Mitral Insufficiency: Cardiac Mechanics as Studied with the Kinetocardiogram and Ballistocardiogram

By William T. Tucker, M.D., John L. Knowles, M.S. and E. E. Eddleman, Jr., M.D.

Precordial movements (kinetocardiograms) and ballistocardiograms were studied in patients with “pure” mitral insufficiency. Certain characteristic features of the records were noted and contrasted with those found in patients with mitral stenosis. The finding suggests fundamental differences in the mode of ventricular contraction and relaxation in mitral insufficiency and mitral stenosis.

A PREVIOUS paper has defined precordial chest-wall movements (kinetocardiograms) in patients with mitral stenosis.1 The present study was undertaken in an effort to ascertain the precordial chest-wall movements in patients with mitral insufficiency, and to compare these with the kinetocardiograms from patients with mitral stenosis.

TECHNICS

Precordial chest-wall movements were obtained by methods previously described.2 The apparatus consists of a bellows-air-conduction system connected to a piezoelectric transducer and recorded on a Sanborn Poly-viso four-channel recorder. A detailed analysis of the normal patterns obtained by such technics has been previously presented.3, 4 Direct ballistocardiograms were taken simultaneously with patients on a sand or putty surface to minimize body oscillations.5, 6 Electrocardiograms and carotid pulses were obtained simultaneously with the kinetocardiograms and ballistocardiograms. Kinetocardiograms were recorded from the precordium in the areas corresponding to the V leads of the electrocardiogram and are designated as KV1, KV2 etc.

PATIENTS

The validity of such a study as this depends upon an accurate diagnosis of the cardiac valvular lesion. In order to evaluate the precordial movements, it seemed desirable to study first only patients in which the diagnosis of “pure” mitral insufficiency was reasonably certain.

There is no uniformity as to what physical signs constitute the most reliable clinical evidence for mitral insufficiency. However, the following criteria appeared most reasonable and consistent with the findings presented by others for mitral insufficiency.7, 8, 9, 10 (1) an apical systolic murmur transmitted to the axilla of grade III or more intensity, (2) absence of a diastolic murmur, (3) history of rheumatic fever, (4) no evidence of any other valvular involvement, (5) left ventricular predominance either by fluoroscopy or by the electrocardiogram, and (6) some clinical evidence of heart disease. Not all of these patients met all of the criteria above. However, 11 patients were selected in which the diagnosis appeared reasonably certain clinically, and in whom the majority of the above findings were noted. The essential clinical features of these patients are listed in table 1. Patient 8 had the presence of a grade I diastolic rumble at the apex and initially was considered to have mitral stenosis as well as mitral insufficiency. However, operation revealed only a large dilated mitral valve with insufficiency and no detectable stenosis. Although it was possible that there was still minimal mitral stenosis present in this patient, mitral insufficiency was certainly the major valvular lesion, and it was felt justifiable to include this patient in the study. Patient 10 did not have a history of rheumatic fever, but the apical murmur was loud and harsh and clinically consistent with the diagnosis of mitral insufficiency. He did have a minimal elevation of blood pressure, but it was not considered of sufficient degree to exclude this patient from the series. In addition, cardiac catheterization studies in this patient revealed no evidence of a congenital defect. Two of the patients (patients 4 and 5) had loud apical systolic murmurs which were musical in quality. Patients 1, 2, 3, 5 and 11 (table 1) had late systolic crescendo murmurs, while patients 8, 9 and 10 had murmurs which extended throughout systole. Figure 1 is an illustration of the phonocardiogram from patient 4 in whom there was a musical quality to the systolic murmur.
<table>
<thead>
<tr>
<th>Patient</th>
<th>History of Rheumatic Fever</th>
<th>Systolic Apical Murmur</th>
<th>Third Heart Sound</th>
<th>Fluoroscopic Examination</th>
<th>Electrocardiogram</th>
<th>Functional Class</th>
<th>Miscellaneous</th>
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<td>Yes</td>
<td>IV</td>
<td>Late</td>
<td>No</td>
<td>Minimal L.V.E.</td>
<td>55° No</td>
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<td></td>
<td></td>
<td></td>
<td>Large pulsating left auricle</td>
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<td>-5° Yes</td>
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<td>-5° Yes</td>
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<td>60° No</td>
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<td>8</td>
<td>Yes</td>
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<td>Minimal L.V.E.</td>
<td>60° Yes</td>
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<td>11</td>
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<td>No</td>
<td>Minimal L.V.E.</td>
<td>60° Strain</td>
<td>I</td>
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</table>

The functional class is based on the standards of the American Heart Association. L.V.E. indicates left ventricular enlargement. The grade of the murmur is according to the classification of Levine.

Fig. 1. The phonocardiogram taken from patient 4 with "pure" mitral insufficiency in which there was a musical quality to the murmur. Note the high-pitch vibrations occurring in midsystole which are of large amplitude.
**Significant Features of the Records from Patients with “Pure” Mitral Insufficiency**

Figure 2 is a drawing of the typical findings of the kinetocardiograms in patients with mitral insufficiency. The typical findings from patients with mitral stenosis are included for contrast as well as those from normal subjects. Figure 3 is a representative tracing from patient 1. Figure 2 is best employed in following the subsequent discussion. It is obvious from figure 2 that the traces from subjects with mitral insufficiency are quite different from those taken from patients with mitral stenosis and from normal subjects. The differences from normal subjects will be presented first. 

**Traces From the Right Parasternal Region of the Chest (KV1)**

Period from the Onset of the QRS Complex to the Carotid Upstroke. Patients with mitral insufficiency usually have a double outward movement in the KV1 area not too different in appearance from that of normal subjects. The onset of the initial outward movement which begins in normal subjects approximately .02 second after the onset of the QRS complex, when present in patients with mitral insufficiency, usually occurs in the normal time rela-

The patient with mitral stenosis. In the KV1 area, the trace taken from a patient with mitral stenosis differs more from that of a normal person, while the trace from the apical region (KV4) differs more from the normal pattern in patients with mitral insufficiency. Note the late systolic outward movement in the region of the apex in patients with mitral insufficiency in contrast to the absence of this movement in a trace from normal persons and from a patient with mitral stenosis. The drawing is best understood by referring to the text in which the motions are broken down according to time periods from the onset of the QRS complex to carotid upstroke and from the carotid upstroke to the carotid incisural notch.

Note that the early footward movement in the ballistocardiogram from a patient with mitral stenosis is absent in the traces of a normal subject and of a patient with mitral insufficiency. Also note that in a patient with mitral stenosis there is a sharp footward movement .04 second after the onset of the QRS complex which is absent in patients with mitral insufficiency. The GH upstrokes in the ballistocardiograms are delayed in mitral stenosis and mitral insufficiency as compared with that in normal traces. Patients with mitral insufficiency again may have a definite notch in the HI downstroke as illustrated.
tionship. However, the peak of the second outward movement is usually delayed when compared with this finding in the normal subjects by approximately .02 to .04 second duration. The parasternal region then begins moving inward approximately .02 second before the onset of ejection as determined by the carotid upstroke.

*Period from the Onset of the Carotid Upstroke to the Carotid Incisural Notch.* As mentioned, the right parasternal region usually begins moving inward before the carotid upstroke. However, the main inward retraction of the parasternal region of the chest takes place during the early portion of ejection. The depth of this systolic retraction goes well below the baseline and is not dissimilar in time or configuration to that noted in normal subjects. It usually reaches its maximal retraction approximately .04 to .06 second after the I point in the ballistocardiogram or during the IJ upstroke. The chest then begins to move outward during the period of mid and late portion of ejection, going above the diastolic or initial resting level of the chest. The outward peak of this motion appears well after the J point in the ballistocardiogram and, therefore, does not correspond in time with the J point as does the midsystolic outward movement E₁-E₂ in normal subjects (fig. 2). The trace also shows a notch in late systole but continues to move outward up to or slightly after the time of the carotid incisural notch. This point, which corresponds in time to the carotid incisural notch, was in all instances above the diastolic or resting level of the chest.

*Period of Diastole Beginning at the Carotid Incisural Notch:* The parasternal region of the chest (KV₁) again moves inward, usually not to the same degree as that noted during the retraction of the chest during early ejection. However, it is a well-defined movement and its nadir occurs after the corresponding point in the normal trace (approximately .08 second). In one instance, it was as much as .20 second after the carotid incisural notch. At this time, the chest moves outward in a slow fashion, reaching the diastolic or resting level of the chest before the next onset of the QRS complex.

**Apical Region of the Chest (KV₄)**

*Period from the Onset of the QRS Complex to the Carotid Upstroke.* In most instances the apical region of the chest begins moving outward similarly to the movement in normal subjects, starting .02 second after the onset of the QRS complex. However, the peak of this outward motion is delayed .04 second as compared with the time of this movement in normal subjects. This is followed immediately by a very sharp retraction of the apical region of the chest before the onset of the carotid upstroke, the retraction reaching its nadir before the carotid upstroke begins. In some instances, this retraction of the apical region of the chest is most evident in the KV₄ region rather than in KV₁. It usually parallels the second outward movement in the parasternal region of the chest. Only one patient did not have an appreciable retraction during this period (patient 6). However, a well-defined inward
notch was present, corresponding in time to that noted in the other patients. This retraction of the apical region of the chest is then followed by an outward movement which begins just before the upstroke of the carotid trace and continues on into ejection.

**Period from the Carotid Upstroke to the Carotid Incisural Notch.** As mentioned above, the outward movement of the apical region of the chest begins approximately .02 second before the upstroke of the carotid tracing and continues for approximately .02 to .04 second after the onset of the carotid upstroke, the peak being delayed as compared with the occurrence of this movement in normal subjects (fig. 2). At this time, in early ejection, there is in some instances a small inward motion of the chest, but never the systolic retraction of the chest seen in normal subjects (fig. 2). This inward movement during the early phase of ejection is of brief duration and immediately the apical region of the chest begins moving outward again at this time, going well above the diastolic or resting level of the chest. This outward motion usually reaches its peak before the carotid incisural notch by .04 to .08 second, at which time it begins moving inward. This late systolic anterior or outward movement of the chest is quite pronounced in all of the records, displacing the chest wall out well above the diastolic or resting level of the chest.

**Period of Diastole Beginning with the Carotid Incisural Notch.** The inward motion of the apical region of the chest, which begins shortly before the carotid incisural notch, is a well-marked movement. It terminates usually .12 second after the carotid incisural notch, following which there is a slow outward movement of the chest wall. This outward movement in the apical region precedes by .04 second the outward movement noted in the parasternal region of the chest (KV₂).

The records obtained from the KV₂ and KV₃ areas usually are of a transitional quality between those observed in the KV₄ and KV₁ areas. However, as some of the movements just described in the KV₄ area occasionally are more apparent in the KV₃ area, it is considered that for full evaluation of the traces, records must be taken from each position. In addition, there are noted in most instances small outward and inward movements following the onset of the P waves in the electrocardiogram which are presumed to be of auricular origin. However, these were not characteristic nor significantly altered from those noted in normal subjects, and therefore will not be discussed.

**Ballistocardiograms in Patients with “Pure” Mitral Insufficiency**

There were no consistent findings in the ballistocardiograms in these patients with mitral insufficiency. However, frequently alterations were noted in the initial portion of the ballistocardiogram which were different from those recorded in normal subjects and in patients with mitral stenosis (fig. 2). None of the patients with mitral insufficiency had a marked footward movement (FG downstroke) .04 second after the onset of the QRS complex, as was usually found in patients with mitral stenosis (fig. 2). The GH upstroke in most instances was delayed when compared with that of normal subjects and similar in time to that observed in patients with mitral stenosis. In three instances it was prominent and similar to that reported by Kuo and Schnabel.¹¹

The HI downstroke in most instances was noted in patients with mitral insufficiency, which is more marked than that observed in patients with mitral stenosis or in normal subjects. The diastolic portion of the ballistocardiogram in some instances was altered from that noted in normal subjects; however, there were no consistent features. Three of the patients had deep K points and large KL upstrokes (fig. 1). In others, this portion of the ballistocardiogram was not significantly altered.

**Differences in Traces from Patients with Mitral Insufficiency from those of Mitral Stenosis**

From figure 2, the differences in traces from patients with mitral stenosis and mitral insufficiency are quite striking. The following significant differences are noted:

1) Patients with mitral stenosis have a marked outward movement of the precordium,
usually in the parasternal region, beginning .04 second after the onset of the QRS complex, while patients with mitral insufficiency have a normal or small outward motion .02 second after the onset of the QRS complex.

(2) Patients with mitral stenosis have an inward motion over the right parasternal area of the chest just preceding and during early ejection which rarely retracts below the diastolic level of the chest, in contrast to the well-defined and deep inward movement noted in patients with mitral insufficiency.

(3) Patients with mitral stenosis have a marked anterior displacement of the precordium usually during early and midsystole, in contrast to the marked anterior displacement of the precordium in late systole in patients with mitral insufficiency.

(4) During the ejection period, patients with mitral stenosis usually retract below the diastolic or resting level of the chest in the KV₄ area during the entire ejection phase, in contrast to a marked anterior and outward displacement of the precordium in patients with mitral insufficiency which becomes maximal before the time of the carotid incisural notch.

In summary, patients with mitral insufficiency lack the anterior swing of the heart which occurs .04 second after the onset of the QRS and which is considered to be due to the hypertrophied right ventricle of patients with mitral stenosis.¹ Patients with mitral insufficiency, instead of having an early or mid-systolic anterior swing of the heart, apparently have a late systolic anterior swing of the heart lasting up to the time of the carotid incisural notch.

**Studies of Patients with Combination of Mitral Stenosis and Mitral Insufficiency**

Only preliminary observations can be made on the characteristics of the records of patients with both mitral stenosis and mitral insufficiency. Observations on eight patients who had, in addition to a well-defined diastolic rumble, a loud systolic apical murmur revealed a combination of the features described for both mitral stenosis and mitral insufficiency. Figure 4 is a record of a patient with both mitral stenosis and mitral insufficiency. Note the exaggerated anterior swing of the heart as well as the presence of a generalized precordial late-systolic anterior motion of the chest. From such limited observations it is uncertain at present whether the degree of the various lesions as reflected in the kinetocardiogram will represent a method of determining the predominance of either mitral stenosis or mitral insufficiency.

**Comments**

The variations in the precordial movements noted in patients with mitral insufficiency, from
both normal subjects and patients with mitral stenosis, suggest fundamental differences in the mode of contraction and relaxation of the ventricles. The mechanism for each of the movements is not at the present time clearly understood; however, there are certain features which were observed and which may be pointed out. The initial pre-ejection period, as observed in patients with mitral insufficiency, was not too different from that of normal persons, and lacked the marked outward movement in early systole found in patients with mitral stenosis (fig. 2). There was a slight delay in onset of the initial outward movement of the entire precordium and the subsequent retraction in the region of the apex (fig. 2). The delay in onset of the retraction may possibly be due to a late arrival of the excitation wave as a result of ventricular hypertrophy or an increase in the electromechanical lag. The retraction was exaggerated in amplitude over normal in all but one instance, in which it was represented only by an inward notch. This patient had the largest heart of any one of the series and the poorest functional capacity. Thus it is possible that the retraction of the apex was absent because of changes in the contraction process as a result of ventricular dilatation. The exaggeration of the apical retraction may be explained as being the result of a more forceful shortening process of the heart. Why this should occur is not apparent at this time.

The late systolic outward or anterior movement of the precordium is one of the most significant features in the records obtained from patients with mitral insufficiency (fig. 2). At the present time there is no objective evidence as to the actual mechanism of this late outward systolic movement of the precordium, but its presence from the apical region to the KV1 site suggests that it is due to an anterior movement of the heart at this time. A possible mechanism may be that of an anterior displacement of the heart as a result of auricular enlargement by the blood being ejected posteriorly and headward through the insufficient mitral valve during systole. The left auricular pressure curves in mitral insufficiency as well as piezoeosaphagographic observations and electrokymographic traces from the left auricular border are compatible with this hypothesis in that they all demonstrate an outward movement in systole, the movement being greater just before the end of ejection. In addition, the work of Wiggers has shown that regurgitation of blood from the ventricles into the auricles probably takes place during late systole and even into the isometric relaxation phase. The late systolic murmur correlates well with this outward movement of the precordium and the time of regurgitation; however, it was present even in those patients with early systolic murmur. Other factors in the genesis of the movement, such as elongation and inflow tract relaxation, have not been excluded.

It is likely that these changes in the precordial movements occur only after some cardiac impairment has taken place. This is exemplified by two patients not included in this series who had grade I to II systolic apical murmurs and a definite history of rheumatic fever. Even though the murmurs were faint and not comparable with those of the patients presented in this study, there was apparently organic mitral insufficiency. Neither of these two patients had fluoroscopic, clinical or electrocardiographic evidence of any functional impairment or enlargement of the heart. Thus alterations in the kinetocardiographic patterns probably depend upon the development of alterations in the contractile processes, and, therefore, minimal lesions do not change the precordial movements. Early and small degrees of mitral insufficiency probably cannot be recognized by kinetocardiographic traces. Thus differential diagnosis between functional and organic murmurs in early stages can probably not be made. This, in itself, does not necessarily limit the potential value of the kinetocardiographic tracings in that it may offer a means to recognize early ventricular functional impairment in mitral insufficiency.

In contrast to the rather consistent findings in the kinetocardiograms of patients with mitral insufficiency, the ballistocardiograms do not appear to be consistent. The pre-ejection portion was more reliable than the diastolic portion. However, exceptions to the pattern and configuration of this early portion of the
ballistocardiogram were encountered. Again caution must be used in interpreting the precordial movements in patients with combined mitral insufficiency and mitral stenosis until a sufficient number of patients have been studied in whom accurate evaluation of the degree of mitral insufficiency and mitral stenosis has been made.

SUMMARY AND CONCLUSIONS

(1) Eleven patients with presumably "pure" mitral insufficiency were studied by the ballistocardiographic and kinetocardiographic techniques.

(2) Certain characteristics of the motions were noted: (a) An exaggerated early systolic retraction in the left precordium. (b) A marked anterior movement of the entire precordium during mid and late systole reaching a peak approximately at the time of the carotid incisural notch.

(3) It is pointed out that records of patients with mitral insufficiency differ from those of patients with mitral stenosis. However, it is uncertain at the present time, whether the kinetocardiogram can furnish an accurate guide to the relative degree of each.

SUMARIO IN INTERLINGUA

(1) Esseva studiate per medio de technicas ballistocardiographic e cinetocardiographic 11 patientes con insufficientia mitral de forma presumitemente "pur."

(2) Certe caracteristicas del motion esseva notate: (a) Un exaggerate retraction in le precordio sinistre al initio del systole. (b) Un marcate movimento del integre precordio verso le anterior durante le phases medie e final de systole. Iste movimento esseva maximal approximativamente al tempore del incisura carotide.

(3) Nos signala que registrazione ab patientes con insufficientia mitral differe ab illos obtenite ab patientes con stenosis mitral. Sed a iste tempore il non es certe que le cinetocardiogramma pote revelar le grado relative de o insufficientia o stenosis mitral.

REFERENCES


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