Kinetocardiographic Findings of Myocardial Infarction

By Soon Kyu Suh, M.D., and E. E. Eddeleman, Jr., M.D.

A study of the precordial pulsations (kinetocardiograms) in patients with acute myocardial infarctions is presented. A paradoxic pulsation (bulge) over either the precordium or the epigastric area was noted in all patients studied.

Abnormal pulsations of the chest wall due to myocardial aneurysms have been previously described1-4 and emphasized by Dressler and Pfeiffer5 and Vakil.6 However, these pulsations have not been systematically evaluated by objective methods. In addition, it is not certain whether such pulsations are due to actual anatomic aneurysms or "functional" bulging of the ventricular wall. The fact that the heart may have paradoxic pulsations in myocardial infarction has been well established.7-11 This communication presents a study of graphically recorded precordial movements (kinetocardiograms) from patients with myocardial infarctions.

Patients

Forty-two male patients with clinically and electrocardiographically proved myocardial infarctions were studied. Their ages varied from 30 to 71 years, with an average age of 51. Of the 23 patients with anterior infarctions* the lesion was acute in 17, old in 3, and of uncertain age in 3. The posterior infarctions were acute in 12 and old in 7. The 10 patients with old infarctions (including anterior as well as posterior) had clinical pictures on hospital admission suggestive of a recent lesion that could not be substantiated by serial electrocardiograms or serum transaminase levels.

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*Anterior, anteroseptal, anterior lateral infarctions were grouped together, since no significant differences were encountered in the kinetocardiograms.

Technics

Kinetocardiograms (low-frequency displacement precordial movements) were recorded with the technic previously described.12 However, the crossbar was modified so that it could be attached directly to the bed. Thus records could be readily obtained without discomfort to the patient. Tracings were secured from positions over the precordium corresponding to the standard electrocardiographic V leads and designated as Ks, K1, K2, etc. Tracings from other areas were labeled with a second numeral to indicate the intercostal space. Thus, K13 represents the record from the right parasternal position in the third intercostal space, and K35 the tracing from the V5 position in the fifth intercostal space, etc. Records from points just inferior to the costal margins in the right and left midclavicular lines and just below the xiphoid process were labeled KER, KEL, and KEM, respectively. The electrocardiogram, heart sounds, and the carotid pulse were recorded simultaneously with the kinetocardiogram with use of a Sanborn Polyviso instrument at a paper speed of 50 mm. per second. Serial tracings were obtained with the same sensitivity of the recording apparatus as on the initial examination in order to determine variations in amplitude. Nine patients were studied every other day for a week and then at weekly intervals during hospitalization. Tracings were obtained from the remaining patients at weekly intervals, since it became apparent that more frequent examinations were unnecessary.

Results

Configuration of the Abnormal Pulsations

The principal kinetocardiographic change due to myocardial infarction was an abnormal outward systolic movement* reaching a maximum amplitude (peaking) in early, mid, or late systole. The tracings were classified as

*Cornally the precordium retracts or moves inward during ejection; however, in the K1 position there is a middigystolic outward movement that rarely goes above the diastolic line.
“high bulges” when the amplitude of the paradoxic movement was well marked, and were classified as “low bulges” when the amplitude was small. The separation of the tracings into high and low bulges is arbitrary and the differences are probably due to the many factors that can modify the absolute amplitude of precordial pulsations. These patterns are illustrated in figure 1 and the data are summarized in table 1.

A paradoxic movement of the precordium was observed in all 42 patients sometime during the 3 or 4 weeks of study. In 4 of the patients a “bulge” appeared subsequent to the initial tracing. Bulges of large amplitude (high bulges) were noted in 18 of 23 patients (78 per cent) with anterior myocardial infarctions and in 10 of 19 patients (53 per cent) with posterior infarctions. Bulges that peaked during early or late systole (fig. 1) occurred more frequently in patients with anterior myocardial infarction, whereas a prominent mid-systolic movement was more common in those patients with posterior infarctions. The significance of this variation is unknown.

### Location of the Paradoxic Movements

The distribution and point of maximum amplitude of the “bulges” are illustrated in figure 2 and tabulated in table 2. The “high bulges” associated with infarctions extended from the left parasternal line to the anterior axillary line, while those associated with posterior infarctions were more frequently present in the mid, left, or right epigastric areas.

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**Fig. 1.** The 4 types of paradoxic pulsations encountered in patients with myocardial infarctions. An upward curve on the tracing indicates an outward movement of the precordium, while a downward curve represents an inward motion. The first arrow indicates onset of the QRS complex of the electrocardiogram. The second arrow indicates the onset of ejection as determined by the carotid pulse, and the third arrow indicates the incisural notch in the carotid pulse. In A, the first type, the peak of the outward movement occurs in early systole or shortly after the onset of ejection (second arrow). In B the bulge peaks in midsystole; in C it peaks in late systole or just before the carotid incisural notch. Tracing D is a low bulge, with small amplitude; nevertheless, there is an outward systolic movement throughout the entire ejection period. The difference in D from A, B, and C is probably of no significance, as it may represent only an amplitude variation.

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**Table 1.** The Incidence of the “‘Bulge’” According to the Electrocardiographic Pattern

<table>
<thead>
<tr>
<th>EKG</th>
<th>KCG*</th>
<th>Early systolic</th>
<th>Mid-systolic</th>
<th>Late systolic</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior infarction</td>
<td>High</td>
<td>8</td>
<td>2</td>
<td>8</td>
<td>18</td>
</tr>
<tr>
<td>Posterior infarction</td>
<td>High</td>
<td>4</td>
<td>6</td>
<td>3</td>
<td>13</td>
</tr>
<tr>
<td></td>
<td>Low</td>
<td>2</td>
<td>4</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>16</td>
<td>15</td>
<td>11</td>
<td>42</td>
</tr>
</tbody>
</table>

*Kinetocardiogram.

**Table 2.** The Location of Maximum Amplitude of the Bulges

<table>
<thead>
<tr>
<th>Anterior infarction</th>
<th>Posterior infarction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>High</td>
</tr>
<tr>
<td>Ke</td>
<td>1</td>
</tr>
<tr>
<td>Kern, Kern, Kern</td>
<td>15</td>
</tr>
<tr>
<td>Kp</td>
<td>0</td>
</tr>
<tr>
<td>Ke</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>18</td>
</tr>
</tbody>
</table>

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However, all patients with posterior infarctions, with 1 exception, had paradoxic pulsations over a precordial as well. The point where the bulge was of maximum amplitude in anterior infarctions was the K₃ position in 15 of the 18 patients (83 per cent) and was variable in the other 5 patients. The location of the "bulges" associated with posterior infarctions was variable. However, in contrast to anterior infarctions, only 1 patient with a posterior infarction exhibited a bulge of maximum amplitude at the K₃₄ point.

The upstroke of the bulges began from 0.00 to 0.18 second after the onset of the QRS complex of the electrocardiogram. The early onset at the same time as that of the QRS complex may be due to the fusion of the wave with the movements due to atrial contraction.

**KINETOCARDIOGRAM IN MYOCARDIAL INFARCTION**

**ANTERIOR INFARCTION**

High systolic "bulge"

LVH

LVH

LVH

LVH

LVH

LVH

Low systolic "bulge"

LVH

LVH

LVH

LVH

LVH

LVH

**POSTERIOR INFARCTION**

High systolic "bulge"

LVH

LVH

LVH

LVH

LVH

LVH

Low systolic "bulge"

LVH

LVH

LVH

LVH

LVH

LVH

\[ k_{13}, k_{14}, k_{23}, k_{24}, k_{34}, k_{44}, k_{45}, k_5 \]

**Location of "bulge"**

- Maximum amplitude of "bulge"

LVH - Patients with left ventricular hypertrophy as well as myocardial infarction

![Diagram of Kinetocardiogram in Myocardial Infarction](http://circ.ahajournals.org/)

**Fig. 2.** The location and the maximum amplitude of the systolic bulges encountered in all of the patients with myocardial infarctions studied. K₃₁, K₃, K₃₂, K₄, etc., represent the points at which the kinetocardiograms were taken, the first digit referring to the "V" location and the second to the interspace. KEL refers to the left epigastrium, KEM the midepigastrium, and KER the right epigastrium. The line represents the total area where a paradoxical bulge was recorded, while the circle in the line represents the point at which the bulge was of maximum amplitude. In the anterior myocardial infarctions with high systolic bulges the maximum amplitude usually occurred at the K₃₄ point, while the point of maximum bulge in the posterior myocardial infarctions was considerably more variable.

**The Onset of the Myocardial Infarction as Related to the Kinetocardiographic Findings**

Paradoxic pulsations were recorded within 5 hours after the onset of the infarction (estimated from the onset of chest pain) in 6 patients; in 1 patient, however, the "bulge" was detected 1 hour after the onset of chest pain. In contrast, in 3 of the 17 patients with acute anterior infarctions the bulge first appeared 1 week after the initial tracing.

**Serial Kinetocardiographic Findings**

The kinetocardiographic changes observed during the 3 weeks of hospitalization are illustrated in figure 3 and tabulated in table 3.

No changes in the serial tracings were noted in 15 patients (39 per cent). In 13 patients there were variations in amplitude or duration
of the bulge, and in 9 the bulge completely disappeared (22 per cent). The bulge was absent on the initial tracing in the remaining 4 patients (10 per cent) but appeared on subsequent records. Control kinetocardiograms taken prior to the infarction were available in 2 patients, and in both instances the bulge appeared only after the episode of myocardial infarction.

**Findings on Palpation**

In the supine position a diffuse and forceful heaving pulsation of the chest wall was pal-
pated in 28 of the patients having large (high) kinetocardiographic bulges; however, the point of maximum pulsation was usually localized. In only 2 patients showing "low bulges" was it possible to palpate the abnormal pulsation. Thus, abnormal movements of the chest wall were palpable in 30 (71 per cent) of 42 patients, whereas the remaining 12 patients had bulges demonstrable only by the kinetocardiographic technic. Often the point of maximal pulsation was tender during the first few days of illness.

Clinical Observations

Serial changes in the kinetocardiograms were correlated with the clinical course of the patients and are tabulated in table 4.

Although 29 patients had uneventful hospitalizations, in only 6 did the abnormal bulge disappear on subsequent serial tracings. The bulge disappeared in 2 patients in whom the infarction was of an undetermined age. Of 9 patients with old infarctions the bulge persisted in 7, appeared in 1, and disappeared in 1. Three of these 9 patients had anginal episodes during hospitalization with persistent systolic bulges.

There were no significant differences in the serial kinetocardiographic changes in patients with either anterior or posterior infarctions. Also there was no correlation between the kinetocardiographic changes and the laboratory findings.

The fact that the bulges often changed or even disappeared in patients with left ventricular hypertrophy indicates that the systolic outward movements were not due to left ventricular hypertrophy per se.

Kinetocardiographic and Autopsy Findings

Autopsy findings were available in 4 patients in whom kinetocardiographic traces were taken.

Patient 1. The patient was a 53-year-old man who had a typical clinical history with electrocardiographic changes of an acute anterior myocardial infarction. The patient died on the thirty-sixth day of hospitalization as the result of intractable left-sided congestive heart failure. The patient had a history of hypertension but there was no evidence of valvular heart disease. At autopsy the heart weighed 710 Gm. and both ventricular chambers were markedly dilated. There was a large mural thrombus in the apex and anterior wall of the left ventricle, next to the interventricular septum, underlying very thin myocardial muscle. Somewhat laterally and posteriorly on the left ventricle near the left atrium was a large patch of fibrinous material measuring approximately 6 cm. in diameter, which suggested an additional infarcted area. No definite myocardial aneurysm was noted. The infarcted areas appeared to be of fairly recent occurrence. Cross sections of the left coronary artery revealed complete occlusion of the left anterior descending branch approximately 2 cm. distal to the bifurcation.

Kinetocardiograms from this patient revealed a bulge with prominent early systolic
TABLE 4.—Clinical Course and Kinetocardiographic Changes

<table>
<thead>
<tr>
<th>Stage of infarction by EKG</th>
<th>Clinical course and EKG changes</th>
<th>Systolic bulge</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No change</td>
<td>Decreased in amplitude</td>
</tr>
<tr>
<td></td>
<td>Favorable course</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td>Persisting ST elevation</td>
<td>0</td>
</tr>
<tr>
<td>Acute</td>
<td>Angina pectoris after infarction</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Death due to acute left ventricular failure</td>
<td>0</td>
</tr>
<tr>
<td>Intermediate</td>
<td>Favorable course without EKG changes</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Favorable course</td>
<td>1</td>
</tr>
<tr>
<td>Old</td>
<td>Angina pectoris after infarction</td>
<td>2</td>
</tr>
</tbody>
</table>

Out of 4 autopsy cases, 1 patient died after the first kinetocardiographic examination and is not included in the above data. One other patient, who had a favorable course during the first 4 weeks of hospitalization, is included above under "favorable course," although he died 6 months after admission.

outward movement from K23 to K45 with maximum amplitude at K34. In addition, the atrial waves were accentuated (fig. 4). Thus, the kinetocardiograms revealed a bulge of the precordium overlying the area of infarction, with a paradoxical pulsation during systole, although there was no demonstrable aneurysm present at autopsy.

Patient 2. This 69-year-old man was admitted with the usual clinical history, and electrocardiograms revealed an acute anterior infarction. There was a previous history of angina pectoris and hypertension for a duration of 8 years. The patient died 6 months later during an episode of acute pulmonary edema. At autopsy the heart weighed 600 Gm. The mid and anterior portion of the interventricular septum revealed an old thick scar which extended up to the right ventricular wall. On the anterior portion of the left ventricle just adjacent to the infarcted area of the septum was a large myocardial aneurysm. There was essentially no ventricular muscle in this area, and anteriorly the aneurysm was attached to the pericardium. The aneurysm measured approximately 7.5 cm. in diameter and was coated everywhere by mural thrombus; however, it was not filled with clotted blood. Cross sections of the coronary arteries revealed old and recent thrombi, occluding the left anterior descending coronary artery. There was marked atherosclerosis of the right coronary artery with calcification, and in addition it was almost occluded.

The kinetocardiogram revealed a late systolic outward movement from K23 to K5 with the maximum amplitude in K45 (fig. 5). In this instance the paradoxical pulsation of the
kinetocardiogram occurred as the result of a true ventricular aneurysm, and the location of the bulge recorded correlated well with the anatomic aneurysm.

Patient 3. This patient was a 49-year-old man who had typical clinical and electrocardiographic findings of an acute posterior myocardial infarction. The patient died on the eleventh hospital day following the onset of shock and left ventricular failure. Postmortem examination revealed the heart to weigh 500 Gm. The coronary arteries were markedly atherosclerotic, and there was a complete occlusion at a point 3 cm. from the origin of the right coronary artery. The left ventricle throughout had an extensive yellowish discoloration of its walls. There was an extensive recent infarction of the posterior wall (diaphragmatic) of the left ventricle extending up to the base of the heart.

The kinetocardiograms revealed a typical high systolic outward movement in the epigastric tracings (KEM and KER) (fig. 6). This illustrates an instance when a posterior

![Image](http://circ.ahajournals.org/)

**Fig. 4.** Kinetocardiograms from the first autopsied patient. The tracings are aligned so that the arrows apply to the lower tracings as well. The area of infarction as determined at autopsy was in the region of the apex and the anterior wall of the left ventricle just adjacent to the interventricular septum. There was a marked outward systolic bulge of the anterior precordium from K33 to K45. This represents an anterior and apical infarction in which the paradoxic systolic bulge recorded from the precordial chest wall corresponded well to the area of infarcted myocardium.
infarction produced paradoxic pulsations in
the epigastrium. In addition this probably
represents a bulge that is functional and not
anatomic, as there was no demonstrable an-
erysm of the myocardium at autopsy.

**Patient 4.** This patient was a 61-year-old
man. One month prior to admission the pa-
tient had an anterior myocardial infarction.
Frequent episodes of angina pectoris
prompted the admission. One week later the
patient had a sudden onset of very severe
chest pain; just before death the electrocar-
diogram revealed an acute posterior myocar-
dial infarction. The patient died a few hours
later in acute pulmonary edema. At autopsy
the heart weighed 470 Gm. The posterior two
thirds of the septum was softened and yellow-
ish in color. In addition, there was a patchy
softening and yellowish discoloration of the
subendocardial aspect of the posterior wall of
the left ventricle. Both old and recent myo-
cardial infarctions were present. The coro-
nary arteries were sclerotic, and there was
considerable narrowing of all arteries. The
right coronary artery at the point where it
curves over the base of the ventricle was
completely occluded by hemorrhage within an
atheromatous plaque. The left descending
coronary artery and the left circumflex artery
were sclerotic but there were no occlusions.

Kinetocardiograms did not reveal any defi-
nite bulge in the tracings taken prior to the
onset of the severe pain, but the kinetocardi-
ograms 1 week prior to death revealed an in-
creased systolic outward movement in the
KEL area and a new bulge at the K₃₄ point.
However, at this time there were no electro-
cardiographic changes (fig. 7). This patient
possibly illustrates an example in which the
"bulge" developed before electrocardiograph-

![Fig. 5. Kinetocardiogram taken prior to death from the second autopsied patient. This represents an instance of a true myocardial aneurysm in the lower anterior region of the left ventricle adjacent to the interventricular septum. The kinetocardiogram revealed a systolic bulge in the areas K₂₄, K₃₄, K₄₄, and K₅. This is an example of a paradoxic precordial bulge due to an underlying anatomic aneurysm.](image-url)
ic changes became manifested. In addition, the finding of a bulge of the precordium with no demonstrable anterior infarction indicates that septal infarctions may produce a paradoxical pulsation of the precordium, possibly due to the bulging of the septum anteriorly into the right ventricle.

DISCUSSION

The similarity of the previously described palpable precordial pulsations due to myocardial infarctions\(^5\) to those recorded in the present study is impressive. It emphasizes the accuracy of careful clinical observations and confirms the validity of these studies.

There has been little doubt of the existence of ventricular aneurysms. There are numerous pathologic reports, and in addition systolic paradoxical bulging of the myocardium has been experimentally demonstrated by the ligation of the descending branch of the coronary artery.\(^12\) The incidence of true aneurysms due to myocardial infarction had been reported from 9 per cent\(^14\) to 38 per cent\(^3\); however, paradoxic pulsations have been noted to occur in 74 per cent of patients with recent myocardial infarctions and in 75 per cent of patients with old myocardial infarctions, as studied by the roentgenokymograph.\(^5\) The demonstration that 100 per cent of the patients in our series had precordial or epigastric bulges was unexpected. It was anticipated that anterior infarctions would be more easily detected by the kinetocardiographic technic, whereas posterior infarctions might be better recorded with the electrokymograph.
or roentgenokymograph. The fact that posterior infarctions were as frequently recorded with this technic as anterior infarctions can possibly be explained in part by the locations of the infarcts. Anatomically myocardial infarctions do not correspond very well with the electrocardiographic areas. The interven-

tricular septum may be involved in anterior as well as posterior infarctions. The diaphragmatic area of the left ventricle is more commonly involved in posterior infarctions, and, since the epigastric area faces the diaphragmatic surface of the heart, abnormal pulsations should have been detected in this area. As the apex may be involved in posterior infarctions in addition to the septum, paradoxic pulsations obviously should be detected anteriorly. However, the only apparent explanation for the paradoxic pulsations of the parasternal areas of the chest in posterior myocardial infarctions is that the involved septum bulges into the right ventricle. High posterior infarctions are difficult to recognize electrocardiographically and none was encountered in the present series.* It would seem, however, that high posterior wall infarctions should be as silent kinetocardiographically as electrocardiographically; however, this remains to be proved.

Although it is generally accepted that an infarcted area of myocardial muscle will show a paradoxic pulsation during systole, the present study emphasizes the validity of this concept. Three of the 4 subjects in whom autopsies were performed did not have an anatomic aneurysm present, although there were demonstrable paradoxic pulsations on the kinetocardiograms. The kinetocardiogram does not differentiate between anatomic and "functional" aneurysms, except in those instances in which the bulge disappeared during observation. These therefore can be assumed to be "functional" rather than anatomic. The clinical significance of myocardial aneurysms and "functional" myocardial aneurysms remains obscure. It has been stated that the presence of a myocardial aneurysm does not alter the clinical course, although instances associated with intractable heart failure have been noted. Since this was an acute study, no statement could be made as to the eventual prognosis, although only 22 per cent of the paradoxic pulsations disappeared during hos-

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*It is possible that some patients were excluded who had high posterior infarctions, since only those proved by electrocardiogram or serum transaminase levels were included in this study.
KINETOCARDIOGRAM IN MYOCARDIAL INFARCTION

Hospitalization. It is tempting to postulate that these 22 per cent had some recovery of muscular function to account for the disappearance of the paradoxical pulsations, and therefore one would think that they might fair better clinically. Paradoxical pulsations can be noted very early after clinical onset of the infarction, as one was recorded 1 hour after the onset of pain.

As only proved myocardial infarctions were studied in the present series, the value of this technic for diagnosis is still unknown, and whether it will be superior or comparable to the electrocardiogram is similarly unknown. However, instances were encountered in which a bulge was detected before electrocardiographic changes occurred. Previously it has been reported from this laboratory that paradoxical pulsations can occur during attacks of angina pectoris, and then disappear after the alleviation of the pain. Therefore it would appear that this technic should be useful in differentiating status anginosus (coronary insufficiency) from myocardial infarctions. Any bulge present after the cessation of pain would suggest a persistent infarcted area of myocardial muscle. That the bulge did not disappear during hospitalization in 78 per cent of the patients and that no bulge disappeared within the first week of observation suggest that a persistent bulge indicates an infarction, although the age of the infarct cannot be determined kinetocardiographically. A bulge also may be present at rest in some patients with angina pectoris. Whether the bulge in these instances is due to an old myocardial infarction or a chronically ischemic area of muscle is unknown.

The value of precordial palpation in the clinical examination of the patient must be emphasized again. When a paradoxical pulsation can be felt in as many as 71 per cent of the infarctions, its clinical value is obviously important. With some practice the diagnosis of myocardial infarction can often be made at the bedside or at least suspected in many instances without the electrocardiogram. In addition, the observation that the bulges were often tender to palpation is of interest. This appears to be a valid finding and not related to psychogenic factors, as the tender point always occurred at the place of the maximum bulge and not in the surrounding areas, such as on the adjacent ribs. However, the tenderness lasted only during the very acute phase of the infarction.

There are some possible difficulties in evaluating paradoxical pulsations by palpation. The apical thrust due to the presence of left ventricular hypertrophy may simulate a bulge, as the thrust is usually a very forceful outward movement, and sustained throughout systole. However, the apex impulse of left ventricular hypertrophy is usually localized in only 1 intercostal space, and usually outside the V4 position, whereas the paradoxical bulges in myocardial infarction usually occur over most of the precordium, with the maximum impulse more frequently occurring at the V4 area, or well inside the midclavicular line. Although the bulge due to an infarction usually has a point of maximum impulse, in general it is more diffuse in its location. However, if the infarct is confined only to the apical area of the heart, it is quite possible that clinical differentiation cannot be made. Occasionally the bulges due to myocardial infarction may be so diffusely located and so forceful that they may be confused with the anterior precordial "heave" associated with right ventricular hypertrophy. In this instance other clinical information usually offers sufficient evidence for differentiation.

Summary

Forty-two patients with clinically proved myocardial infarctions were studied with the kinetocardiographic technic (low-frequency precordial motions).

Aneurysmal bulges (paradoxical outward motions) were recorded in all patients either over the precordium or in the epigastric region, including both posterior and anterior myocardial infarctions. The point of maximum bulge was usually at the V3 area in anterior myocardial infarctions.

Serial studies of the patients with acute myocardial infarction revealed that the bulge persisted throughout hospitalization in 78 per cent of the patients, including the 10 per cent
who had no bulge on first examination but who showed bulges on subsequent records. In only 22 per cent of the patients did the bulge disappear during the period of hospitalization.

The abnormal paradoxic pulsations were palpable in 71 per cent of the patients studied.

Autopsy findings from 4 patients revealed that the abnormal paradoxic pulsation of the chest wall corresponded anatomically rather well to the location of the myocardial infarction and could have occurred either as the result of a definite myocardial aneurysm or of paradoxic pulsations of the myocardium without definite anatomical aneurysm.

**SUMMARIO IN INTERLINGUA**

Quaranta-duo patientes con clinicamente demonstrate infarcimento myocardial esseva studiate per medio del technica cinetocardio-graphic (motiones precordial a bass frequen-
tia).

Extrusiones aneurysmal (paradoxe motiones extrorse) esseva registrate in omne patientes—supra le precordio o in le region epigastric, in posterior e etiam anterior infarcimento myocardial. Le puncto del extrusion maximal esseva usualmente al area de V₃ in anterior infarcimento myocardial.

Studios serial del patientes con acute infarcimento myocardial revelava que le extrus-

ION persisteva durante le integre periodo de hospitalisation in 78 pro cento del casos. Iste cifra includo le 10 pro cento del patientes in qui le extrusion non esseva presente in le pri-

me registration sed appareva in registrations subsequente. Le extrusion disparea in le curso del hospitalisation in solmente 22 pro cento del patientes.

Le anormal pulsationes paradoxe esseva palpabile in 71 pro cento del patientes stu-

diate.

Constatationes necroptic in quatro casos revelava que le anormal pulsationes paradoxe in le pariete thoracic correspondeva anatomicamente satis ben al sito del infarcimento myo-

cardial. Los occurrentia pote esser explicate como resultato de un definite aneurysmo myo-

cardial o de pulsationes paradoxe del myocardio sin definite aneurysmo anatomic.

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