The Abnormal Ballistocardiogram in Mitral Stenosis

The Relationship of the Abnormal Waves to Right Ventricular Ejection and to the Mean Pulmonary Artery Pressure

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Two abnormal waves present in the ballistocardiograms of 100 cases of mitral stenosis are described and the abnormality is classified into five grades. Evidence is presented which indicates that the abnormal waves are a result of ejection from the right ventricle and that the degree of abnormality of the ballistocardiogram bears a close relationship to the mean pulmonary artery pressure in mitral stenosis.

Despite the increased interest shown in the study of mitral stenosis since surgical treatment became possible, there have been few reports on the ballistocardiographic changes in this disease. Starr, using a high frequency bed, described the changing size and improved form after digitalis therapy in several cases of chronic rheumatic heart disease and Starr and Mayock found abnormal forms in 45 per cent of their cases of this disease. Brown and co-workers described large diastolic L and N waves in some cases of mitral stenosis and Mather and associates, using a low frequency bed, secured records with bowing of the JK segments in two cases. Dock and Taubman commented on the prominent L waves which sometimes exceeded the J waves on the direct body ballistocardiogram in rheumatic heart disease. In contrast to these obviously divergent results, Davis and colleagues described a deformity of the late diastolic and early systolic portions of the ballistocardiograms which they found consistently in 13 of their 14 cases of uncomplicated mitral stenosis and they divided these abnormalities into four grades of severity. A similar abnormality is illustrated by de Soldati and co-workers.

The large number of patients referred to the Newcastle-upon-Tyne General Hospital with chronic rheumatic heart disease provided an excellent opportunity to compare the ballistocardiograms of these patients with their clinical findings, with the results secured by cardiac catheterization, with the surgeon's impression of the lesions at operation and with the changes which resulted after mitral valvotomy.

It is the purpose of this report to describe the characteristic abnormalities of the ballistocardiogram found in the cases of compensated mitral stenosis studied. Evidence is presented chiefly in the form of simultaneous recordings of the electrocardiogram, ballistocardiogram and pressure curve from the pulmonary artery or right ventricle which provides an explanation of the genesis of these abnormal ballistocardiographic waves. The evidence indicates that the degree of abnormality of the ballistocardiogram bears a close relationship to the mean pulmonary artery pressure.

Method and Material

The diagnosis of mitral stenosis in the 100 patients studied was made by Dr. W. G. A. Swan, Dr. F. Jackson or Dr. P. Szekely in the Regional Cardiovascular Department, Newcastle General Hospital, Newcastle-upon-Tyne, on the basis of clinical, radiological and electrocardiographic findings. The sex and age incidence of the 100 patients were, 26 males, 74 females.
aged from 21 to 50 years and 74 females aged from 17 to 53 years. Eighteen males and 52 females had normal rhythm and 8 males and 22 females had auricular fibrillation. No case clinically diagnosed as having predominantly mitral incompetence or found to have severe mitral incompetence at operation and no case of severe aortic incompetence or right ventricular failure was included in the series. The systemic blood pressure in all cases was within normal limits and in no instance did the heart rate exceed 120 beats per minute. In 23 of these cases an electrocardiogram, ballistocardiogram and pressure tracing from the pulmonary artery or right heart were recorded simultaneously and in 77 the pressures obtained at cardiac catheterization were not recorded simultaneously with the ballistocardiogram but were made under similar conditions and within 15 minutes of one another.

A high frequency Starr-type table ballistocardiogram (10 cycles per second when loaded with 150 pounds of dead weight) with a platform weight of 32 pounds, was used in the investigation and the tracings were photographed and recorded on a tribeam electrocardiograph. Each record was standardized, the instrument being adjusted so that a 1 cm. displacement of the base line was produced by the application of a 280-Gm force to the platform. At first, the electrocardiogram, simultaneously recorded with the ballistocardiogram, was taken from the right arm and right leg; later the right arm and left leg were used. The beginning of the first deflection of the QRS complex of the electrocardiogram was utilized to time the waves of the ballistocardiogram and the pressure tracings; these times were measured to the nearest 0.01 second.

Catheterization of the pulmonary artery and right heart were performed on each patient by Dr. F. Jackson, Dr. R. E. Irvine or the author. A Southern Instrument Company recording electromanometer was used to record the pressures at the catheter tip. Repeated experiments using the same damping needle and the same length of polythene tubes as were used for catheterization, showed this apparatus to have a time lag of 0.02 second in transmitting pressure changes from catheter tip to record. A saline manometer was included in the system to permit us to obtain mean pressures at any time during the experiment and to calibrate the graphic record.

In order to avoid excitation during the catheterization and so to avoid changes in heart rate which might seriously alter both the intracardiac pressures and the form of the ballistocardiograms, the patients were given 1/4 grain (20 mg.) of Omnopon and 1/40 grain (0.4 mg.) of scopolamine one hour before the test. At first, the catheterization was performed and the pressures recorded in one room after which the patient was transported by stretcher to a room nearby where the ballistocardiogram was recorded. Later in the investigation a technique was developed by which electrocardiogram, ballistocardiogram and pressure curves were recorded simultaneously. In these experiments after a routine catheterization, the patient was transferred to the ballistocardiograph room with the catheter tip in the main pulmonary artery, the catheter being kept patent by a slow injection of saline from a 50 cc. syringe. As soon as the patient had been placed on the ballistocardiograph table the pressure pickup unit was reconnected with the catheter and simultaneous records of pulmonary artery pressure, electrocardiogram and ballistocardiogram were taken. Then the catheter tip was withdrawn, first into the right ventricle then into the right auricle and finally into the superior vena cava, the character of the pressure curve as seen on an oscilloscope indicating the position of the tip. Records simultaneous with ballistocardiograms were secured with the catheter tip in each of these positions.

Some days after these records had been secured, mitral valvotomies were performed on the 61 patients who were thought to be suitable candidates for this operation by Mr. G. Mason or Mr. S. Griffin in the Department of Thoracic Surgery, Shotley Bridge General Hospital. The surgeons noted the state of the mitral valve before and after operation and I have accepted their findings unconditionally. In 56 cases ballistocardiograms were repeated three weeks after operation.

Results

Description of the abnormal waves

In the cases of mitral stenosis investigated, small abnormal waves are superimposed on the normal waves of the ballistocardiogram. These waves are illustrated diagrammatically in figure 1, where they have been given the letters RI and RJ, the letter R being chosen because evidence will be presented suggesting that the genesis of these waves is due to events connected with right ventricular ejection. As is shown on figure 1, the position and amplitude of the extra waves are not the same on all records so that it is convenient to classify the abnormality into five grades depending on the relative distances below the base line of the nadirs of the first abnormal footward wave, RI, and the subsequent I wave. As the normal II wave is usually absent in auricular fibrillation because of the failure of effective auricular contraction, the abnormal waves are more easily seen when the auricles are fibrillating than in the presence of normal sinus rhythm. The findings in the cases having auricular fibrillation will therefore be described first.
**Atrial fibrillation.** In what I have called a grade 1 abnormality, the first extra wave, RI, shown by the solid arrow in figure 1, is a small footward deflection which commences 0.06 to 0.1 second and has its nadir at 0.08 to 0.12 second after the beginning of the QRS segment of the electrocardiogram. The nadir of this deflection is usually at, or immediately below, the base line. The second extra wave RJ shown by the broken arrow in figure 1, is a small headward wave with its peak at 0.13 to 0.16 second after the beginning of the QRS segment. Immediately after this wave the downward deflection of the usual I wave of the ballistocardiogram occurs, which is followed by a headward J wave, both much larger than the abnormal waves. The footward RI wave is absent in a few records with grade 1 abnormality, and in these the first deflection is the headward RJ wave. In a grade 2 abnormality (fig. 1) the RI wave is deeper than in grade 1 although the nadir is not so far below the base line as is the nadir of the I wave itself. In grade 3 (fig. 1) the nadirs of the RI and I waves are on the same horizontal level and in grade 4 (fig. 1) the nadir of RI is lower than that of the following I wave. Grade 5 (fig. 1) shows a more complicated picture, for an additional headward and a footward wave occur between the RI and RJ waves and the nadir of the RI wave is usually at a level lower than that of the I wave. The time interval from the QRS segment of the electrocardiogram to the beginning of the RI wave is approximately the same in all the grades of abnormality as is the interval from the QRS segment to the nadir of the RI wave. As the grade of abnormality increases, however, there is a lengthening of the time interval from the QRS segment to the top of the RJ deflection.

**Sinus rhythm.** In the cases of mitral stenosis with sinus rhythm studied, the abnormal waves RI and RJ occur after the normal H wave. The configuration produced by the abnormal waves superimposed on the normal waves of the ballistocardiogram permits the grouping of the abnormalities into the same five grades as described in the preceding paragraph and are diagrammatically represented in figure 1.

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**Fig. 1.** Diagrammatic representation of the abnormal ballistocardiograms, RI and RJ, present in 100 cases of mitral stenosis. The abnormality is divided into five grades in both auricular fibrillation (left) and sinus rhythm (right). The electrocardiogram represents lead II and the time lines represent 0.2 second. The abnormal waves, RI and RJ, are marked respectively by solid and broken arrows. In a grade 1 abnormality the nadir of the RI deflection is at, or just below, the base line. In grade 2 the nadir of RI is deeper than that in grade 1 but not so far below the base line as the nadir of the I wave. In grade 3 the nadirs of the RI and I waves are at the same horizontal level and in grade 4 the nadir of RI is below that of the I wave. Grade 5 shows an additional headward and footward wave between the RI and RJ waves.
subsequent text grade 5 will be described as being the most abnormal grade and grade 1 the least abnormal.

The results of the investigation show certain facts on which it is proposed to base an interpretation of these abnormal waves. It is easiest to describe the findings in auricular fibrillation and in normal sinus rhythm separately. (1). In atrial fibrillation, variation of the length of the diastolic pause is accompanied by alteration in the amplitude and timing of the abnormal waves. Figure 2E gives a typical example. The longer the diastolic pause the lower the classification of the abnormality when this is judged by the criteria shown in figure 1. Complexes showing grades 2, 1 and 4 abnormalities are present in the example shown in figure 2E, the lowest grade following the longest diastole and vice versa. This association was clearly seen in 21 records of the cases with...
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Fig. 3. The relationship between the mean pulmonary artery pressure in millimeters of mercury, and the highest grade of abnormality of complexes repeatedly seen in the ballistocardiogram of each of 100 cases of mitral stenosis. The heart rates during the pressure recording and the ballistocardiogram were closely similar. The dots represent 70 cases with normal sinus rhythm and the crosses 30 cases with auricular fibrillation. Circles have been drawn about those points which represent the grade of ballastic abnormalities of the 10 patients who were the last to be investigated in this series and in whom the ballistocardiograms were graded before the pulmonary artery pressures were known. It is seen that the mean pressures of these 10 patients fall within the pressure limits of the 90 patients investigated earlier who had comparable grades of ballastic abnormalities.

Companied by corresponding changes in the positions relative to the base line of the nadirs of the RI and I waves. This is well shown in figure 2B where it can be seen that when the preceding end-diastolic pressure is high, the grade of abnormality of the extra waves is high and vice versa. I have records from 10 cases which show this relationship. (3) The association of the mean pulmonary artery pressure with the abnormal ballastic waves is also manifest in the 30 patients of the series with auricular fibrillation. In these, the mean pulmonary artery pressures ranged from 17 to 74 mm. Hg. The relation of the grade of abnormality of the ballistocardiogram of each patient to this pressure is not a simple one, for the grade varies from beat to beat as was described before. But, when the highest grade of abnormality of the complexes seen repeatedly in any tracing, disregarding those seen only once or twice, is plotted against the mean pulmonary artery pressure, a very close association between the two is clearly seen (crosses on fig. 3). The only exceptions to this close relationship were found in cases not included in the present series in which pulmonary artery pressures were measured at a time when the heart rate was very different from that present when the ballistocardiograms were taken. (4) In 10 cases with auricular fibrillation, ballistocardiograms were taken a few days before and three weeks after mitral valvotomy. After valvotomy, judged to be successful, the character of the abnormal waves improved in seven of the cases and they were then classified in a lower grade. An example of such improvement is shown in figure 5C. In the remaining three cases, the abnormal waves remained unchanged after operation.

In general, the observations recorded in sections (3) and (4) of the preceding paragraph were repeated in the 70 cases of mitral stenosis studied while in normal sinus rhythm. Additional features of these results, however, require further description. (5) In sinus rhythm the character of the abnormal waves varied with the respiratory cycle in almost all cases, an effect masked in auricular fibrillation by the greater effect of variation in duration of diastole. During expiration the grade of ab-
normality of the ballistic complexes was higher than during inspiration (fig. 4C, 4F and 4G). (6) Two or more ballistocardiograms were secured from each of six patients with normal rhythm whose heart rates showed a marked difference on each record. In these, the higher rates were associated with higher grades of abnormality. Similarly, when extrasystoles occurred, the ballistic complex accompanying the premature contraction gave wave abnormalities graded higher than those following normal sinus beats. (7) As in the cases with auricular fibrillation, the records of 70 cases in sinus rhythm showed a striking relationship between the mean pulmonary artery pressure and the grading of the most abnormal complexes repeatedly seen on the ballistocardiogram, and this is shown by the dots on figure 2. This relationship existed only if the heart rates were closely similar when both investigations were made. (8) There was also improvement in the grading of 31 out of 40 cases of mitral stenosis in sinus
rhythm after successful mitral valvotomy. Two examples are shown in figure 5, A and B. The data on the effect of this operation on the ballistocardiogram have not been fully assembled and will be presented in total at a later date.

Further facts about these abnormal waves became evident after studying the records secured in the 23 patients in whom electrocardiogram, ballistocardiogram and pressure curves from the main pulmonary artery or right ventricle were recorded simultaneously. Thirteen of these patients had sinus rhythm and the characteristic ballstic abnormality seen on the records was classified grade 1 in two patients, grade 2 in four patients, grade 3 in three patients, grade 4 in two patients and grade 5 in two patients. Of the 10 patients with auricular fibrillation two had a characteristic abnormality of grade 1, two of grade 2, two of grade 3 and four of grade 4. No simultaneous records were obtained from any patients with auricular fibrillation and a grade 5 ballstic abnormality.

When the time relations of the simultaneous ballistocardiograms and pressure records are compared, adjustment for the time lag in the pressure recording apparatus has always to be made. Inspecting my records, I find it easiest to make the adjustment by moving the ballistocardiogram 0.02 second to the right. As the pulmonary artery pressure tracings give a more accurate timing of the onset of ejection from the right ventricle than do the right ventricular tracings, greater emphasis will be placed on the former records when we seek to locate the onset of right ventricular ejection.

Figure 4, A to D shows typical examples of tracings taken with the catheter tip in the pulmonary artery from patients in sinus rhythm. Ballistic complexes illustrating abnormalities graded 1 to 5 are also shown. A sharp pressure fluctuation is recorded at the onset of systole in some of the pulmonary artery tracings which I attribute to movement of the catheter tip at the onset of the ventricular contraction. Despite this artefact, it is easily seen that the RI wave begins at the time of onset of the rise of pulmonary artery pressure or within 0.02 second before it and the RJ wave begins at or immediately after the onset of pulmonary artery pressure rise. When the right ventricular pressure curves are studied.
(fig. 4, E to H) the RI waves are seen to begin at a time when the pressure in the right ventricle is rapidly rising and the onset of the RJ wave corresponds to a point located higher on this curve.

The tracings from the patients with auricular fibrillation (fig. 2, A to F) show the abnormal waves very clearly and disclose a similar time relationship between the RI and RJ waves and the pulmonary artery and right ventricular pressure curves.

**DISCUSSION**

The abnormal waves described in this paper seem identical with those described in 13 cases of mitral stenosis by Davis and associates, in one such case by Kuo and his colleagues, and in one illustrated by de Soldati. This more extensive investigation was designed to throw light on the genesis of these waves and their relation to the pathological physiology which results from the presence of mitral stenosis.

Kuo and his associates suggested that the so-called H wave of the ballistocardiogram, sometimes seen in patients without auricular contraction, could be attributed to asynchronous ejection of the ventricles and they also noted that “double peaked” systolic ballistic complexes were encountered almost exclusively in patients, including one with mitral stenosis, in whom the electrokymogram showed a systolic pulsation of the pulmonary artery which preceded that of the aorta. Davis and co-workers, seeking for an explanation of the abnormal waves, mentioned the effects of the pulmonary vascular resistance, ventricular asynchrony and the auriculoventricular gradient as possibly contributing towards the production of the abnormal early systolic waves. Later, Kuo and Schnabel described similar ballistic waves in 10 cases of mitral stenosis with proven severe incompetence and suggested that the early systolic headward wave in these cases was probably produced by the backflow of blood through the incompetent mitral valve.

The results obtained in the present series of cases demonstrate clearly that the character and timing of the abnormal waves seen in mitral stenosis vary as the pulmonary artery pressure varies. Changes in pulmonary artery pressure go hand in hand with changes in the abnormal RI and RJ waves of the ballistocardiograms when heart rate changes, when the duration of the preceding diastole varies, with the changing phases of respiration and with the improvement following successful mitral valvotomy. Indeed, whenever the pulmonary artery pressure changes in any direction in the cases studied a similar change in the abnormal waves accompanies it. Therefore, the conclusion is unavoidable that the two are strongly correlated and one has the right to believe that the abnormal ballistic waves have their origin in events connected with the contraction of the right ventricle.

The two waves, RI and RJ, by which abnormality of the ballistocardiogram may be recognized in mitral stenosis, occur both in cases with normal sinus rhythm and in those with auricular fibrillation; therefore, auricular contraction is not a factor in their genesis. In the cases with normal sinus rhythm, the footward RI wave begins immediately after the usual H wave of the ballistocardiogram; in the cases with auricular fibrillation, the H wave being absent, RI is usually the first footward deflection of the ballistocardiogram, coming 0.06 to 0.1 second after the beginning of the QRS complex of the electrocardiogram. As has been shown, the RI wave begins at, or immediately before, the onset of the pressure rise in the pulmonary artery and therefore close to the beginning of ejection into that vessel. The artefacts occurring at the onset of systole in some of the pulmonary artery pressure tracings make it impossible to define the relationship more accurately. The RJ wave is seen to begin in all cases at or shortly after the onset of pulmonary artery pressure rise, and so at or just after the onset of right ventricular ejection. As the evidence indicates that the RI wave so closely coincides with the beginning of right ventricular ejection, it seems reasonable to suggest that this wave is caused by the footward recoil from right ventricular ejection and the following RJ wave may well be due to the headward impact of ejected blood striking the curve and bifurcation of the pulmonary artery, so
that the abnormal waves are in some respects comparable to the usual I and J waves of left ventricular ejection. It is not suggested, however, that the RI and RJ waves represent the right ventricular ballistocardiogram completely. Indeed, as the pulmonary artery pressure continues to rise after the completion of the RJ wave, it seems more probable that this wave of right ventricular origin is interrupted by the footward recoil wave of left ventricular ejection; the normal I wave. In the cases of mitral stenosis investigated when the pulmonary artery pressures were only slightly above normal the RI and RJ waves were small. In the cases with high pulmonary artery pressure when ballistic complexes with abnormalities of grades 4 and 5 are seen, the position and frequently the amplitude of the RJ wave approach more closely the position and amplitude of the normal J wave. This suggests that in a high pressure system the forces generated during systole by the hypertrophied right ventricle increase in magnitude and approach in timing those produced by the left ventricle and that they influence the ballistocardiogram accordingly.

In the past, it has been assumed that the effects of right ventricular ejection are summed with those of the left ventricle in producing the I and J waves of the normal ballistocardiogram; and, except in abnormal tracings which show a bifid J wave, the effects of ejection from each ventricle cannot be separated. If, as is suggested in this communication, the abnormal waves of the ballistocardiogram in mitral stenosis, RI and RJ, are a result of right ventricular ejection, then the question to be asked is why do these waves appear appreciably earlier than the I and J waves of the same tracing. Consideration of two important differences between the anatomy and physiology of the right and left sides of the heart may help to answer this question. First, the distance from the pulmonary valve to the bifurcation of the pulmonary artery is much shorter than that from the aortic valve to the arch of the aorta. Thus, although waves in the two vessels cannot be expected to travel with an equal velocity, because of the shorter distance to travel, the headward impact when blood changes direction at the pulmonary artery bifurcation may precede that, due to blood rounding the aorta.

The second difference is that the end diastolic pressure found in the pulmonary artery is much lower than that in the aorta. It is conceivable that because of this great difference, the right ventricular ejection pressure is reached sooner and right ventricular ejection occurs earlier, and the ballistic effects of this ejection are manifest earlier than are those due to left ventricular ejection. A search of the literature has failed to find any mention of simultaneous right and left ventricular pressure tracings taken in cases of mitral stenosis. However, the author had the opportunity of securing pressure tracings from both the arch of the aorta and the main pulmonary artery in a proven case of patent ductus arteriosus when the catheter tip passed through the ductus. In this case, the mean pulmonary arterial pressure was elevated above the normal, being 38 mm. Hg, while the aortic mean pressure was 83 mm. Hg. The ballistocardiogram in this patient showed abnormal waves similar to those just described in cases of mitral stenosis with a grade 3 ballistic abnormality. The beginning of the pressure rise at the arch of the aorta was 0.04 second after the onset of the pressure rise near the bifurcation of the pulmonary artery. Therefore, the ballistic effects of ejection from the right ventricle must have preceded those of left ventricular ejection in this case.

The evidence suggests that a similar asynchrony of recoil and impacts also takes place in mitral stenosis when the pulmonary artery pressure is elevated by another mechanism. This line of reasoning indicates that the occurrence and position of the abnormal ballistic waves described as RI and RJ depends primarily on asynchronism of ventricular ejection and not on any particular anatomical abnormality. In the cases I have described, the asynchronism was associated with elevation of the pulmonary artery pressure, but Scarborough and his co-workers reported cases of constrictive pericarditis which showed ballistic abnormalities similar to those shown by the one case of patent ductus arteriosus and the many cases of mitral stenosis described in this investigation.
There is an important corollary to these observations. When the investigation of the mitral stenosis cases had proceeded far enough to indicate the striking relationships between the grade of abnormality of the ballistocardiogram and the mean pulmonary artery pressure (as shown in fig. 2), one could guess the height of this pressure before knowing the cardiac catheterization findings, by observing the highest grade of abnormality repeatedly seen in the ballistocardiogram and noting the pressure range of the patients previously investigated with the same abnormal ballistic grade. In figure 3 circles have been drawn about the points representing abnormalities graded in this unbiased fashion. It will be seen that they correspond well with the pulmonary artery pressures found. This relationship was only seen if the heart rates during both investigations were closely similar.

SUMMARY

(1) Two abnormal waves present in the ballistocardiograms of 100 cases of mitral stenosis are described. These waves occur both in patients with sinus rhythm and in patients with auricular fibrillation and are named the RI and RJ waves. The RI wave is a footward deflection commencing 0.06 to 0.1 second after the beginning of the QRS complex of the electrocardiogram and the RJ is a headward deflection immediately following the RI and commencing 0.08 to 0.12 second after the QRS complex. In sinus rhythm these waves follow the H wave.

(2) This ballistocardiographic abnormality is divided into five grades of severity; the classification depending on the relative distances below the base line of the nadirs of the RI and the I waves. In grade 1, the least abnormal grade, the waves are very small and the nadir of the RI wave is at or immediately below the base line while the nadir of I is well below it. In grade 2, the nadir of the RI is deeper than in grade 1 but not as far below the base line as that of the I wave. In grade 3, the nadirs of RI and I are on the same horizontal level. In grade 4, the nadir of RI is further below the base than that of the I wave. Grade 5 is more complicated and shows an additional headward and footward deflection occurring between the RI and RJ waves.

(3) The grade of ballistic abnormality changes with variation in the mean pulmonary artery pressure when the heart rate changes, when the duration of the preceding diastolic pause varies, with the changing phases of respiration and with the improvement following successful mitral valvotomy. The higher the mean pulmonary artery pressure the higher is the grade of ballistic abnormality.

(4) A close correlation is shown between the mean pulmonary artery pressure and the maximum grade of abnormality of complexes repeatedly seen in the ballistocardiogram in each of the 100 cases of mitral stenosis studied.

(5) Simultaneous records of electrocardiogram, ballistocardiogram and pressure tracings from the pulmonary artery or right ventricle in 23 cases of mitral stenosis show that the RI and RJ waves are closely related in time to the commencement of right ventricular ejection. It is suggested that the RI wave is caused by the footward recoil resulting from right ventricular ejection and the following RJ wave is caused by the headward impact of ejected blood striking the curve and bifurcation of the pulmonary artery.

SUMMARIO IN INTERLINGUA

Es describite duo undas anormali incontrate in le ballistocardiogrammas de 100 casos de stenosis mitral. Le anormalitate es classificate in cinque grados. Es presentate datos que indica que le undas anormal es le resultato de un ejection ab le ventriculo dextere e que le grado del anormalitate del ballistocardiogramma es netemente relacionate al pression pulmonarterial median in stenosis mitral.

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