitral valve is in a relatively wide open position at the onset of ventricular contraction. F = filling wave.

also occurs at the same time that the mitral valve is closing rapidly.

Examples of the relation of mid-diastolic rumbles to mitral valve motion in patients with pure mitral regurgitation are presented in Fig. 5 and 6. In this situation the opening of the valve may be accompanied by a soft opening snap (Fig. 5). A third heart sound follows 40-60 msec after the peak of valve opening and introduces a rumbling murmur. The pattern of valve motion resembles the normal situation except that the early diastolic closing velocity is more rapid than normal. The mid-diastolic murmur occurs as the valve is closing rapidly and disappears as the valve maintains a semiclosed position for the duration of diastole. Both of the patients illustrated had atrial fibrillation, and in each the valve closed after the Q wave of the electrocardiogram. The temporal relation of the valve closing
movement to the mid-diastolic rumble illustrated in Fig. 5 and 6 was observed in all patients with mitral regurgitation or left-to-right shunts. The velocity of valve closure during mid-diastole was in all cases in excess of normal (>150 mm/sec).

**Discussion**

The studies reported here support the view of Criley and others (Criley et al., 1971; Criley and Hermer, 1971; Constant, 1969) that the crescendo presystolic murmur observed in patients with mitral stenosis is not due to atrial systole, as has been widely accepted for many years (Gairdner, 1861; McKusick, 1958). Our studies of mitral valve motion by echocardiography are in close agreement with those of Criley who used cineangiographic techniques (Criley et al., 1971; Criley and Hermer, 1971). By both methods the presystolic murmur has been shown to increase in a crescendo after the peak of atrial systole but at a time when the mitral valve is closing rapidly. Since the gradient between left atrium and left ventricle is decreasing at the time the murmur is increasing, it is unlikely that increasing flow across the valve can be invoked as an explanation for the murmur. Criley has hypothesized that the velocity of blood flow across the valve...
Echocardiographic studies of genesis of mitral diastolic murmurs

Fig. 6 Mitral regurgitation and a mid-diastolic rumble. The rumble occurs after the third heart sound as the mitral valve is closing rapidly.

may be increasing because the orifice of the mitral valve is diminishing more rapidly than flow through the orifice. The increasing intensity of the murmur may therefore be related to the increased blood flow velocity. Alternatively, the moving valve in the face of continued antegrade flow may provide a mechanism for vortex shedding and murmur production by this mechanism.

That atrial systole is not necessary for production of the crescendo presystolic murmur in mitral stenosis is illustrated by the patient in atrial fibrillation in Fig. 4 and those recently studied by Criley and Hermer (1971). In our patient the crescendo murmur was present with short cardiac cycles, and disappeared with longer cycles. In Criley's patients the gradient between left atrium and ventricle was present at the end of the shorter cardiac cycles, whereas with longer diastolic intervals the gradient fell to lower levels. Thus, with the shorter cycles mitral valve closure occurred in the face of continued antegrade flow. The turbulence or velocity necessary to generate the requisite acoustic energy for murmur production may not have been present at the lower flow rates at the conclusion of longer cycles.

Our studies illustrate the abnormal mechanism for mitral valve closure which exists in patients with mitral stenosis in normal sinus rhythm. In the normal heart, valve closure occurs largely as a result of atrial systole and is about totally completed at the onset of isometric ventricular systole (Henderson and Johnson, 1912; Little, 1951; Sarnoff, Gilmore, and Mitchell, 1962; Zaky, Steinmetz, and Feigenbaum, 1969). Studies recently reported have shown that the point of initial systolic movement of the apex cardiogram occurs precisely at the onset of left ventricular systole (Willems, De Geest, and Kesteloot, 1971). We have therefore used the apex cardiogram to time the onset of systole in our patients. In patients with mitral stenosis in normal sinus rhythm, valve closure is initiated by atrial systole, but the valve is in a relatively wide open position at the onset of ventricular systole (Fig. 3). Thus, ventricular contraction is largely responsible for closing the valve. Since the crescendo presystolic murmur occurs simultaneously with valve closure, this murmur is more closely related to ventricular systole than to atrial systole.

The mid-diastolic rumble frequently heard in patients with severe mitral regurgitation has been generally attributed to excess flow across the mitral valve (McKusick, 1958). Nixon (1961), however, observed that the rumble occurred after the period of rapid ventricular filling and flow, and suggested that another mechanism may be operative. The relation of diastolic murmur to left ventricular filling is illustrated in Fig. 7. More recent studies have confirmed that the major portion of left ventricular filling occurs very early in patients with mitral regurgitation (Silverman and Fortuin, 1972), before the third heart sound or subsequent murmur. Nixon and Wooler (1963) also found that there was a reproducible pressure gradient in early diastole in patients with pure mitral regurgitation which corresponded temporally with the diastolic murmur. Nixon
suggested that the murmur and gradient might occur because the mitral valve was drawn into a closed or semiclosed position as the left ventricle filled. Earlier workers had also proposed mid-diastolic closure of the mitral valve and suggested a relation to the production of a third heart sound (Gibson, 1907; Hirschfelder, 1907; Dock, Grandell, and Taubman, 1955). Our studies support Nixon's hypothesis and show that the mitral valve is closing very rapidly at the time of the mid-diastolic murmur. We would postulate that because of its large extra volume, the left atrium cannot empty completely in early diastole. Thus, antegrade flow continues across the mitral valve as the valve is closing rapidly. This closing valve structure in the face of continued antegrade flow may result in increased velocity of flow, turbulence, or a mechanism for vortex shedding. By any of these, or perhaps other mechanisms, a rumbling murmur is produced. A similar explanation can be advanced for the mid-diastolic murmur accompanying left-to-right shunts and is probably also responsible for diastolic murmur production in other high flow states such as anaemia, or thyrotoxicosis, or the right-sided rumble accompanying atrial septal defect.

Our studies, and those of Criley, have shown only a temporal relation between these diastolic murmurs and movements of the mitral valve. A cause and effect relation is only hypothesized and has not been proven. None the less, the observations reported here and those concerning the Austin Flint murmur reported previously (Fortuin and Craigie, 1972) do permit a unitary explanation for diastolic rumbling murmurs which have very similar acoustic characteristics but accompany different cardiac abnormalities. In each of these conditions, i.e. mitral stenosis with crescendo presystolic murmur, aortic regurgitation with Austin Flint murmur, mitral regurgitation or left-to-right shunt with mid-diastolic murmur, there is reason to suspect that the left atrium does not empty in a normal fashion. In mitral stenosis this is the result of the narrowed orifice size; in aortic regurgitation, it is because of left ventricular filling from two sources and too rapid closure of the mitral valve, and in mitral regurgitation or left-to-right shunt, because of increased left atrial volume and rapid mitral valve closure. In each condition, antegrade flow persists as the mitral valve is closing rapidly and a murmur with low frequency characteristics is produced.

The authors wish to acknowledge the expert technical assistance of Mrs. Sally Moos.

References
Echocardiographic studies of genesis of mitral diastolic murmurs


Requests for reprints to Dr. N. J. Fortuin, Cardiovascular Division, Department of Medicine, The Johns Hopkins University School of Medicine, The Johns Hopkins Hospital, Baltimore, Maryland 21205, U.S.A.