The Dynamics of Ventricular Contraction and Relaxation in Patients With Mitral Stenosis as Studied by the Kinetocardiogram and Ballistocardiogram

By E. E. Edleman, Jr., M.D., Robert H. Yoe, M.D., William T. Tucker, M.D., John L. Knowles, M.S. and Kathryn Willis, M.D.

Kinetocardiograms (precordial movements) and ballistocardiograms were studied in patients with "pure" mitral stenosis. The findings were correlated with catheterization data and clinical findings. Certain characteristics of the kinetocardiograms were presented as well as hypothesis for the movements of the heart and dynamics of ventricular contraction and relaxation in patients with mitral stenosis.

The purpose of this communication is to present an analysis of the precordial movements (kinetocardiograms) and ballistocardiograms in patients with mitral stenosis. The relationship of these events to the movements of the heart and the dynamics of ventricular contraction will be discussed.

Methods

The kinetocardiograms were obtained by the use of a bellows connected by air conduction to a piezoelectric transducer. The pickup arm on the bellows was perpendicular to the chest wall; thus absolute movements of the chest wall could be determined from any point desired. The normal patterns, calibrations, and distribution of forces over the chest have previously been presented. Records were obtained from positions corresponding to electrocardiographic "V" leads and designated as KV, KV2, etc. The kinetocardiograms have been shown to reflect primarily the movements of the heart and are not posteroanterior ballistocardiograms. Volume changes within the chest cavity also probably influence the records during ejection.

Direct displacement ballistocardiograms were recorded with the patient on a sand or putty surface to minimize oscillatory movements. The direct displacement ballistocardiograms as recorded are probably not linear, and, therefore, the amplitudes of the movements are not necessarily proportional to the cardiovascular forces involved. However, time relationships and configuration are reasonably constant. Therefore, the ballistocardiograms are employed principally to indicate the association of precordial movements with headward and footward forces. Carotid pulses, electrocardiograms, kinetocardiograms, and ballistocardiograms were recorded simultaneously with either a Sanborn Poly-Viso or a Cambridge Simpli-Scribe four-channel recorder. Cardiac catheterization data on six of the patients were available through the courtesy of Dr. R. J. Bing.

Patients

The patients selected for this study were those in whom the diagnosis of mitral stenosis without insufficiency was reasonably certain. Table 1 lists the data on the 13 patients selected. In 10 of the 13 patients the clinical opinion of "only" mitral stenosis was confirmed at operation, in that the surgeon did not feel a "jet" of blood during systole. The other three patients included in this study had typical presystolic crescendo murmurs without the presence of a systolic murmur. All patients who had any evidence of other valvular involvement or in whom the diagnosis of "pure" mitral stenosis was uncertain were excluded. Clinical data on the 13 patients in this study are listed in Table 1. Note that the patients had very little difference in their functional impairment. Only two of the patients were not taking digitalis; however, the three patients not operated on were in a somewhat better functional state than those who had the commissurotomy. Postoperative findings will be discussed in a subsequent communication.
RESULTS

Electrocardiograms

Typical electrocardiographic patterns of right ventricular hypertrophy were noted in only 4 of the 13 subjects. The changes were those of right axis deviation; a QR complex in V1, and a prominent R in aVR. The mean QRS axis varied from 62 to 122 degrees. The majority of the patients had very small R waves in aVR, with the presence of RS complexes in V1; the R in most instances was less than half the amplitude of the S wave. It is important to note that four patients had an elevation of right ventricular or pulmonary artery pressure in the absence of a typical right ventricular hypertrophy electrocardiographic pattern (table 1). This finding has been noted previously by other investigators.8

Kinetocardiograms

There were three types of variations in the precordial movements noted in the 13 patients studied. However, there were certain similarities in the records from all patients with mitral stenosis. For sake of presentation, the features that were common to all will be presented in the following discussion and illustrated in figure 1.

1. An exaggerated initial outward movement of the chest wall (I-E1) that is associated with an exaggerated footward movement (FG) of the body. Approximately 0.04 second after the onset of the QRS complex of the electrocardiogram there occurs a marked outward movement of the lower chest wall (KV). In normal subjects it begins earlier (approximately 0.02 second after the onset of the QRS complex) and is of smaller amplitude. Simultaneous with this large outward movement of the chest, there is usually a well-marked footward movement of the body (FG) noted in the ballistocardiogram.

2. An inward movement of the precordium (I-E1) associated with a headward movement of the body (GH upstroke). This inward movement parallels the GH movement of the ballistocardiogram and is noted over the entire anterior precordium. The movement usually begins before the carotid upstroke but extends on into the first part of rapid ejection, terminating approximately at the I point of the ballistocardiogram. One significant feature is that the inward movement rarely goes below the diastolic baseline, except in a few instances which will be discussed subsequently. This is in marked contrast

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Table 1.

<table>
<thead>
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<th>Patients</th>
<th>Estimated Size of Valve at Baseline (mm)</th>
<th>Catheterization Data</th>
<th>Electrocardiographic Data</th>
<th>Fluoroscopic Examination</th>
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<td>Cardiac Output (L./Min.)</td>
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* The patients indicated with no F-G downstroke did have a small notch present corresponding in time to the marked F-G downstroke noted in the other patients.

† Classification of cardiac function is based on that of the American Heart Association.
3. The apex thrust. The apex thrust (I_2-I_3) at KV is conspicuously small and on most records is difficult to recognize. Assuming that in patients with mitral stenosis it has approximately the same time relationship to the carotid upstroke as in normal subjects (0.04 to 0.02 second before the carotid upstroke), a small outward movement can be noted over the region of the apex at this time. Since in some instances the anterior swing is directed leftward, there may be an exaggerated outward movement (0.04 second after the QRS complex) which can be confused with the apex thrust. The true apex thrust occurs later (just before the carotid upstroke) and is small or obscured.

4. A prominent outward motion of the chest (E_1-E_2) paralleling the II upstroke in the ballistocardiogram. The systolic outward movement of the chest wall (E_1-E_2) which parallels the II upstroke of the ballistocardiogram is frequently prominent and displaces the anterior chest even further outward during ejection. The chest usually begins moving inward with the JK downstroke; however, the entire parasternal region of the chest is usually displaced outward and above the diastolic or resting level during the entire period.

5. An exaggerated inward movement which begins with JK downstroke and extends into isometric relaxation (D_3 point). There is a large inward movement of the chest beginning in most subjects during late systole and extending into isometric diastole. This inward movement is usually pronounced and as a result the chest wall retracts below the diastolic or resting level.

In summary, all patients exhibited an exaggerated but delayed outward motion of the chest 0.04 second after the onset of the QRS complex. There is a well-defined inward motion beginning before the carotid upstroke but often extending into the phase of rapid ejection; however, this is not of sufficient magnitude to retract the chest below the diastolic ejection; thus the entire chest during systole is displaced sustained out above the diastolic baseline throughout the ejection period. As right ventricular relaxation occurs the left ventricle pulls the heart posteriorly, producing the inward movement of the precordium which extends into the isometric relaxation phase.
anteriorly above its diastolic level, in contrast to a well-marked inward movement in normal subjects.

Variations in the Records. Although all patients had essentially the same features presented in the previous discussion, notable variations did occur. These may be best explained by dividing the patients into three groups. It is emphasized that this division has been done for the sake of presentation and that there is only meager evidence that the groups have any characteristics which are fundamentally different.

Group I: Four patients are classified in this group (table 1). Figure 2 is a drawing and record of the precordial movements (KV₁ and KV₄) and ballistocardiogram from patient 4. The significant features of the records of these four patients are:

1. The initial outward movement of the chest (I₁-I₂) 0.04 second after the onset of the QRS complex was of greatest magnitude over the right lower chest. The lateral chest (KV₁ and KV₄) moved inward simultaneously with the outward motion noted on the right chest.

2. Electrocardiograms typical of right ventricular hypertrophy were noted in these four patients only.

3. Cardiac catheterization in two of these patients revealed elevated pulmonary artery and right ventricular pressures (table 1); however, patient 5 in another group had comparable elevations in pulmonary artery pressure.

4. Two of the four patients had small outward movement (E₁-E₂) paralleling the I-J upstroke of the ballistocardiogram; nevertheless, the parasternal region of the chest was displaced above its diastolic or resting level during systole.

5. The ballistocardiograms were similar in configuration to those previously reported by others in mitral stenosis⁹⁻¹¹,ⁱ⁹ with a prominent FG downstroke (early marked systolic footward movement). In one subject the FG downstroke was small in amplitude but nevertheless present (patient 1).

Thus the records of these three patients were characterized by a large early anterior movement of the chest wall which was of greatest magnitude over the right lower chest. The chest was sustained out above the diastolic or resting level during systole, in contrast to a well-defined inward movement in normal subjects.

Group II: Figure 3 presents a drawing and record of the kinetocardiograms and ballistocardiograms from patient 8. The four other patients included in this group had similar records (table 1). The variations from those previously presented are:

1. The early systolic anterior movement (I₁-I₂) occurring 0.04 second after the QRS complex was more pronounced over the left lower anterior chest than on the right. It is important to point out that although this outward movement may be of maximal amplitude in the area of the apex (KV₄), it occurs too soon after the onset of the QRS to be the true apex thrust. In a previous study of comparable age groups the apex thrust was found to occur 0.08–0.10 second after the onset of the QRS complex, or about 0.03–0.04 second before the carotid upstroke. Thus this early outward movement is apparently due
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Fig. 3. A drawing of the trace obtained from a patient (Case 8) in group II, in which the anterior or outward movement of the precordium is more pronounced over the left chest than over the right. The arrows on the right-hand figure indicate the onset of the QRS complex. Ce indicates the onset of the carotid upstroke and Cin the carotid incisural notch. The vertical lines represent a time interval of 0.04 second. Electrocardiograms from the patients in group II were not typical of right ventricular hyper trophy and the mean QRS axis was directed more to the left than that noted in the patients in group I. The outward movement of the chest 0.04 second after the onset of the QRS complex is most marked in the KV2 record, indicating the anterior swing of the heart in this instance is directed more to the left than to the right, as noted in group I patients. Although the trace in KV1 dips slightly below the diastolic baseline during early ejection, KV2 and KV3 (not shown) were displaced outward during the entire ejection period, thus lacking the systolic retraction as occurs in normal subject.

to the same mechanisms as the outward movement in the other patients but is directed to the left instead of the right, as noted in group I patients.

2. All five patients had electrocardiograms in which the QRS mean electrical axis was normal (less than 90 degrees). None had typical electrocardiographic record of right ventricular hypertrophy, even in the presence of an elevated mean pulmonary artery and right ventricular pressure (table I).

3. The inward movement of the kinetocardiogram (I-Ia) that parallels the G-H upstroke of the bal listocardiogram moved slightly below the diastolic baseline on the right side of the chest in two patients, but never to the degree noted in normal subjects. Despite the inward movement of the right chest below the resting baseline, the records taken at KV2 and KV3 still exhibited a sustained outward motion above the diastolic baseline during systole.

4. The mean outward movement of the chest (E1-E2) which parallels the IJ upstroke in the bal listocardiogram was more prominent in these five patients than those in either of the other two groups.

5. The precordial movements following the onset of the P wave in the electrocardiogram were occasionally prominent (fig. 3), with the largest movements noted in the records from the left lower chest (KV4). However, other patients with a sinus rhythm did not have exaggerated auricular movements, while still others had large outward movements in KV1 and large inward movements in KV4. Thus the auricular movements in the kinetocardiograms were variable, and are not characteristically altered in patients with mitral stenosis.

In summary, the patients in group II had initial outward movements of the chest directed leftward, in contrast to its more rightward direction in the patients of group I.

Group III: The last four patients listed in table 1 are included in this group. Figure 4 is a drawing and record obtained from patient 12. These four patients differ from the other patients in the following ways:

1. There was a double outward motion occurring early in systole instead of the single outward motion, as noted in the other patients. The first outward movement began 0.04-0.06 second after the onset of the QRS complex with a duration of 0.04 second, as in the previous patients. This was followed by an abrupt inward motion and then a second outward movement beginning 0.10-0.14 second after the onset of the QRS complex. In two patients the two outward motions were separated only by a small notch in the right parasternal records.

2. The ballistocardiograms in patients of this group had more marked variations than in groups I and II. Two of the patients in group III had notched I-J upstrokes, while one had a very atypical pattern.

3. The mean QRS axis was, in general, intermediate between those noted in the first two groups.

4. Two of these patients had commissurotomies; one patient (case 11) had "pure" mitral stenosis while the other patient (case 13) had an infundibular type of stenosis, with no regurgitation being present in either case. Catheterization data in one was not remarkably different from those in the other groups.
I. Exaggerated Anterior Swing of the Heart (I₁-I₅). The initial outward movement of the chest paralleling the F-G footward movement in the ballistocardiogram is exaggerated in patients with mitral stenosis. This movement is considered to be due to an anterior swing of the heart, as is the comparable movement in normal subjects. It occurs about 0.02 second after the QRS complex in normal subjects but is delayed in patients with mitral stenosis (0.04 second after the onset of the QRS complex). Usually, in normal subjects, it is directed to the right; however, in patients with mitral stenosis it may be directed rightward, anteriorly, or slightly leftward. There is possibly a correlation of the direction of the swing to the mean QRS axis; the patients with right axis deviation had a large outward movement to the right while those with a normal axis tended to swing more to the left. Because the right ventricle is hypertrophied, or at least is the dominating ventricular force in mitral stenosis, it is probable that contraction of the right ventricle lifts the heart anteriorly at this time. This is in keeping with the classic concept that the right ventricle is electrically excited before the left. However, the directional variations of this anterior swing are not easily explained. There are several possible explanations inclusive of the following:

(1) The degree of right ventricular hypertrophy may be a factor, since the patients with typical electrocardiographic patterns of right ventricular hypertrophy tend to swing more to the right. Patients with large right ventricles due to congenital heart disease tend to swing to the right also; however, the records resemble more closely the traces from group III with the double anterior movements than those in group I (anterior swing directed to the right). In addition, there were comparable elevations of intracardiac pressures in the various groups. Thus if the degree of hypertrophy is the determining factor, one must postulate that the elevations of intraventricular and pulmonary artery pressures are not reliable indices of right ventricular hypertrophy. Neither fluoroscopic examination nor the clinical status of the patient has been of help in evaluating the degree of right ventricular hypertrophy.

(2) Another possible explanation is that hypertrophy of the right ventricle may be somewhat asymmetric in location. If the hypertrophy is located more on the right aspects or along the right border of the ventricle, the anterior swing could be

In summary, the patients of group III had two anterior movements of the chest occurring early in systole instead of only one, as noted in the other groups.

Interpretation Based on the Movements of the Heart

The kinetocardiograms have been shown to be related primarily to the movements and shape changes of the heart. Thus, by correlating the various events, a scheme of movements of the heart in mitral stenosis can be postulated. The interpretation is based on a comprehensive study of normal subjects, and is offered as a possible explanation for the various events. Figure 1 is best employed to follow the subsequent discussion.
directed rightward as in group I patients. If the hypertrophy is located more on the left aspects, the swing could then be directed leftward as in group II patients.

(3) Alterations in the anatomic arrangement of the muscle fibers could explain the variations in both the kinetocardiograms and the electrocardiograms. Although there is no evidence for this, it can be stated that alignment of the right ventricular fibers in a slightly altered direction would be expected to change the axis of the anterior swing.

(4) The last obvious possible explanation is a variation in the spread of the ventricular excitation wave. Only the variation in the electrocardiograms tends to support this concept.

Preliminary observations seem to suggest that the direction of the initial anterior swing has little effect on the postoperative prognosis. Postoperative findings will be discussed in a subsequent communication.

II. Apparent Right Ventricular Ejection and Inflow Tract Contraction. Following the anterior swing of the heart which produces the initial outward movement of the chest, the parasternal area moves inward (I₁-E₁), associated with the G-H upstroke in the ballistocardiogram (fig. 1). The onset of this inward movement in the KV₁ area may be slightly delayed (0.01-0.02 second), as compared with the sharp inward movement in normal subjects. The inward movement in normal subjects is considered to be the result of right ventricular ejection. Thus in patients with mitral stenosis right ventricular ejection may be delayed, as is the anterior swing.

Shortening of the heart in the base-to-apex diameter is not as clearly defined in these patients with mitral stenosis as in normal subjects; however, the inward movement about this time in the apical records may represent shortening. Thus the G-H upstroke which occurs at this time in the ballistocardiogram may represent shortening and inflow tract contraction, with blood displaced at the outflow tracts, as well as headward motion as the result of right ventricular ejection.

In summary, the inward movement possibly represents right ventricular ejection. Shortening of the heart and inflow tract contraction may be an additional factor in the production of this movement.

III. Apex Thrust (I₁-E₁). The apex thrust was conspicuously small in all patients and in some instances could not be identified. Often at the time of the expected apex thrust (0.08-0.12 second after onset of the QRS complex) a small notch or outward movement could be detected at KV₄. It has been shown that the apex thrust in normal subjects is probably related to a physiologic shape change with contraction of the thickbasilar muscles of the left ventricle, causing a physiologic bulge of the apex. Since the posterior wall of the left ventricle is shortened and atrophied in patients with mitral stenosis, the resulting decreased force of left ventricular contraction could account for the decrease or absence of the apex thrust. An additional factor in the decrease of the apex thrust is the fact that inflow tract contraction and shortening apparently occur late in patients with mitral stenosis, or about the same time as the apex thrust. Thus the inward movement at the onset of apical contraction and the physiologic bulge associated with the contraction of the deep portion of the left ventricle may tend to nullify each other.

IV. Decreased Systolic Retraction of the Chest (Ejection portion of I₂-E₁). The predominating force of the right ventricular contraction producing the anterior swing of the heart, and the decrease in cardiac output in patients with mitral stenosis, are both probable factors in decreasing the inward motion of the lower anterior chest during ejection in patients with mitral stenosis. Thus there is only a small inward movement of the chest which rarely dips below the diastolic baseline during early rapid ejection (only two patients had this inward movement go below the diastolic baseline). In the two exceptions, the swing was primarily to the left, while the inward motion occurred over the right chest. In these two patients KV₂ and KV₃ were nevertheless displaced above the diastolic baseline during ejection.

V. Continuation of Anterior Swing of the Heart (E₁-E₂). The anterior swing of the heart apparently continues during systole and the chest is displaced even further anteriorly. This usually parallels the IJ upstroke and is
probably due to domination of right ventricular swing over ejection.

VI. Leftward and Posterior Swing of the Heart (E2–D1). The chest usually begins moving inward about the time of the JK downstroke but in some instances may be sustained outward until approximately 0.04 second before the carotid incisural notch. The inward motion which is most pronounced in the parasternal region is occasionally associated with a smaller outward motion of the left lateral chest. This probably represents the leftward component of the leftward and posterior swing of the heart. The outward motion occasionally noted in KV4 and KV5, which begins before the carotid incisural notch and usually terminates with the carotid incisura, probably results from two factors: (1) elongation of the heart (opposite of shortening) and (2) the leftward component of the leftward and posterior swing. This outward motion is never very large and is localized almost entirely to the left lateral chest. The inward motion of the right parasternal region usually continued after the carotid incisura, and is probably the result of right ventricular relaxation, which now allows the heart to be pulled leftward and posteriorly in the chest by the still contracting left ventricle. Thus, because of the marked anterior displacement of the heart during systole, this motion is much more exaggerated than in normals.

VII. Diastolic Anterior Movement of the Chest (D1–D2). This last outward movement of the chest during early diastole is most noticeable in records taken from the apical region of the chest. It may represent the onset of filling if a sufficient lag is assumed to occur in the jugular “v” wave. As the lag in the “v” wave is probably variable, this hypothesis could not be established definitely. The time of the outward movement was variable, as measured from the carotid incisural notch. However, the mean value fell within the usual values as occur in normal subjects. There was no constant relationship of this movement to the ballistocardiogram.

Summary of the Movements of the Heart in Patients with Mitral Stenosis. 1. Exaggerated anterior swing of the heart due to predominance of right ventricular contraction over left. 2. Delayed right ventricular ejection and inflow tract contraction with shortening of the heart in the apex-to-base diameter. 3. A diminished apex thrust, and a diminished inward movement of ejection. 4. Continued anterior swing of the heart displacing the chest outward during systole as a result of the dominating contraction of the right ventricle. 5. Exaggerated and early leftward and posterior swing of the heart as a result of relaxation of the right ventricle allowing the contracting left ventricle to pull the heart posteriorly and leftward.

Observations Concerning the Genesis of the Ballistocardiogram in Mitral Stenosis

Certain aspects of this study suggest mechanisms which may be responsible for the alterations in the ballistocardiograms of patients with mitral stenosis. The pre-ejection configuration of the ballistocardiogram in mitral stenosis, as presented in the present report (fig. 1), has been pointed out by others. Some patients with mitral stenosis do not have the deep FG but do show an early small footward movement (fig. 3). This FG pattern has been noted in patients with isolated pulmonic stenosis, indicating that it is certainly not specific for mitral stenosis. The association of this movement with the early marked outward movement of the chest suggests it is the result of right ventricular contraction producing a footward tug on the body. The mechanism by which the right ventricle produces this footward tug is not entirely clear. The right ventricle is attached inferiorly by the right auricle to the inferior vena cava and heavy liver structures, and superiorly to the pulmonary artery and great vessels. It is not apparent through which attachment the forces are mediated. The small headward force (GH) following the FG may be partially due to the same mechanism as in normal subjects, apparently the result of shortening of the ventricles and inflow tract contraction with displacement of blood headward into the ventricular outflow tracts. An additional factor in its mechanism may be right ventricular ejection with headward impact on the pulmonary arteries. The HI downstroke which occurs during the initial phase of ejec-
tion is the result of recoil as in normal subjects, but is of small amplitude, possibly due to decreased cardiac output, decreased acceleration of blood, decreased footward tug by the left ventricle, and headward impact of right ventricular ejection.

The diastolic movements of the ballistocardiogram often have three distinct headward waves present (figs. 1 and 2). These movements observed in early diastole have not been previously pointed out.* Although this part of the ballistocardiogram is somewhat unreliable, due to aftervibrations of the body, the movements often are of equal amplitude and lack the appearance of oscillatory movements. Normal subjects may have these movements in the ballistocardiogram using this recording technic, but these movements are never so distinct nor separated as are the movements noted in most of the patients with mitral stenosis. The separation and exaggeration, therefore, may well be due to the release of the excessive footward tug by the relaxation of the hypertrophied right ventricle, thus producing a distinct movement not normally apparent in the normal ballistocardiogram. Again it must be emphasized that these findings, which occur in the ballistocardiogram, are not specific and have been noted in patients with other conditions.

**Comments**

The kinetocardiograms appear to offer a method by which certain information concerning processes of ventricular contraction and relaxation may be obtained. Thus the development of right ventricular predominance appears to alter significantly the process of contraction and relaxation of the heart with respect to normal subjects, by producing an exaggerated anterior swing during contraction and exaggerated leftward and posterior swing upon its relaxation. Exaggerated anterior swing and the displacement of the chest outward during systole seem to be reliable indices of right ventricular predominance. Similar observations have been noted by Dressler, using other recording technics. It is not clear whether these pulsations represent right ventricular hypertrophy or merely right ventricular dominance over left. Nevertheless, the kinetocardiograms appear to offer a more accurate means of recognizing right ventricular predominance than the electrocardiogram. This is as expected, since the precordial movements represent mechanical effects of the heart instead of electrical events.

Preliminary observations on patients with right ventricular hypertrophy due to other causes suggest that there are similarities between the two, as well as certain differences. Whether the differences are sufficiently specific to warrant a differential diagnosis of right ventricular predominance is still uncertain at the present time. The characteristics pointed out in this study, however, do appear constant in patients with mitral stenosis. These basic movements presented are altered as other types of valvular defects change the load on the heart. Findings in mitral insufficiency will be presented in a subsequent communication.

It is not necessary to discuss the clinical aspects of mitral stenosis, since much has already been written concerning the evaluation and the selection of patients for mitral commissurotomy. The data in table 1 illustrate the limitations of the electrocardiogram and fluoroscopic examination in the study of patients with mitral stenosis.

**Conclusions**

1. Kinetocardiographic (precordial movements) and ballistocardiographic data obtained from patients with "pure" mitral stenosis are presented.

2. The dynamics of ventricular contraction and relaxation and thus the precordial movements (kinetocardiograms) appear to be significantly altered from that noted in normal subjects.

3. The findings are interpreted as indicating the following events in mitral stenosis: (a) An exaggerated anterior swing of the heart in early systole associated with a footward tug on the body (FG downstroke) as the result of the contraction of the right ventricle; (b) Diminished apex thrust; (c) An exaggerated posterior and leftward swing of the heart in early diastole as the result of right ventricular relaxation, al-

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* It is possible that this configuration is only apparent with the damping technic employed in this study.
lowing the still contracted left ventricle to pull the heart posteriorly and leftward.

**CONCLUSIONES IN INTERLINGUA**

1. Es presentate datos cinetocardiographic (movimentos precordial) e ballistocardiographic obtenite ab patientes con “pur” stenosis mitral.

2. Le dynamica de contraction e relaxation ventricular e consequentemente le movimentos precordial (cinetocardiogrammas) pare significativamente alterate in comparation con illo observate in subjectos normal.

3. Le constatazioni indica, secundo nostre interpretation, le sequent phenomenos caracteristic de stenosis mitral: (a) Un exaggerate propulsion anterior del corde al comenziamento del systole, associate con un traction que se exercite super le corde in le direction del pedes. Isto resulta del contraction del ventriculo dextere. (b) Un diminuite pulsata apical. (c) Un exaggerate propulsion posterior e sinistrose del corde al comenziamento del diastole. Isto resulta del relaxation del ventriculo dextere e permitte al ventriculo sinistre (que es ancora contrahite) de exercer super le corde un traction posterior e sinistrose.

**ACKNOWLEDGMENT**

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