Movements and Forces of the Human Heart
V. Precordial Movements in Relation to Atrial Contraction

By T. R. Harrison, M.D., John A. Lowder, M.D., Lloyd L. Hefner, M.D., and Donald C. Harrison, M.D.

Precordial movements starting after P and before Q have been studied. Right atrial contraction appears to cause a forward-rightward motion and left atrial contraction a backward movement of the precordium. The latter is exaggerated in most patients with mitral stenosis and in some patients with myocardial failure.

STUDY of records of precordial movements of certain patients with cardiac disease has indicated that motions beginning after the P wave and before the Q of the electrocardiogram are sometimes strikingly exaggerated. An understanding of these abnormal motions can only be achieved if one can first interpret the significance of the smaller movements occurring in normal subjects at this time. This communication represents an attempt at such an interpretation.

METHODS

Ninety-nine subjects were studied. They were divided into 6 groups, as follows: (1) 22 healthy adult males aged 21 to 39; (2) 3 subjects with varying degrees of heart block; (3) 30 patients with mitral stenosis and sinus rhythm; (4) 10 persons with “pure” mitral insufficiency; (5) 7 patients with cor pulmonale; (6) a miscellaneous group of 27 consecutive subjects with sinus rhythm and various other types of acquired cardiac disease.

Tracings of precordial motions (kinetocardiograms) were obtained either by the crossbarbellows technic previously described,1 or by using a bellows in the center of the funnel, with a terminal cross-sectional diameter of 11 cm. In a few instances a tripod was used rather than the funnel. The results obtained by the different technics were similar. All recordings were made with a 4-channel Sanborn Direct Writer, the electrocardiogram and carotid pulse curves being used for reference.

Most of the traces were taken while the breath was held at the end of normal expiration. In a number of subjects records were made during normal breathing. However, the large respiratory excursion of the chest tended to obscure the smaller circulatory motions.

Tracings were taken from the right and left parasternal regions in the fourth interspace (K1 and K4 areas), and from the left midlavicular line in the fifth interspace (K5 area). In a number of subjects the precordium was “mapped” by taking multiple records from the second to the sixth interspace in the 2 parasternal and in the left midlavicular lines. It was found that these extensive recordings yielded little additional information, and therefore in most of the subjects only records from the K1, K4 and K5 points were analyzed in detail.

In labeling the tracings the letter K indicates “kinetocardiogram,” the first numeral in the subscript refers to the V position, and the second to the interspace. Thus K14 is the record from the right parasternal line in the fourth interspace, K5 the left midlavicular line in the fifth interspace, etc.

In the analysis of the records a movement was considered to be of atrial origin only if it started after the onset of the P wave of the electrocardiogram, and either before or within .02 second after the start of the QRS complex. In subjects with rapid heart rates there were often large waves just prior to the P wave, which were probably related to passive ventricular filling (fig. 1B). Individuals with slow rates often displayed very small motions before the P wave, the cause of these being unknown.

The directions of movement were studied, an upstroke representing an outward motion and a downstroke an inward motion. The time of onset of a given movement in relation to the beginning of the P wave was measured. Since we have no entirely satisfactory method of calibrating the absolute magnitude of precordial movements, as yet, only relative magnitude could be studied. Such relative magnitudes were expressed as the number of millimeters deflection of a given motion.
in relation to the greatest total deflection during the cardiac cycle. Thus an increase in relative magnitude might be the result of augmented atrial motion or of diminished ventricular motion.

After these studies had been completed, it was deemed desirable to investigate the lateral movements in relation to atrial contraction. Therefore, kinetocardiograms were recorded (with the cross-bar technic) on 10 normal young adult males, from multiple interspaces in each midaxillary line. The records from the fifth and sixth interspaces were selected for study because they displayed the least variability. Lateral ballistocardiograms, as obtained by the low-frequency acceleration technic used in this laboratory, 2 were also analyzed. Lateral motions have not yet been studied in patients with cardiac disease. Longitudinal ballistocardiograms were also recorded but are not discussed because they yielded inconsistent results during this portion of the cardiac cycle.

**RESULTS**

The more pertinent findings are illustrated in figures 1 to 5, and the data are summarized in the tables.

**A. Anteroposterior movements.**

The normal magnitude of atrial motion in relation to the largest excursion of the cycle is illustrated in figure 1. It may be noted that the 2 subjects, although displaying con-
Considerable difference in magnitude of the different motions, exhibited similarity in configuration of the movements starting after the P wave and before the QRS. During this period all the normal subjects exhibited 3 successive movements, consisting of an initial upstroke (forward), followed by a downstroke (backward), and a second terminal upstroke (forward). Since there is reason to believe that these 3 motions represent different forces, they may now be considered separately.

1. Initial Forward Movements. This motion was present in the right parasternal (K₁) region in each of the normal subjects, but was less constant in the left parasternal (K₂) and left midclavicular (K₃) areas. The motion was usually larger at the K₁ region than at the other points. In some instances this upstroke consisted only of an abrupt cessation of a previous downstroke; in others, it represented a change in slope of a previous more gradual forward motion. The time of onset of this forward motion was relatively constant, being .05 ± .03 second after the onset of the P wave. The relative magnitude was small, varying from one twentieth to one fourth of the total excursion of the cycle, and averaged about one eighth. In no normal subject was this movement larger than 25 per cent of the total amplitude of the cardiac cycle (table 1).

<table>
<thead>
<tr>
<th>Group</th>
<th>Number of records</th>
<th>Initial upstroke greater than</th>
<th>Initial downstroke greater than</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1:4</td>
<td>1:3</td>
</tr>
<tr>
<td>Normal</td>
<td>22</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Mitral stenosis</td>
<td>30</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Mitral insufficiency</td>
<td>10</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Cor pulmonale</td>
<td>7</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>Myocardial infarction, old</td>
<td>10</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Angina pectoris</td>
<td>5</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Hypertension</td>
<td>5</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Aortic insufficiency</td>
<td>5</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Myocarditis (?)</td>
<td>2</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

Fig. 2. Complete heart block. Paper speed 25 mm. per second. Following the first P wave there is an initial upstroke but the subsequent atrial events are masked by ventricular forces. After the second P wave, which is not followed by ventricular excitation, the kinetocardiogram displays (1) forward motion; (2) a diphasic backward motion; (3) a small up-down noteh; and (4) a second forward motion. The initial forward motion is ascribed to contraction of the right atrium, the backward motion to contraction of the left and relaxation of the right, and the terminal upstroke to left atrial relaxation. The magnitude of excursion of the atrial motions is less than one fourth that of the ventricular forces. The cause of the small up-down notch that precedes the terminal forward movement is uncertain.

This initial forward movement was present after isolated P waves in each of the 3 subjects with heart block (fig. 2), but displayed considerable variation in 30 records obtained from 27 patients with mitral stenosis. At the K₁ area in the latter subjects, the initial up-
Fig. 3. Cor pulmonale. Paper speed 50 mm per second. Note the large upstroke starting approximately .05 second after the beginning of the P wave. This is 50 to 60 per cent of the total excursion of the complex. In normal subjects this upstroke, which is ascribed to right atrial contraction, is not more than one fourth of the total deflection. The downstroke ascribed to left atrial contraction is relatively small. In most healthy subjects this downstroke is as large as the preceding upstroke.

stroke was exaggerated (greater than one fourth of the total excursion of the cycle) in 8 instances, was absent in 7 records, and of a magnitude within the normal range in the remaining records. Consistent differences from the normal tracings were likewise not found in the K2 and K4 regions of the patients with mitral stenosis.

Two of the subjects with mitral insufficiency displayed exaggeration (greater than 25 per cent) of the relative magnitude of the initial forward motion. In the remainder, the amplitude was within the range of normal.

The most interesting variation was found in the subjects with cor pulmonale. Five of these 7 patients displayed larger relative amplitude than was seen in any of the 22 normal subjects (fig. 3 and table 1).

In 12 of the 27 subjects with other types of cardiac disease, the initial atrial forward motion was exaggerated in relative magnitude.

Whether this was the result of an actual increase in the atrial force or of an apparent increase relative to diminished ventricular excursion is uncertain at present.

2. Initial Backward Motion. In normal subjects this was the most constant movement, being present at the K1 point in all of the individuals, and at the K2 and K4 areas in nine tenths of the records. The onset was latest and least variable (.11 ± .04 second after P) at the K1 area (fig. 1), and occurred earliest at K4 (.07 ± .06 second after P). The values in the K2 region were intermediate. The relative magnitude of this downstroke at the different areas varied in different subjects. In none of the healthy individuals was the relative magnitude at the K1 area greater than one third of the total excursion, and in 21 of 22 subjects it was one fourth or less (table 1).

The 3 subjects with heart block all displayed this downstroke, and in each of them it was noted to be biphasic (fig. 2). This biphasic feature was only occasionally noted in normal subjects, or in patients with other types of cardiac disease.

Individuals with mitral stenosis exhibited exaggeration of this backward motion (fig. 4A and C). It was present at K4 and K2 in all such subjects, but was occasionally absent in the K1 region. This backward motion was absent during atrial fibrillation (fig. 4B). At the K1 region this movement was greater in 27 of the 30 records from patients with mitral stenosis than in any of the normal subjects (table 1). The average magnitude was also greater at the K2 and K4 points, but was outside the normal range only in about one half of the records obtained from these regions.

Seven of the 10 patients with mitral insufficiency, and 1 of the 7 patients with cor pulmonale, exhibited increase in the relative amplitude of this backward motion in the right parasternal area. The magnitude was likewise increased in 17 of the 27 patients with other types of cardiac disease (table 1). As in the case of the preceding forward movement, it is uncertain whether the atrial motion was actually increased or whether the ventricular excursion was diminished.
3. Second Forward Motion. This movement was present at all areas in most normal subjects. The onset was \(0.16 \pm 0.02\) second after the beginning of the P wave in the K\(_1\) region, but was more variable in the regions to the left of the sternum. In normal subjects, the beginning of this upstroke is frequently \(0.01\) to \(0.02\) second after the beginning of the QRS. Nevertheless, the movement is apparently not of ventricular origin because it frequently starts before the QRS, and, likewise, because it was present following isolated P waves in each of 3 patients with heart block (fig. 2). This movement began later \((0.23 \pm 0.03\) second\) after the beginning of the P wave in such patients than in normal subjects. The reasons for this are unknown.

This second upstroke appeared to be somewhat larger in patients with mitral stenosis than in normal subjects, but definite conclusions concerning amplitude of this motion cannot be drawn because it is usually interrupted by ventricular forces shortly after it begins.

B. Lateral Movements

A typical lateral ballistocardiogram is illustrated in figure 5. The normal lateral motions of the body and of the axillary interspaces in relation to atrial contraction are summarized in table 2. The ballistocardiogram typically displayed a rightward-leftward motion and that is ascribed mainly to contraction of the left atrium; (3) the terminal portion of this downstroke, which is thought to be related to relaxation of the right atrium; (4) the large terminal upstroke beginning \(0.01\) second before the QRS, which is believed to represent relaxation of the left atrium and which is apparently augmented by ventricular forces. The cause of the small up-down motion beginning \(0.05\) second before the QRS is uncertain.

Middle. Mitral stenosis—atrial fibrillation. Paper speed 25 mm. per second. Note that the first large deflection is abrupt and starts \(0.04\) second after the onset of the QRS. The usual large atrial forces are absent. Bottom. After conversion to sinus rhythm. Paper speed 50 mm. per second. Same subject as Middle. Following conversion to normal rhythm, the atrial forces have appeared. These consist of a small initial upstroke and a huge downstroke after the onset of the P wave and before the beginning of the QRS. Note similarity of these forces to those in Top.
ward-rightward sequence starting after the onset of atrial and before the onset of ventricular excitation. The axillary kinetocardiograms were complex, displaying at least 5 and often 6 directional changes between the P and Q waves of the electrocardiogram. The factors possibly responsible for these movements are considered later.

DISCUSSION

In the study of atrial movements, it would appear that records obtained from the right parasternal (K1) region are likely to be the most useful. The findings in this area in normal subjects, and in the individuals with mitral stenosis, were more constant than the movements encountered to the left of the sternum. Consequently, the discussion will be centered mainly on the motions in this area.

The observations that have been mentioned indicate that the main anteroposterior movements occurring in relation to atrial activity are 3: namely, forward, backward, and then forward. Although these movements are small in relation to the subsequent ventricular motions, they are clearly related to atrial forces and not artifacts. Thus they are present in patients with heart block following isolated P waves (fig. 2), and are absent in subjects with atrial fibrillation (fig. 4B). The initial forward movement is probably related to contraction of the right atrium. This chamber is excited first, and vector studies indicate that the excitation wave spreads leftward, forward, and footward. The forward movement begins about .05 second after the onset of the P wave, which corresponds to the beginning of the right atrial pressure rise found by Testoni et al. The movement is often largest in the region of the right atrium, and is inconstant in other areas. Unlike the following initial backward movement, this forward motion is not consistently increased in patients with mitral stenosis. In 5 of 7 patients with cor pulmonale, this motion was greater than in any normal subject, and in the remaining 2 patients it was greater than the average for normal persons.

This forward movement consequent to right atrial contraction is usually greater in the right than in the left parasternal area. It therefore does not seem to be due entirely to filling of the right ventricle. It does not appear to represent a venous A wave in the superior and inferior cavae, because the movement is not greater in the first and second (superior caval region) and fifth and sixth interspaces (inferior caval area) than in the third and fourth interspaces. It therefore seems likely that this upstroke, which is consistently present in all interspaces in the right parasternal region, represents a rightward pull on the heart by the right atrium. This chamber is fixed by the 2 cavae and shortening of its fibers would be expected to pull the remaining portions of the heart rightward, even though the right border moves leftward, as has been demonstrated by electrokymographic studies.

A rightward pull by the right atrium might tend to produce an inward motion in the left parasternal region. On the other hand, the filling of the right ventricle consequent to right atrial contraction would be expected to produce an outward movement in this area. Thus the inconstancy of the
movements in the left parasternal region at this time could be accounted for by a varying balance between two opposite forces.

The lateral ballistocardiogram exhibited a rightward-leftward sequence as the precordium moved forward (fig. 5). This can be ascribed to recoil followed by impact as blood moves from the right atrium to the right ventricle.

The axillary interspaces usually displayed rightward motion, starting slightly before the forward movement (table 2). This is in keeping with the concept that the initial effect of right atrial contraction is to pull the heart to the right. The succeeding leftward movement that occurred in traces from both axillae may conceivably have been due either to right ventricular filling or to sudden displacement of the interatrial septum to the left by the rising pressure in the right atrium.

The backward motion, starting about .10 second after the onset of the P wave, is nearly always greater in the right parasternal region in patients with mitral stenosis than in normal subjects. It is often increased in persons with mitral insufficiency, and in subjects with coronary disease or hypertension. The time of onset of this motion corresponds well with the inward movement of left atrial contraction, as seen in electrokymograms (Dussaillant), and was slightly later than the rise in left atrial pressure (Braunwald et al.). These observations would suggest that this backward motion is related to contraction of the left atrium. Since this chamber is fixed posteriorly by the pulmonary veins, its contraction would be expected to exert a backward and leftward pull on the heart.

The axillary interspaces displayed 2 motions that may be related to contraction of the left atrium. The first of these was leftward, at .09 ± .02 second after the P onset, and can probably be attributed to a leftward pull on the heart. The second and larger deflection, occurring about .03 second later, was rightward and was associated with a rightward motion in the ballistoeardiogram (fig. 5, table 2). Relaxation of the right atrium probably begins at about this time and might be associated with rightward recoil as the liver begins to empty, or with a rightward impact as the interatrial septum suddenly is shifted to the right by the rising and falling pressures in the left and right atria, respectively.

The terminal forward movement, starting about .16 second after the onset of P, offers some difficulty in interpretation because it is soon interrupted by forces that are clearly of ventricular origin. This is indicated by their abrupt nature, their large magnitude, and their onset about .03 to .04 second after the beginning of the ventricular complex of the electrocardiogram. Such ventricular forces make it impossible to study the amplitude of this terminal forward movement. Possibly it is related to left atrial relaxation, because its direction is opposite to that of the force associated with left atrial contraction, and it appears to be exaggerated in some patients with mitral stenosis.

This division of the atrial forces into 3 phases represents an oversimplification. The axillary kinetocardiograms of normal subjects usually showed 6 motions, and certain patients with cardiac disease display evidence of more than 3 anteroposterior movements. The backward motion may be diphasic (figs. 2 and 4A), and probably is related not only to contraction of the left atrium, but also to relaxation of the right. However, the 3 forward-backward movements that have been described are constant in normal subjects, and afford a point of departure for the study of atrial movements in patients with cardiac disorders.

We have been surprised at the failure to find patients with mitral stenosis, regular rhythm, and evidence of diminished force of left atrial contraction. This would be expected if there is a descending limb of the atrial Starling curve. Possibly, the atrium responds to overdistention, with fibrillation rather than with diminished strength of coordinated contractions. More detailed studies of a large number of subjects are needed to settle this point.

In regard to any possible diagnostic significance of precordial atrial motions, the fol-
Table 2.—

<table>
<thead>
<tr>
<th>Time after onset of P, (seconds)</th>
<th>.03 ± .01</th>
<th>.05 ± .03</th>
<th>.07 ± .02</th>
<th>.09 ± .02</th>
<th>.12 ± .02</th>
<th>.15 ± .02</th>
</tr>
</thead>
<tbody>
<tr>
<td>Axillary KCG</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Direction of motion</td>
<td>Rightward</td>
<td>Leftward</td>
<td>Inconstant and small rightward in 6 subjects</td>
<td>Leftward</td>
<td>Rightward</td>
<td>Leftward</td>
</tr>
<tr>
<td>Number of subjects showing motion</td>
<td>7</td>
<td>9</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range of relative amplitude</td>
<td>1/20 to 1/4</td>
<td>1/25 to 1/5</td>
<td>1/25 to 1/4</td>
<td>1/9 to 1/2</td>
<td>Interrupted by ventricular forces</td>
<td></td>
</tr>
<tr>
<td>Lateral BCG</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Direction of motion</td>
<td>----</td>
<td>Rightward</td>
<td>Leftward</td>
<td>Previous leftward motion continues</td>
<td>Rightward</td>
<td>Previous rightward motion continues</td>
</tr>
<tr>
<td>Number of subjects showing motion</td>
<td>----</td>
<td>8</td>
<td>8</td>
<td></td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>Possible cause of motions</td>
<td>Rightward pull by right atrium (KCG)</td>
<td>Right ventricular filling (KCG): recoil as rt. atrium ejects (BCG)</td>
<td>Impact of blood on rt. ventricle (BCG)</td>
<td>Leftward pull by left atrium (KCG)</td>
<td>Relaxation of rt. atrium with move of interatrial septum to rt. (KCG) &amp; rightward recoil as liver empties (BCG)</td>
<td>Leftward movement of interatrial septum as left atrium relaxes (KCG)</td>
</tr>
</tbody>
</table>

Following may be stated: The presence of marked exaggeration of the left atrial backward motion does not per se constitute significant evidence for mitral stenosis, because such exaggeration is not invariably present. Likewise, it is frequently seen in patients with mitral insufficiency, and in subjects with other types of cardiac disease. The absence of this phenomenon in a patient with sinus rhythm would appear to constitute some, but not conclusive, evidence against a dynamically significant degree of mitral stenosis. The same reasoning appears to apply, although less emphatically, to the relationship between cor pulmonale and augmentation of the initial forward motion of right atrial contraction. Thus it would appear that the precordial motions occurring between the P and Q waves of the ballistocardiogram may be of some value in differentiating mitral stenosis from cor pulmonale but are at present of limited if any value in distinguishing these two conditions from other causes of left and right heart stress, respectively.

Patients with congenital cardiac disorders have been omitted from this study because their atrial and other precordial motions are often so bizarre as to defy interpretation at the present time. In patients with acquired heart disease who have no evidence of valvular lesions, it would appear that exaggeration of one or both of the deflections ascribed to atrial contraction may indicate either (1) an absolute increase in atrial force, probably caused by incomplete ventricular emptying, with some increase in residual atrial blood, or (2) an absolute decline in magnitude of ventricular precordial motion, with only a relative increase in atrial force, or (3) a combination of these two situations. In any event, the occurrence in such a subject of increase in the relative magnitude of the precordial motions induced by atrial contraction is perhaps suggestive of decrease in the reserve power of the corresponding ventricle. Because of the great variation in normal subjects of the relative magnitude of these atrial motions, it would seem
advisable to study a series of individuals during various degrees of cardiac failure before arriving at definitive conclusions concerning this point. Such studies are in progress and will be the subject of a later report.

**Summary**

Precordial motions beginning between the onset of the P and Q waves of the electrocardiogram have been studied in a group of normal subjects, and in patients with various types of cardiac disease. During this portion of the cardiac cycle tracings from the right parasternal region have appeared to yield more consistent findings than records taken to the left of the sternum. A sequence of motions consisting of forward-backward-forward was regularly observed. These motions are clearly of atrial origin because they were absent in patients with atrial fibrillation, and were observed following isolated P waves in each of 3 subjects with heart block.

About .05 second after the onset of the P wave, a small rightward and forward motion occurred. This motion did not exceed one fourth of the total amplitude of the cardiac cycle in any of the normal subjects. It was greater than the average normal amplitude in each of 7 subjects with cor pulmonale, and in 5 of these was outside the normal range.

About .10 second after the onset of the P wave a backward and leftward motion occurred. This was exaggerated beyond the normal range in most of the 30 tracées from patients with mitral stenosis, and in 7 of 10 records from subjects with “pure” mitral insufficiency. This movement is mainly ascribed to contraction of the left atrium, which being fixed posteriorly by the pulmonary veins appears to pull the heart backward. Since the motion is sometimes biphasic, it is probable that relaxation of the right atrium may also be a contributing factor.

Patients with various types of myocardial disease may or may not show exaggeration of either or both these movements. It therefore appears that exaggeration of these atrial motions may be related to (1) a direct strain due to a valve lesion; (2) an indirect strain due to disease or overload of the corresponding ventricle.

Aside from these 2 movements that are ascribed to successive contraction of the 2 atria, the traces exhibit a second upstroke, starting just before or after the onset of the QRS of the electrocardiogram. This motion is tentatively ascribed to relaxation of the left atrium, but it cannot be studied in detail because it is soon interrupted by the larger motions due to ventricular contraction.

Lateral ballistocardiograms (low-frequency acceleration) appear to reflect right atrial activity. They exhibit a rightward-leftward-rightward sequence ascribed respectively to recoil, impact and, possibly, to right atrial relaxation.

The motions of the axillary interspaces, as recorded by the kinetoelectrocardiographic technic, are complex and usually exhibit 6 directional changes between the onset of P and the beginning of ventricular contraction. Possible causes of these movements have been considered.

The study of the precordial reflections of atrial motion would appear to have only very limited diagnostic value. Such motions may possibly be of aid in deciding whether mitral stenosis is dynamically significant, and in differentiating between mitral stenosis and cor pulmonale. At present, they are of little or no aid in distinguishing mitral stenosis from insufficiency, or from left ventricular failure.

**Summario in Interlingua**

Le movimentos precordiale que se initia inter le declaration del unda P e le declaration del unda Q in le electrocardiogramma esesse studiate in un gruppo de subjectos normal e in patientes con vari typos de morbo cardiac. Durante le mentionate portion del ciclo cardiac, registrationes ab le dextere region parasternal pareva resultar in constatazione plus regular que registrationes ab le region al sinistra del sterno. Un sequentia de movimentos consistente de proverso-retroverso-proverso esesse observate regularmente. Iste movimentos es clarmente de origine atrial, proque illos esesse absente
MOVEMENTS AND FORCES OF THE HUMAN HEART

in pacientes con fibrilación atrial e esseva observate post isolate undas P in cata un de tres subjectos con bloco cardiaco.

Circa 0,05 secundas post le declaration del unda P, un micre movimento dextroverse e proverbse se manifestava. Iste movimento non excedeva un quarto del amplitude total del cyclo cardiaco in ulle del subjectos normal. Illo eseva plus grande que le normal amplitude medie in cata un de 7 subjectos con corde pulmonal, e in 5 de istes illo eseva foras del limits normal.

Circa 0,10 secundas post le declaration del unda P, un movimento retroverse e sinistroverse se manifestava. Isto eseva exaggerate in ultra del limits normal in le majoritate del 30 registrationes ab patientes con stenosis mitral e in 7 inter 10 registrationes ab subjectos con “pur” insufficiency mitral. Iste movimento es ascribe principalmente a contraction del atrio sinistre. Proque le atrio es fixate posteriormente per le venas pulmonar illo pare traher le corde in retro. Viste que le movimento es a vices biphasic, il es probable que relaxation del atrio dextere es etiam un factor contributori.

Patientes con varie typos de morbo myocardic exhibi o non exhibi exaggeraciones del un o del altere o de ambes de iste movimentos. Ergo il pare que le exaggeration de iste movimentos atrial es relationate (1) a un effortio directe in le atrio correspondente, causate per un lesion valvular, (2) a un effortio indirecte causate per morbo o excesso de carga in le ventriculo correspondente.

A parte iste 2 movimentos que es ascribe al contraction successive del 2 atrios, le registrationes exhibi un secunde tiro in alto, comenciante justo ante o post le declaration de QRS. Iste movimento es ascribe tentativalemente a relaxation del atrio sinistre, sed illo non pote esser studiate in detal proque illo es tosto interrupmite per le plus marcate movimentos que es causate per le contraction ventricular.


Le movimentos del interspatios axillar, registrate per le technica cinetocardiographic, es complexe e exhibi usualmente 6 alterationes directional inter le declaration de P e le comenciamento del contraction ventricular. Causas possibile de iste movimentos es discutite.

Il pare que le studio del reflexiones precordial de movimento atrial possede solmente un limitate valor diagnostic. Il es possibile que iste movimentos se prova utile in determinar si stenosis mitral es dynamically significa- e in diferenciate inter stenosis mitral e corde pulmonal. Al tempore presente, illos es de paue o nulle adjuta in distinguere stenosis mitral ab insufficentia o stenosis mitral ab disfallimento ventricular.

REFERENCES
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