Portable Serial Roentgenkymography in Acute Myocardial Infarction

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Serial roentgenkymographic records were taken at the bedside of 31 patients with acute myocardial infarction. Most such tracings are adequate for determining "ballooning" (systolic reversal) and other defects of left ventricular contraction and expansion. These abnormalities are observed frequently in this condition and fluctuate from day to day, appearing or disappearing at various rates. Ballooning is the most serious defect, in that it was observed in the roentgenkymograms of all the patients who died and in all those with severe shock. Serial roentgenkymograms are of value as diagnostic aids, as evidence of the immediate and changing functional state of the myocardium and as an index of prognosis. They may herald further extension of the infarct or ventricular rupture. Roentgenkymographic abnormalities, including ballooning, may persist after clinical recovery.

Certain abnormalities in ventricular movement, both in systole and diastole, have been observed during the course of myocardial infarction by fluoroscopic, roentgenkymographic, and electrokymographic means. The systolic abnormalities consist of (a) delay in the onset or rate of normal systolic retraction of the ventricular wall, (b) marked diminution or absence of retraction waves, and (c) actual reversal of the usual retraction and the occurrence of expansion ("ballooning").

The diastolic abnormalities are seen usually only in those cases in which there is an abnormality of the systolic contraction curve. They consist either of no expansion of the ventricular wall or of paradoxical retraction for part or all of the diastolic period.

These abnormalities, although they may occur in various other types of cardiac disorders, are most often associated with myocardial infarction. This is because diminution or reversal of retraction of the ventricular wall in systole occurs most often in a myocardium weakened by necrosis or ischemia, or both. The seriousness of systolic reversal is shown by the fact that if sufficiently severe and extensive, it can lead to failure of cardiac output and, possibly, to death. It may, however, be present without signs or symptoms of heart failure.

Although these characteristic patterns have been observed frequently in myocardial infarction, we know of no studies based on frequent serial roentgenkymographic records and their correlation with the clinical status of the patients. The purpose of this paper is to report on 31 patients suffering from acute myocardial infarction who were studied by means of bedside roentgenkymograms taken during the first 72 hours of their illness and subsequently every 24 to 48 hours for 5 to 10 days and less frequently until their death or hospital discharge. The data obtained were classified to determine if the abnormalities observed by roentgenkymographic means could be correlated with the clinical status of the patients and if they carried any diagnostic and, especially, any prognostic significance.

Materials and Methods

The study consisted of 31 patients with electrocardiographic and clinical evidence of myocardial infarction. Twenty-three were males and 8 females; their ages ranged from 43 to 87 years. The location of the infarct as established by electrocardiograms was as follows: anteroseptal, 10 cases; anterolateral, 9 cases; posterolateral, 4 cases; posterior, 8 cases. Nine of the patients had had previous myocardial infarcts; 22 had not.
The cases were classified as to degree of clinical severity as follows:

"Mild." No evidence of shock or heart failure and no serious complications, 6 cases.

"Mild failure." Transient pulmonary basal rales, 7 cases.

"Moderate to severe." Persistent rales and other evidence of serious involvement (recurrent severe pain, oliguria, nausea and vomiting, transient blood pressure levels below 90 mm. Hg systolic, tachycardia of greater than 110 beats per minute, rectal temperature elevation over 38 C., and persistent arrhythmias), 8 cases.

"Shock." Hypotension for at least 45 minutes with systolic blood pressure below 90 mm. Hg, 10 cases.

Roentgenkymographic tracings were taken within 24 hours of the onset of myocardial infarction in 21 cases and within 72 hours in the remaining 10. Successive records were made daily for 4 or 5 days in 25 cases and on at least alternate days in the remaining 6. In 27 instances additional films were taken every 3 to 7 days until death (4 patients) or discharge.

Roentgenkymographs* were taken in the anteroposterior projection, except in a few instances when left anterior oblique exposures were made. Dack and associates21,22 and others, have found that anteroposterior exposures reveal abnormal contractions with great consistency, regardless of the site of the lesion in the left ventricle. The tracings were made with the patient in a semierect sitting position in bed, leaning back against the smooth surface of the roentgenkymograph. The roentgen tube, supported by a bracket on a horizontal rod attached to a mobile vertical standard, was placed anteriorly to the patient's chest. In general, bedside roentgenkymography produces no untoward emotional reactions in the patient. The procedure is contraindicated, however, in certain patients in severe shock who cannot be maintained in the semierect position without further fall of blood pressure.

The roentgenkymographic tracings were studied by photographing the x-ray film on a standard 8.3 by 10.2 cm. lantern slide, and then projecting the photograph on a screen 15 to 20 feet distant. The projections were enlarged three- or fourfold to produce complete cardiac cycles of 3 cm. in length. The representative tracings (fig. 4, 6 and 7) were made from such enlargements. Minor deviations in these tracings were disregarded and only major directional patterns were recognized in the interpretations.

For simplicity of presentation, roentgenkymographic abnormalities of the left heart border were grouped in arbitrary order of descending severity as: type 1, "systolic reversal," i.e., expansion (or ballooning) of the major portion of the systolic period; type 2, "systolic and diastolic abnormalities," i.e., delayed initial systolic contraction or late systolic expansion, or partial diastolic collapse or mesial motion of the border during some portion of diastole; and type 3, "flattened," i.e., a flattened pattern and poorly defined waves in systole and diastole. Calculation of the percentage of abnormalities was based on the occurrence sometime during the course of illness of the three types of roentgenkymographic abnormalities. If two or more abnormal forms were observed in the same patient's records, however, only the "dominant" or presumptively most significant defect was entered in the figures.

These abnormalities were also grouped by location as high ventricular, midventricular and low ventricular or apical.

Results

Roentgenkymographic abnormalities of the left cardiac border (fig. 1) were observed in 24 (77 per cent) of the 31 cases at some time during the course of acute myocardial infarction. Systolic reversal, with or without other abnormalities, was noted in 16 (52 per cent) of the patients; other systolic and diastolic abnormalities, but not complete systolic reversal or flattening, in 11 (35 per cent); and flattening, with or without other deviations, with the exception of major systolic reversal (ballooning), in 7 (23 per cent). In the majority of the cases these changes appeared early in the course of infarction; 70 per cent of the patients examined within the first 24 hours after an attack showed some roentgenkymographic deviation.

Of the 27 patients who survived, 13 (48 per cent) had normal roentgenkymograms at the time of discharge from the hospital (fig. 1). The records of six of these patients had shown abnormalities previously; in the remaining seven, the roentgenkymograms had been normal at all times. In 14 cases (52 per cent) the

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* The apparatus used was a Liebel-Flarsheim model K3 multiple slit kymograph with slits at 12 mm. distance and the film moving at determined rates behind the slits. The portable generator (Picker Co.) delivered from 30 ma. at 100 v to 60 ma. at 220 v. The distance from the tube target to the skin varied from 63 cm. to 76 cm., and to the film approximately 110 cm. The output was from 30 to 60 ma./sec. at 80 to 90 kv. A 1 mm. aluminum filter was used. This dosage of radiation is considered free of hazard to the patient for as many as 20 records taken within three weeks.
Fig. 1. Frequency of occurrence of roentgenkymographic abnormalities in myocardial infarction. The first set of columns indicates the appearance of an abnormality alone, or in combination with other abnormalities, at some time during the course of illness in the 31 patients studied. Abnormalities are listed as Sys. Rev., extensive systolic expansion or ballooning, S/D Abn., other types of abnormal forms in the systolic or diastolic curves, and Fl., flattening of the entire pattern. The cases total more than 100 per cent, since different abnormalities may appear in the same patient at different times.

The final records still showed deviations: local ballooning in 5 (18 per cent), flattening in 7 (26 per cent) and other abnormalities in 2 (7 per cent). In no case was there fluoroscopic or roentgenographic evidence of massive paradoxical motion or local distention of the ventricle suggesting ventricular aneurysm.

The 5 patients whose final records showed localized systolic reversal (fig. 1) had no cardiac enlargement or evidence of heart failure at the time of discharge, although other observers have noted their occurrence several months after clinical recovery from attack in patients with apparently normal cardiac function. The followup of the surviving patients in our group, however, was not long enough to determine the eventual course in such cases.

It is possible that local ballooning may have been present at the onset of the current attack in those patients with histories of previous infarction. Ballooning was noted in the first roentgenkymograms in 7 of the 9 patients with such histories, but in only 6 of the 22 who had no known previous attack. It is apparent that defects of myocardial contractions, especially systolic reversal, are more prevalent and that mortality is higher after successive attacks.

The frequency of the three types of abnormalities according to the position of the heart border and the presumptive site of the infarct as determined by electrocardiogram is shown in figure 2. The high percentage of normal curves in the group with anteroseptal infarcts suggests that lesions in the mesial position do not affect a large enough area of the myocardium to alter the left heart border. However, when the lesion is so extensive that its effect extends to the left border of the heart, ballooning of a wide area occurs. Also, less distinctive forms of abnormal tracings than ballooning are produced when the myocardium is less seriously impaired. Posterolateral infarcts (inferior surface), as might be expected, involve the apex uniformly and thus may alter myocardial function at both high and low levels, depending on their location.

A close relationship exists between the clinical status of the patient and the incidence and frequency of roentgenkymographic abnormalities. In our patients the incidence of all abnormalities, particularly systolic reversal, was proportionate to the clinical evidence of the severity of the infarct. Systolic reversal was not observed in patients who had "mild" attacks, but was noted in each of the four patients who died (fig. 1).

Experiments on animals have shown that the duration of dominant abnormalities of
contraction in the areas surrounding a myocardial infarction varies according to the degree and duration of hypoxia and the load of work of the heart. In 17 of our 31 cases adequate roentgenkymograms were obtained on the first day and with sufficient frequency thereafter to show (1) the rate of change from normal patterns or minor abnormalities to systolic reversal, (2) the recession of a systolic reversal to a lesser abnormality and (3) the recovery from all abnormalities and return to a normal pattern.

Systolic reversal appeared abruptly within one day in 4 patients, 3 of whom died; in a fifth case it appeared slowly. In six patients, all of whom recovered, the ballooning disappeared, either rapidly or slowly; apparently the muscle had partially or completely regained contractile function at the time of discharge from the hospital. In a third group of six patients who exhibited no abnormalities at the time of discharge, the complete recession of abnormalities took place slowly.

**ILLUSTRATIVE CASE HISTORIES**

The following case histories serve to illustrate certain features, namely:

1. The diagnostic value of records taken shortly after the onset of an attack.
2. The heralding, by changing patterns of the records on the preceding day, of the clinical and electrocardiographic evidence of an extension of the infarction.
3. The transient appearance of “ballooning” accompanying mild shock.
4. The heralding of ventricular rupture by a broadening of the area of abnormal contraction without the warning of clinical signs or symptoms.
5. The effect of elevation of blood pressure by L-norepinephrine on the pattern of myocardial contraction.

For purposes of comparison, the roentgenkymographic tracings in a normal man, aged 35, and the tracing of enlarged frames are shown in figures 3 and 4. The onset of systole as timed from the onset of aortic expansion is indicated by horizontal lines.

In two cases, the records of which are not presented here, the patients had persistent precordial pain for two and five hours, respectively, before the attack of acute myocardial infarction, but no electrocardiographic evidence of infarction for 36 and 70 hours, respectively. Systolic reversal was noted in each patient within 24 hours of the onset of pain; this finding pointed toward the diagnosis of an infarct.

**Case 1.** W. L., male, aged 48 years, with no previous history of heart disease, had a characteristic attack of precordial compressing pain on the first day of his illness and electrocardiographic evidence of a posterolateral infarction on the second day. A
roentgenkymogram taken on the second day, 20 hours after the onset of pain, showed flattening of the apical contraction. Another roentgenkymogram taken on the eleventh day revealed ballooning (fig. 5). Figure 6 is an assembly of the enlarged tracing of one cardiac cycle starting with the aortic-timed onset of systole on the second, third, fifth, seventh and fourteenth days.

On the seventh day the patient was more pallid and sweated heavily; his blood pressure fell slightly. On this day, early systole exhibited an expansile form (fig. 6, frame 10). On the eighth day a recurrence of severe persistent pain necessitated a subcutaneous dose of 100 mg. of Demerol, which relieved the pain in 2 hours. On the ninth day the patient's temperature rose, and an electrocardiogram indicated an extension of the infarction further anteriorly (inversions of T waves in leads V 5-6).

Roentgenkymograms taken on the ninth and eleventh days showed systolic expansion in the mid and lower ventricle (fig. 5, frames 12-15, and fig. 6, frames 10-16), as timed with aortic expansion (fig. 6, frames 4 and 5). By the fourteenth day systolic contraction had returned (fig. 6), but was irregular (frame 10) and delayed (frames 15 and 16). The patient made an uneventful recovery and was discharged from the hospital on the twenty-eighth day.

This case illustrates that modification of myocardial contraction is probably associated with diminishing local coronary blood supply and that it may be accompanied by indefinite clinical signs that precede the characteristic onset of a secondary infarct as indicated by prolonged pain and electrocardiographic changes.

**Case 2.** J. P., male, aged 41, who had hypertension for the previous 7 years and angina pectoris for 16 months, developed severe, persistent substernal pain lasting 6 hours, which was partially controlled by subcutaneous injections of morphine. An electrocardiogram revealed a pattern characteristic of posterolateral infarct. Enlarged tracings from roentgenkymograms taken on the first, third, fourth and thirteenth days are shown in figure 7. On the first day the only definite abnormality was a shortened systolic contraction (frame 10). On the third day when mild shock appeared, ballooning was evidenced (frames 10 and 11) and flattening at the apex (frame 12). Twenty-four hours later (fourth day) early systolic contraction had returned in the midventricle, but flattening persisted in the low ventricle. By the thirteenth day systolic contraction,
although imperfect, was evident throughout the left cardiac border. The patient made an uneventful recovery and was discharged, ambulatory, on the twenty-fourth day.

This case illustrates the transiency of ballooning in local areas during mild shock and probable dependence on the adequacy of coronary blood supply and myocardial oxygenation.

Case 3. S. G., female, aged 70, who had shown no previous signs or symptoms of cardiac disorder, developed severe substernal pain radiating to the throat and back, which subsided in 4 hours without medication. During the next three days she had two or three episodes of spontaneous pain, lasting from 5 to 20 minutes. An electrocardiogram taken at this time revealed a high anterolateral infarct. A roentgenkymogram showed ballooning of the upper left ventricle on the first and fourth days and systolic reversal of the lower ventricular wall on the fourth day. At that time the patient felt quite well and appeared free of shock or heart failure. On the fifth day she died abruptly; an autopsy revealed rupture of the high posterolateral wall of the left ventricle.

This case illustrates the localization of a high left ventricular lesion and the possible prognostic significance of a widening of the area of systolic expansion in a patient who exhibited no other signs of impending ventricular rupture.

Case 4. M. H., male, aged 70, had angina pectoris for two and one-half years prior to the onset of severe left precordial pain radiating to the left arm. Three intramuscular doses of 100 mg. of Demerol within 4 hours were required to control the pain. An electrocardiogram was consistent with posterior myocardial infarction. Roentgenkymograms showed marked flattening of the waves at the apex from the first day to the morning of the fourth day. On the fourth day the patient's blood pressure fell abruptly from 106/72 to 72/40 mm. Hg. and he appeared to be in shock. L-norepinephrine, in a concentration of 8 mg./L. of 5 per cent glucose solution, was administered by intravenous drip infusion for 18 hours. The blood pressure rose promptly and was maintained by this therapy at levels over 100/64 mm. Hg. A roentgenkymogram taken one hour after the start of this therapy showed large, deep systolic contractions through the entire ventricular wall. A tracing taken on the eighth day showed a delayed systolic contraction high in the left ventricle, but the apical region maintained systolic contraction. In later records the patterns were approximately normal. The patient made an uneventful recovery.

This case illustrates the improvement of ventricular contraction that accompanies elevation of blood pressure and therefore more effective coronary arterial circulation. In addition, this change reflects the direct action of L-norepinephrine on myocardial contraction.

A similar case of myocardial infarction with severe shock was treated with L-norepinephrine. Continuous drip infusion maintained the blood pressure at satisfactory levels for 10 hours, at which time the patient abruptly died. The roentgenkymograms in this case showed increased amplitude of aortic expansion, but apical and midventricular flattening of curves was not modified. This suggests that inability of the myocardium to recover contractile quality despite adequate maintenance of blood pressure indicates a poor prognosis.

Summary and Conclusions

1. Roentgenkymographic tracings were made at the bedside of 31 patients with myocardial infarction. Although interpretation is sometimes difficult, such records are usually adequate for determining ballooning (systolic reversal) and other defects of left ventricular contraction and expansion.

2. Roentgenkymographic abnormalities were manifested by 77 per cent of the patients at some time during the course of the illness. Extensive ballooning during the systolic period was observed in 52 per cent, systolic and diastolic abnormalities in 35 per cent and flattening of the systolic and diastolic excursions in 23 per cent.

3. Defective ventricular contraction curves are sufficiently common during the early stages of attacks to serve as a diagnostic sign, but the absence of such an abnormality in a roentgenkymogram does not exclude myocardial infarction. Seventy per cent of the patients in our series exhibited roentgenkymographic abnormalities during the first 24 hours of the attack.

4. In man, as has been observed in experimental animals, the changes in the contraction patterns may occur abruptly and fluctuate from day to day, frequently without accompanying clinical or electrocardiographic manifestations.

5. In our series impaired contraction seemed
to herald the advancing ischemia prior to a secondary infarction in one case and to rupture of the ventricle in another.

6. Presumptive improvement of coronary circulation by relieving severe hypotension with L-norepinephrine resulted in improved contraction in one patient who recovered, but had no observable effect in a second patient who subsequently died.

7. Although there is no absolute correlation between circulatory failure and ballooning, a high incidence of ballooning was found in the roentgenkymograms of patients with shock and other evidence of a severe attack.

8. Ballooning may occur without shock, but none of the patients who died and none with shock failed to exhibit this defect.

9. Previous infarction increased the tendency toward ballooning.

10. The roentgenkymograms of 18 per cent of the patients discharged as clinically free of signs of circulatory failure exhibited ballooning and 50 per cent showed some type of roentgenkymographic abnormality. One may assume that hypoxia or physical stress would be a hazard to patients manifesting this evidence of myocardial dysfunction.

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SUMMARIO IN INTERLINGUA

1. Registrationes roentgenokymographic esseva obtenite al lecto del patientes in 31 casos de infarimento myocardial. Ben que le interpretation es a vices difficile, usualmente tal registrationes es adequate pro determinar ballonamento (reversion systolic) e altere defectos del contraction e expansion sinistro-ventricular.

2. Anormalitates roentgenokymographic esseva manifestate per 77 pro cento del patientes a un o altere tempore in le curso del maladia. Un ballonamento extense durante le periodo systolic esseva observate in 52 pro cento del casos, anormalitates systolic e diastolic in 35 pro cento, e applattamento del excursiones systolic e diastolic in 23 pro cento.

3. Defective curvas de contraction ventricular es sufficientemente commun durante le prime phases del attaccos pro servir como signos diagnostic, sed le absentia de un tal anormalitate ab le roentgenkymogramma non exclude le possibilitate de infarimento myocardial. Septanta pro cento del patientes in nostre serie exhibiva anormalitates roentgenokymographic durante le prime 24 horas del attaco.

4. De accordo con observationes in animales experimental, le alterationes del figuration contractional in humanos potre occurrer abruptemente. Illo potre fluctuar ab un die al altere, frequentemente sin accompaniamento de manifestationes clinic e electrocardiographic.

5. In nostre serie, defectivitate del contraction pareva annunciar le progresso de ischemia ante le formation de un infarimento in un caso e ante le ruptura del ventriculo in un altere.

6. Le melioration (presumptive) del circulation coronari effeicute per le uso de L-norepinephrina in le alleviation de sever hypotension resultava in contraction meliorate in un patiente qui recuperava, sed illo habeva nulle effecto observabile in un altere patiente qui moriva subsequentemente.

7. Ben que il non existe un absolute correlation inter disfallimento circulatori e ballonamento, un alte frequentia de ballonamento esseva trovate in le roentgenokymogrammas de patientes con choc e altere signos de un attaco sever.

8. Ballonamento pote occurrer sin choc, sed omne le patientes con choc e omnes qui moriva habeva ballonamento.


10. Le roentgenokymogrammas de 18 pro cento del patientes dimittite como clinicamente libere de signs de disfallimento circulatori exhibiva le phenomeno del ballonamento, e 50 cento monstrava le un o le altere typo de anormalitate roentgenokymographic. Il es a
suponer que hypoxia o stress physic esserea un hasardo pro patientes qui manifesta este signo de dysfunction myocardial.

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Portage Serial Roentgenkymography in Acute Myocardial Infarction
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