The Pathogenesis of Spontaneous Cardiac Rupture

By Stanford Wessler, M.D., Paul M. Zoll, M.D., and Monroe J. Schlesinger, M.D.

Twenty patients with cardiac rupture were compared with suitable control groups. In each ruptured heart there was an acute coronary artery occlusion and a recent, transmural myocardial infarction which was unprotected at the site of rupture by scar or anastomotic circulation. Clinically, rupture usually occurred during the first two weeks of an acute infarction in a hypertensive patient with no previous history of old infarction or congestive failure; persisting hypertension or excessive effort usually preceded rupture. The role of an increased intraventricular pressure in the pathogenesis of cardiac rupture has therapeutic implications.

SUDDEN DEATH from rupture of the heart is so dramatic an event that it has interested both clinicians and pathologists since the time of Harvey.1 Despite an extensive literature its exact causation is still not completely clear. New data concerning the pathogenesis of cardiac rupture are presented in this paper.

The term myocardial rupture usually denotes a complete, acquired defect through the muscular wall of one or more cardiac chambers. It, therefore, includes perforation of the interventricular or interauricular septum but does not refer to incomplete myocardial tear or rupture of the coronary arteries, coronary veins, cardiac valves, papillary muscles, chordae tendineae or aorta. In the absence of extracardiac causes rupture is said to be spontaneous.

METHODS AND MATERIALS

Between 1936 and 1950 a total of 1641 autopsies were performed at the Beth Israel Hospital, Boston, in which the hearts were adequately studied by a special technic of injection plus dissection of the coronary arteries.2 Briefly this method consisted of (1) injecting a radioopaque lead-agar mass of different colors into the main coronary arteries, (2) unrolling the heart so that its entire arterial tree lay in one plane, (3) taking a roentgenogram of the unrolled heart, and (4) carefully dissecting the coronary arteries, using the film as a guide. With the aid of this special technic precise data concerning occlusions and narrowings in the coronary arteries and interarterial anastomotic pathways were obtained. The methods by which such occlusions, narrowings and interarterial anastomoses were determined have been described previously.3

In this series of 1641 injected hearts 19 instances of spontaneous myocardial rupture were found. In 14 of the ruptured hearts, multiple, carefully localized histologic sections of the myocardium were also available. Myocardial fibrosis and recent infarction were graded in these sections as absent, slight, moderate or marked. A twentieth ruptured heart that came from another hospital* was studied in the same way and is included in this report. The method of study and the technic used to record the data are illustrated in figures 1, 2A, 2B. In every one of these 20 ruptured hearts a recent myocardial infarct was found. Therefore, a similarly studied, control group of 104 hearts with fresh infarcts in which rupture did not occur was examined for comparative purposes. The clinical records of these 124 patients and of 104 additional patients who survived an episode of acute myocardial infarction were also carefully reviewed.

PATHOLOGIC OBSERVATIONS

1. Incidence of Rupture. The entire series of 1641 injected hearts contained 281 histologically proven acute infarcts including the 19 with rupture. Thus, the incidence of rup-

* This heart was given to us for study by Dr. David Skinner, pathologist of the Newton-Wellesley Hospital. It was not included in the analysis of incidence of cardiac rupture at the Beth Israel Hospital.
ture was 1.2 per cent in the autopsy population and 7 per cent in the group with acute infarction.

2. Location and Size of Rupture. The ruptures were limited to the left ventricle and septum (fig. 1); six ruptures were through the anterior and nine through the posterior left ventricular wall; of five interventricular septal ruptures, one was anterior and four were posterior. The ruptures were extremely variable in size and shape on both epicardial and endocardial surfaces. Some were ragged, gaping holes, some linear slits and still others exhibited a V-shaped external opening. The tears on the epicardial surface were easily found and varied in length from 3 to 30 mm. The endocardial origin of the ruptures were often small and hidden behind trabeculae carnea; frequently they were demonstrated only after careful probing.

3. Heart Weight. The weights of the hearts in this series are presented in table 1.* No significant difference in weight distribution was found between the hearts with and without rupture.

4. Heart Group. The relative lengths of the right and left coronary arteries vary and form the basis for a classification of human hearts.† Rupture of the heart was not found to be related to this anatomic pattern of the coronary arteries (table 2).

5. Coronary Artery Occlusion. Old coronary artery occlusions antedating the acute infarct were present in only one third of the ruptured hearts but were found in three-fourths of the nonruptured controls. Furthermore, in hearts with old occlusions there were less than half as many old occlusions per heart among the ruptured hearts as among the control hearts (table 2). Thus, the incidence of old coronary artery occlusions was significantly less among the ruptured hearts than in the control group.

Concomitant with this infrequency of old coronary artery occlusions was a high incidence of fresh occlusions in hearts with rupture. In every one of the 20 ruptured hearts there was a fresh occlusion in the coronary artery system; on the other hand fresh occlusions were present in only half of the nonruptured controls (table 2).

6. Fibrosis. As might be expected from the infrequency of old coronary artery occlusions, there was little myocardial fibrosis among the ruptured hearts. Areas of marked fibrosis were not found in any of the 15 ruptured hearts with multiple sections, and moderate myocardial fibrosis was found in only one instance. In contrast, two thirds of the control hearts showed either marked or moderate fibrosis (table 2). Furthermore, in all 15 ruptured hearts areas that were entirely free of fibrosis were found within the infarcted zone; similar unscarred areas were found within the infarcts in only one third of the nonruptured hearts.

A myocardial aneurysm was not found among the group with rupture, but was present in 5 of the 104 control hearts.

7. Size and Shape of Fresh Infarction. The recent infarcts varied considerably in size in both the hearts with and without rupture; there was no significant difference between the two groups.

The transmural path of rupture, however, was not usually confined to a zone of infarction but traversed some noninfarcted myocardium. In every ruptured heart with multiple sections fresh infarction was found to ex-
PATHOGENESIS OF SPONTANEOUS CARDIAC RUPTURE

<table>
<thead>
<tr>
<th>Table 2.—Pathologic Findings in 124 Hearts with Acute Infarction (80 with Rupture; 104 without Rupture)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart Group</td>
</tr>
<tr>
<td>I (Right coronary preponderance)</td>
</tr>
<tr>
<td>Rupture (20 Cases)</td>
</tr>
<tr>
<td>Control (104 Cases)</td>
</tr>
<tr>
<td>Number of hearts with old occlusions</td>
</tr>
<tr>
<td>Number of old occlusions</td>
</tr>
<tr>
<td>Number of hearts with fresh occlusions</td>
</tr>
<tr>
<td>*Amount of myocardial fibrosis</td>
</tr>
<tr>
<td>absent</td>
</tr>
<tr>
<td>1+</td>
</tr>
<tr>
<td>2+</td>
</tr>
<tr>
<td>3+</td>
</tr>
<tr>
<td>*Area of acute infarction without fibrosis</td>
</tr>
<tr>
<td>Myocardial aneurysm</td>
</tr>
<tr>
<td>*Size of acute infarct</td>
</tr>
<tr>
<td>1+</td>
</tr>
<tr>
<td>2+</td>
</tr>
<tr>
<td>3+</td>
</tr>
<tr>
<td>*Transmural infarct</td>
</tr>
<tr>
<td>Pericarditis</td>
</tr>
<tr>
<td>Mural thrombi</td>
</tr>
<tr>
<td>Intercoronary anastomoses</td>
</tr>
<tr>
<td>Collateral circulation poor within infarct</td>
</tr>
</tbody>
</table>

* Based on 15 of 20 ruptured hearts in which multiple microscopic sections were available.

tend across from endocardial to epicardial surface in one or more places, even though most of the infarction was not usually transmural. In only one third of the hearts without rupture were the fresh infarcts transmural (table 2).

The age of infarction was estimated in each available microscopic section according to the criteria of Mallory, White and Salcedo-Salgar. Although many sections were taken, the age of every zone of infarction could be determined adequately in only 8 of the 15 ruptured hearts with multiple sections. In all eight the acute infarction was found to vary in age in different areas. This is also true of most fresh myocardial infarcts in which multiple sections are studied. In these eight hearts the path of the rupture always traversed the area of most recent infarction that was also entirely free of fibrosis. In six of these eight carefully studied ruptured hearts the tear occurred at the junction of the most recently infarcted muscle and histologically normal myocardium.

8. Pericarditis and Mural Thrombi. Together with the transmural extension of the infarcts, the ruptured hearts more often presented a fibrinous pericarditis or a mural thrombus than did the hearts with nonruptured infarcts (table 2). Neither process apparently afforded protection against rupture.

9. Collateral Circulation. Intercoronary coronary anastomoses were significantly less frequent in the 20 ruptured hearts than in the 104 control hearts (table 2).

In all 20 ruptured hearts direct or anastomotic filling of the vessels with injection mass was incomplete or entirely absent within the area of fresh infarction, a condition found in only a third of the control group. In every instance the rupture occurred through such
an uninjected, presumably avascular zone. Mass injected into the coronary arteries did not leak from the torn edges of the ruptured myocardium in any instance. All the blood found in the pericardial sac after cardiac rupture must, therefore, have come from the chamber of the left ventricle or from torn coronary veins or capillaries rather than from coronary arteries.

10. Mechanism of Death. Major, commonly fatal pathologic conditions in addition to fresh infarction were present in 42 per cent of the 104 control patients (table 3). These included advanced valvular disease, multiple fresh occlusions in different coronary arteries, uremia, pneumonia, pulmonary infarction, intestinal obstruction, bowel gangrene, and massive hemorrhage into the brain, thorax or abdomen. These conditions may themselves have caused or hastened death before evolution of the infarct had been completed. Such an accessory lethal condition was found in only one (5 per cent) of the 20 cases with rupture (table 3).

11. The Pathologic Substrate for Rupture. These comparisons between acute myocardial infarcts that ruptured and those that did not rupture delineate the pathologic conditions common to all ruptured hearts. These are:

(1) a fresh coronary artery occlusion
(2) a recent myocardial infarct
(3) a myocardial infarct which is transmural
(4) a myocardial infarct which is poorly supplied with collateral vessels.
(5) a myocardial infarct in which fibrosis is entirely absent in at least one area.

These five factors form the common denominator or pathologic substrate of spontaneous cardiac rupture.*

In this study there were 115 consecutive hearts with acute myocardial infarction in which adequate multiple microscopic sections were available. Among these 115 hearts were 22 with the pathologic substrate for rupture; rupture occurred in 11. The remaining 11 may be regarded as hearts that might have ruptured. Eight of these 11 cases showed other accessory lethal factors which may have themselves caused or hastened death. Thus, only three cases (cases 63, 65 and 89) in the "potential" rupture group were pathologically

---

* The five ruptured hearts that lacked multiple, tagged, microscopic sections did fulfill all the criteria that could be determined in them.

---

Fig. 2A, B. Roentgenogram and diagram of ruptured heart (case 16). "0" indicates absence of both fibrosis and necrosis. Slight fibrosis (1+ on scale of 0 to 5+) was found as indicated in only one of the 27 microscopic sections. Acute infarction, found in six sections (A-F) was graded in extent for each section (on scale of 0 to 5+). The age of the acute infarction varied from less than 1 to 10 days with section F showing the most recent infarction—all less than 24 hours' duration.

<table>
<thead>
<tr>
<th>Section</th>
<th>Age Range of Infarct (Days)</th>
<th>Amount of Infarcted Muscle</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>10-2</td>
<td>2+</td>
</tr>
<tr>
<td>B</td>
<td>10-2</td>
<td>4+</td>
</tr>
<tr>
<td>C</td>
<td>10-2</td>
<td>4+</td>
</tr>
<tr>
<td>D</td>
<td>10-1</td>
<td>3+</td>
</tr>
<tr>
<td>E</td>
<td>10-1</td>
<td>5+</td>
</tr>
<tr>
<td>F</td>
<td>&lt;1</td>
<td>1+</td>
</tr>
</tbody>
</table>

indistinguishable from the group with rupture; death occurred in these three patients, four, five and eight days after the onset of acute infarction. On the basis of the pathologic data alone rupture might have been expected in
these three hearts. An explanation for its absence must therefore be sought among the clinical phenomena occurring in patients with acute myocardial infarction.

CLINICAL OBSERVATIONS

The clinical data in all 124 patients, 20 with cardiac rupture and 104 controls with non-ruptured acute infarcts, were reviewed in detail for distinctive features which might forewarn the clinician of impending rupture. In each instance the referring physician as well present if either the systolic or diastolic pressure was 150 or 90 mm. of mercury, respectively, or higher. The readings were made under varied conditions in the outpatient clinics, by private physicians or in the hospital; they were subject to the many influences that inevitably prevail under these variable circumstances. In the total series of 124 patients there were 13 in whom the usual blood pressure level could not be determined. These 13 equivocal cases, one of which occurred in the rupture group, were excluded from the present

Table 5.—Comparison of Clinical Findings in Patients with Acute Myocardial Infarction (1) Who Died with Cardiac Rupture, (2) Who Survived, (3) Who Died without Rupture

<table>
<thead>
<tr>
<th>Prior to Terminal Infarction</th>
<th>During Terminal Infarction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angina Pectoris</td>
<td>Hypertension (during last 24 hours of life)</td>
</tr>
<tr>
<td>Congestive Heart Failure</td>
<td>Hypertension (1)</td>
</tr>
<tr>
<td>Old Myocardial Infarction</td>
<td>(2)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>(3)</td>
</tr>
</tbody>
</table>

* (20 Cases)

<table>
<thead>
<tr>
<th>Acute Infarction with Rupture (+... -...</th>
<th>10 (50%) 19 20 12 (63%) 9</th>
</tr>
</thead>
</table>

* (101 Cases)

<table>
<thead>
<tr>
<th>Acute Infarction with Survival (+... -...</th>
<th>46 (44%) 94 81 56 (55%) 16 (16%)</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Acute Infarction with Death but no Rupture (+... -...</th>
<th>67 (68%) 34 (34%) 24 (25%) 16 (17%) 22 (21%)</th>
</tr>
</thead>
</table>

* Totals do not equal 20 or 104 in all categories because of instances in which factors were not determined.

† Effort in this group of survivors refers to unusual effort at any time during the first three weeks of acute infarction.

as the hospital record were consulted in order to check carefully the clinical details.

1. **Age.** The ages of the patients with and without rupture were listed in decades (table 4). No differences between the two groups are apparent.

2. **Sex.** Among the 20 patients with rupture, 11 were men and 9 women; among the 104 control patients without rupture there were three times as many men as women. The relatively high incidence of women among patients with cardiac rupture is not statistically significant in this small series.

3. **Hypertension.** For purposes of this study, arterial hypertension was considered to be analysis. Hypertension was present in all of the remaining patients with rupture and in only two thirds of the control patients (table 5). This difference is significant. It was found, moreover, that the blood pressure remained elevated after onset of the terminal acute infarct in two thirds of the patients with rupture and in only one sixth of the control cases (table 5). This higher incidence of persistent hypertension during infarction in the group with rupture is also statistically significant.

4. **Congestive Heart Failure.** In considering the incidence of congestive failure prior to the terminal infarction, patients with congestive failure of less than one month's duration were
omitted in order to eliminate those in whom the congestive failure may have been an associated symptom of the terminal infarction. The presence or absence of congestive heart failure was satisfactorily established on clinical and laboratory grounds in all but 5 of the 124 patients. These five patients, all in the control group, were omitted from this analysis. Congestive failure was present in only one of the 20 patients with rupture but was found in one third of the control patients (table 5). This difference is highly significant statistically.

5. Angina Pectoris. In evaluating the incidence of angina pectoris prior to the terminal infarction, patients with angina pectoris of short duration (less than one month before death) were omitted from the analysis in order to eliminate instances in which angina pectoris might have been entirely a prodromal manifestation of the terminal myocardial infarction. The presence or absence of anginal pain was clearly determined in all but six of the control group. Angina pectoris was present prior to the terminal infarction in half of the patients with rupture and in two thirds of the control patients (table 5). This difference between the two groups is not significant.

6. Clinical Episode of Old Infarction. The clinical diagnosis of myocardial infarction was accepted only after careful evaluation of the history, physical findings, clinical course and laboratory data which included leukocyte counts, erythrocyte sedimentation rates and electrocardiograms. A myocardial infarction was considered old or healed if it occurred more than two months before death. In eight of the control patients the presence or absence of a clinical episode of old myocardial infarction could not be determined with certainty. None of the 20 patients with rupture had a history of old infarction whereas it was present in one fourth of the control patients (table 5). This difference is probably significant.

7. Effort during Terminal Infarction. The amount of effort expended by a patient is difficult to quantitate clinically. The usual activities, physical and emotional, attendant upon enforced bed rest frequently produce excessive expenditures of energy. It is also true that occasional strenuous exertions by hospitalized patients are not always recorded or called to the attention of the professional staff. Furthermore, the duration of effort, its time of occurrence and associated circumstances may be of critical importance for its effect in producing myocardial rupture. In the face of these difficulties it was decided to limit analysis of this factor to "unusual" effort during the last 24 hours of life. Although use of the bedpan is associated with increased effort7,8 and although one of the patients with cardiac rupture actually expired on the bedpan, such activity was not included as unusual effort since it was so common among most patients hospitalized for acute infarction. Patients who were psychotic or sufficiently disturbed to require restraints or heavy sedation, patients who refused to stay in bed and those who were put through strenuous diagnostic procedures when the diagnosis of infarction had not been entertained, were considered to have experienced "unusual" effort.

In 12 of the 124 patients the presence or absence of unusual effort in the last 24 hours of life could not be determined; all 12 were in the control group. Sixty-one per cent of the patients with rupture and 24 per cent of the control patients experienced unusual effort (table 5). This is a significant difference. Of the 11 patients with rupture in whom unusual effort was noted within 24 hours of death, five were disturbed and psychotic, two refused to remain in bed, two were walking about, one had rapidly climbed three flights of stairs and one had undergone strenuous diagnostic tests during which marked elevations of the blood pressure had been recorded.

8. Antemortem Diagnosis of Acute Myocardial Infarction. In one patient from the rupture group and in seven control patients it was not clear whether the diagnosis of terminal acute infarction had been made during life. Acute myocardial infarction was diagnosed clinically in 90 per cent of the patients with rupture, but in only two thirds of the control cases.

In many of the 35 control cases with unrecognized myocardial infarction, the coronary heart disease was overshadowed by more prominent clinical conditions such as cerebral and gastrointestinal hemorrhage, pneumonia, uremia, dis-
secting aneurysm, surgical operations and pulmonary infarction. Furthermore, several of these clinically unnoticed infarcts were so small they were not recognized on gross pathologic examination but were found only after microscopic study of the myocardium.

9. Clinical Diagnosis of Rupture. Only 1 of the 15 ruptures through the left ventricular wall was diagnosed during life (case 14). In this instance the patient developed cardiac tamponade while being examined. Four of the five interventricular septal ruptures were correctly diagnosed or suspected during life on the basis of the sudden appearance of a loud systolic murmur after the onset of the infarction. The fifth patient (case 12) in whom the diagnosis of septal rupture was not suspected ante mortem had had a persistent loud rough apical systolic murmur for two years preceding the terminal illness. The duration of life from the recognition of the septal rupture to death varied from three hours (case 19) to nine days (case 6).

10. Clinical Duration of Myocardial Infarction. Among 19 patients with rupture in whom duration of the infarct could be determined, the interval from the clinical onset of infarction to death ranged from 2 to 21 days. Four-fifths of them expired between the fourth and eleventh days. In contrast, only one-third of the control patients died within this same time interval (fig. 3). This difference is statistically significant.

11. Fever during Terminal Infarction. The height and duration of fever was recorded sufficiently well in 87 of the 124 patients so that they could be classified into groups with absent or slight fever, moderate and high fever. No significant differences were found here between the patients with and without rupture.

12. Leukocytosis during Terminal Infarction. Leukocyte counts were recorded in 86 of the 124 patients. Although the determinations varied markedly in frequency from patient to patient, the 86 patients could be divided into three groups on the basis of the highest recorded counts: those with minimal or no elevation of the white count, those with moderate and those with marked leukocytosis. Here, also, no significant differences were noted between the patients with and without rupture.

13. Electrocardiogram during Terminal Infarction. Electrocardiographic tracings were recorded in 17 of the patients with rupture and in 81 of the control patients. The number of leads, and the number and timing of individual tracings varied considerably. Nevertheless, abnormal electrocardiograms were obtained in 97 of the 98 patients. Among the 17 patients with rupture the available electrocardiograms were consistent with a diagnosis of acute infarction in 14, abnormal but not diagnostic in two and entirely normal in one. The tracings were diagnostic of infarction in 50 of the control group and abnormal but not diagnostic in the rest. The incidence of diagnostic tracings was not significantly different in the two groups. The nondistinctive abnormalities in the two ruptured and the 31 control cases included variable atrioventricular block, bundle branch block, auricular fibrillation, auricular flutter, other arrhythmias and deviations of the S-T segments and T waves. In the one patient with a normal electrocardiogram (case 15) a normal 12 lead tracing was recorded seven days after the onset of the myocardial infarction and 48 hours before rupture. The roentgenogram and diagram of this patient’s heart are shown in figures 2A and 2B.

14. Drugs during Terminal Infarction. The role of pharmacologic agents in the precipitation of myocardial rupture is difficult to evaluate. A wide variety of potent drugs was given in many different ways. Large doses of digitalis substances, quinidine, benzedrine, ephedrine, caffeine, Coramine, plasma, intravenous glucose and water, intravenous epinephrine, paraldehyde, mercurial diuretics, 50 per cent glucose in water, strychnine (case 27), aminophylline, prostigmine (case 41), papaverine, atropine,
heparin, magnesium sulfate, testosterone (case 101) and dicumarol (bishydroxycoumarin) were given to many of these patients with myocardial infarction. The evidence is inadequate that any of these drugs caused rupture. Experimental studies in dogs have indicated that dicumarol produces no adverse pathologic effects in freshly infarcted myocardium; clinical reports also have not shown this anticoagulant to be associated with an increased incidence of rupture. In one patient there was a striking temporal relation between the administration of a drug and cardiac rupture. This patient (case 2) was not suspected of having a myocardial infarction and was given 0.3 cc. of Pitressin (vasopressin) subcutaneously for the successful relief of abdominal distention; she died suddenly 65 minutes later from myocardial rupture and cardiac tamponade. Although the dose of Pitressin given to this patient was less than that recommended as a test for coronary insufficiency, this drug is a profound coronary vasoconstrictor and has produced myocardial damage in animals. In man severe myocardial ischemia and sudden death have followed its administration.15, 16, 17

15. The Clinical “Profile” of the Patient with Cardiac Rupture. From these clinical data conclusions may be drawn concerning the type of patient in whom rupture is most likely to occur. Rupture usually develops between the fourth and eleventh day of an acute myocardial infarction in a hypertensive patient with no previous history of old myocardial infarction or congestive failure. The hypertension frequently persists during the period of infarction and “unusual” effort often occurs within 24 hours of rupture. Angina pectoris is equally common among patients with and without rupture. Rupture appears to occur as frequently in women as in men. Age, leukocytosis, fever and the use of cardiac drugs during the acute infarct bear no relation to the occurrence of rupture.

This clinical “profile” is limited in that it is based on a consideration only of patients who died during an episode of acute myocardial infarction. It does not appraise the likelihood of rupture among all patients with clinical acute myocardial infarction before they die. Accordingly, the clinical records of a third group of 104 consecutive patients who survived an episode of acute myocardial infarction were reviewed for analysis of the factors of hypertension, angina pectoris, old myocardial infarction, congestive failure and unusual effort (table 6).

Hypertension and angina pectoris preceded the acute infarction in half, old infarction in one fourth and congestive failure in one twelfth of those who survived. Elevated blood pressure persisting during the acute infarction was found in only 16 per cent of the patients. Unusual effort, as previously defined, was rare during the acute illness (table 5).

Hypertension and unusual effort were more frequent and old myocardial infarction was less common in the rupture group than among the other two groups. Since congestive failure, prior to the terminal infarction, was uncommon in the rupture group and in the survivors, patients who will die of rupture resemble closely patients who will survive such infarcts.

Discussion

The series of ruptured hearts presented here differs from others previously reported in that the coronary arteries and myocardium were studied with an adequate injection technique and with multiple labelled microscopic sections. In addition a large group of nonruptured control hearts was also examined by the same methods. Finally, the clinical data in the group of cases with rupture and in comparable groups without rupture were correlated with the pathologic observations.

This unique body of data has delineated a uniform pathologic substrate common to hearts with spontaneous myocardial rupture. This substrate consists of an acute coronary artery occlusion and a recent myocardial infarct which is transmural, poorly supplied by collateral circulation and in which fibrosis is entirely absent in at least one area. Regardless of the presence elsewhere in the heart of myocardial fibrosis or collateral circulation from previous coronary artery obstruction, these pathologic criteria select hearts which are totally unprotected by scar or anastomosis at the site of the acute infarction.
PATHOGENESIS OF SPONTANEOUS CARDIAC RUPTURE

Occasionally rupture may be produced by direct injury to the heart,25; rarely the cause may be indirect trauma,19, 20 blast,21 infection,22, 23, 24 or tumor.25 In most reported instances, however, and as in all our cases, rupture took place through an area of recent myocardial infarction. Contrary to general belief cardiac rupture secondary to recent myocardial infarction is not a pathologic curiosity. It occurred in 9 per cent of 2609 patients who died with acute infarction in 29 general hospitals (table 6).

Rupture was found by others as well as ourselves to occur through large and small infarcts.25, 22 When an infarct involves the full thickness of the wall, the muscle is said to break more easily as soon as granulation tissue appears.40 On the other hand ruptures have been reported through spotty infarcts and through infarcts which did not reach the endocardium.28 The path of such rupture may be determined in part by the arrangement of the muscle planes.41 Special histologic characteristics have not been described for infarcts which rupture; some are necrotic and heavily infiltrated with polymorphonuclear leukocytes whereas others show relatively little softening.28 Tedeschi42 observed the perforation of an abscess within an area of fresh infarction. It has been suggested that pericarditis occurring with an acute infarct may prevent or delay the onset of rupture.

Although myocardial rupture usually occurs within the first two weeks after the onset of infarction25, 34, 36, 37, 44–47 it has occasionally been observed in the third week.25, 36 The range in the present series was from 2 to 21 days. Mallory, White and Salcedo-Salgar4 believe that after the third week rupture is rare and usually the result of some complicating factor such as another fresh infarction.

Rupture has been reported through old infarcts42, 45, 49 and through myocardial aneurysms42, 44, 49–53. Such observations, however, are exceedingly uncommon. In several studies old infarcts and aneurysms were present in many hearts, but rupture occurred only through areas of fresh infarction.35, 31, 34, 45, 54–58 Furthermore, among three series of cardiac aneurysms 57–59 no myocardial rupture was reported. Finally, Edmondson and Hoxie28 noted that when scarring was present in the myocardium, the likelihood of rupture was only one-fourth as great as in unscarred hearts. These observations on the relation between rupture and acute and healed infarction are in accord with our own findings.

As in our series, rupture has been found usually to take place in a single tear through the left ventricle or less commonly through the interventricular septum. Rupture of the right ventricle does occur and has often been reported because of its relative infrequency.43, 50, 60–68 Atrial rupture also occurs50, 62, 63, 69–72 but is rare. Double ruptures perforating the interventricular septum and the wall of the left ventricle73, 74 or both ventricles61 have been observed; Morgagni75 described a heart with three discrete ventricular ruptures and Muller76 reported a septal rupture with nine complete perforations.

A fresh occlusion has usually been found when the coronary arteries of ruptured hearts have been examined44, 45, 51, 72, 73–75; it was found in all our hearts with rupture. Occasionally rupture has been reported to occur in hearts without a fresh coronary artery occlusion,41, 45 or even with normal coronary arteries.79, 81 Meticulous dissections of the un.injected coronary arterial tree, however, may uncover only about one half of the points of occlusion.82 One cannot, therefore, be completely certain that the coronary arteries are normal unless they have been injected and carefully dissected.

A positive correlation between myocardial rupture and hypertension was suggested by de la Chapelle.44 Later Edmondson and Hoxie28 conclusively demonstrated that hypertension persisting after the onset of a myocardial infarct increased significantly the likelihood of rupture. Others57, 67 have also recently emphasized the significance of hypertension.

The relation of exertion to acute myocardial infarction has been the subject of many recent communications.53–59 Physical effort has been ascribed a role in the pathogenesis of myocardial rupture by many investigators,11, 34, 37, 44, 47, 48, 50, 51, 72, 73 although denied by some.28, 92 Rupture has been described during epileptic seizures.93 There are many references to the
frequent occurrence of cardiac rupture in mentally disturbed patients. In such patients an acute myocardial infarction is cardiac infarction would seem to be a common factor responsible for the high incidence of rupture among these two groups of patients.

Table 7.—Comparative Incidence of Cardiac Rupture in Autopsy Populations from General Hospitals, Mental Institutions, and Coroners' Series

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Hospital</th>
<th>City</th>
<th>Number of Autopsies</th>
<th>Cardiac Rupture</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Number</td>
</tr>
<tr>
<td>1872-1925</td>
<td>Krumbhaar (48)</td>
<td>Philadelphia General Hospital</td>
<td>Philadelphia, Pa.</td>
<td>16,000</td>
<td>7</td>
</tr>
<tr>
<td>1922-25</td>
<td>de la Chapelle (44)</td>
<td>Bellevue Hospital</td>
<td>New York, N. Y.</td>
<td>1,525</td>
<td>4</td>
</tr>
<tr>
<td>1924-41</td>
<td>Edmondson (28)</td>
<td>Los Angeles County Hospital</td>
<td>Los Angeles, Calif.</td>
<td>25,000</td>
<td>72</td>
</tr>
<tr>
<td>1926-45</td>
<td>Wang (29)</td>
<td>Massachusetts General Hospital</td>
<td>Boston, Mass.</td>
<td>7,018</td>
<td>23</td>
</tr>
<tr>
<td>1931-48</td>
<td>Foord (31)</td>
<td>Huntington Memorial Hospital</td>
<td>Pasadena, Calif.</td>
<td>3,173</td>
<td>33</td>
</tr>
<tr>
<td>1935-40</td>
<td>Wartman (32)</td>
<td>Western Reserve Institute of Pathology</td>
<td>Cleveland, Ohio</td>
<td>2,000</td>
<td>7</td>
</tr>
<tr>
<td>1939-40</td>
<td>Diaz-Rivera (34)</td>
<td>Louisville General Hospital</td>
<td>Louisville, Ky.</td>
<td>1,250</td>
<td>5</td>
</tr>
<tr>
<td>1940-49</td>
<td>MacDonald (36)</td>
<td>St. Lukes Hospital</td>
<td>New York, N. Y.</td>
<td>1,636</td>
<td>8</td>
</tr>
<tr>
<td>1928</td>
<td>Buckley (111)</td>
<td>New Haven Hospital</td>
<td>New Haven, Conn.</td>
<td>1,330</td>
<td>3</td>
</tr>
<tr>
<td>1933</td>
<td>Benson (51)</td>
<td>Good Samaritan Hospital</td>
<td>Portland, Ore.</td>
<td>3,784</td>
<td>7</td>
</tr>
<tr>
<td>1948</td>
<td>Cleland (39)</td>
<td>Royal Adelaide Hospital</td>
<td>Adelaide, Australia</td>
<td>6,000</td>
<td>12</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Total of General Hospitals</td>
<td>68,716</td>
</tr>
<tr>
<td>1910-27</td>
<td>Stewart (104)</td>
<td>Warren State Hospital</td>
<td>Warren, Pa.</td>
<td>840</td>
<td>6</td>
</tr>
<tr>
<td>1920-30</td>
<td>Beresford (103)</td>
<td>Tooting Bee Hospital</td>
<td>England</td>
<td>2,374</td>
<td>31</td>
</tr>
<tr>
<td>1927-33</td>
<td>Peterson (105)</td>
<td>St. Peter State Hospital</td>
<td>St. Peter, Minn.</td>
<td>207</td>
<td>3</td>
</tr>
<tr>
<td>1935-39</td>
<td>Snyder (79)</td>
<td>Western State Hospital</td>
<td>Ft. Steilacoon, Wash.</td>
<td>490</td>
<td>9</td>
</tr>
<tr>
<td>1941</td>
<td>Simburg (107)</td>
<td>Brandon Hospital</td>
<td>Brandon, Canada</td>
<td>470</td>
<td>2</td>
</tr>
<tr>
<td>1948</td>
<td>Cleland (39)</td>
<td>Southern Australia Mental Hospitals</td>
<td>Australia</td>
<td>600</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Total of Mental Hospitals</td>
<td>4,981</td>
</tr>
<tr>
<td>1937-43</td>
<td>Rabson (110)</td>
<td>—</td>
<td>New York, N. Y.</td>
<td>2,030</td>
<td>24</td>
</tr>
<tr>
<td>1933</td>
<td>Benson (51)</td>
<td>—</td>
<td>Muninomah County, Ore.</td>
<td>2,112</td>
<td>27</td>
</tr>
<tr>
<td>1940</td>
<td>Martland (55)</td>
<td>—</td>
<td>Newark, N. J.</td>
<td>2,000</td>
<td>42</td>
</tr>
<tr>
<td>1948</td>
<td>Cleland (39)</td>
<td>—</td>
<td>Australia</td>
<td>250</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Total of Coroners' Cases</td>
<td>6,392</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1944</td>
<td>Jetter (108)</td>
<td>—</td>
<td>Massachusetts</td>
<td>115</td>
<td>16</td>
</tr>
</tbody>
</table>

frequently unsuspected clinically and the patient is usually ambulatory. Similarly, there is a high incidence of rupture among coroners' cases in whom antemortem diagnosis and bed rest are also uncommon. Severe physical effort after the onset of acute myo-

Cleland compared the incidence of cardiac rupture in necropies, all examined under the same auspices, from a general hospital, from mental institutions and from coroners' cases. He found a strikingly different incidence of cardiac rupture in the three groups: the lowest
was in the general hospital population, and the highest in the coroners' series. We have arranged 21 reported series comprising 337 cardiac ruptures in a total of 80,089 necropsies according to Cleland's classification. The percentage of rupture was 0.26 in the general hospitals, 1.1 in the mental institutions and 1.6 in the coroners' cases (table 7).

In comparison the number of ruptures in a special group reported by Jetter and White is of unusual interest. They found an incidence of cardiac rupture of 14 per cent in a consecutive series of 115 necropsies performed on patients who died suddenly and unexpectedly in Massachusetts psychiatric hospitals. Although frequently referred to as evidence of the high incidence of rupture in mental hospitals, this series is, in fact, a doubly selected group in which the additive effect of coroners' cases and psychiatric patients is dramatically apparent.

Our own data are in agreement with the observations that hypertension and effort play a role in the production of cardiac rupture. In our series, persistence of hypertension during the acute infarction or unusual effort during the last 24 hours of life were found in 15 of the 19 patients in whom these factors were determined. Among the remaining four, one had unusual effort 36 hours before death, and another three days before death. As mentioned earlier there were three hearts in the control group (cases 63, 65, 89) which were—aside from the absence of rupture—pathologically indistinguishable from the hearts with rupture. In not one of these instances was either sustained hypertension or unusual effort present during the acute infarction. On the other hand, several patients in the control series exhibited sustained hypertension or unusual effort without rupture; the hearts of these patients, however, did not show the appropriate pathologic substrate for rupture. The necessary conditions, both pathologic and clinical, must be present before rupture will occur.

More than a century ago Quain noted that the sexes were equally represented in his series of ruptured hearts. Since then others have noted a relatively high incidence of rupture among women. In general, however, relatively little attention has been given to the sex ratio among patients with rupture. We have reviewed several series of ruptured hearts from general hospitals but have excluded medical examiners' cases in which there is a marked preponderance of males. This tabulation (table 8) confirms the equal sex incidence originally noted by Quain. Since coronary disease and myocardial infarction are more common in men the fact that the incidence of rupture is equal among the two sexes indicates that rupture is more prone to occur in the female who has an acute myocardial infarction.

The reported differences between the sexes in the extent of coronary disease and the presence of hypertension assume significance because of the importance of these factors in the production of myocardial rupture. We have, therefore, reviewed 1018 unselected necropsies from the Beth Israel Hospital in which satisfactory injections of the coronary arteries were carried out. The sex, the number of hearts with old coronary artery occlusions, the number of occlusions per heart and the presence of hypertension, determined according to the criteria mentioned above, are presented in table 9. Coronary artery occlusions were significantly more common among men and hypertension significantly more common among women.

It has already been demonstrated that rupture tends to occur in the hypertensive patient with minimal previous coronary disease. Since women fit these requirements more often than men, the relatively high frequency of rupture in women may be caused, in part, by these factors. Males predominate in groups of patients with angina pectoris, myocardial infarction or myocardial aneurysm. To our knowledge cardiac rupture is the only manifestation of coronary disease in which the usual male preponderance does not obtain.

It has long been noted that rupture tends to occur among older people. Gradually, it has become clear that the age of patients with rupture parallels that for myocardial infarction. In a review of men who died from coronary artery disease, Yater found that rupture occurred more often in the older age groups. He
also found, however, that the incidence of hypertension increased with age in his series. Increasing hypertension, rather than coronary artery disease, may account for the higher incidence of ruptures among the older patients observed by him.

There is no fundamental difference in the pathogenesis of left ventricular and interventricular septal rupture. They have the same pathologic substrate and they appear in the same type of patient; their occurrence depends simply on the location of the infarct. Clinically, the important difference is in the mode of death; in the former exitus is rapid from cardiac tamponade; in septal rupture, death is less abrupt and usually results from congestive heart failure.

The antemortem diagnosis of ventricular wall rupture has rarely been made because of the rapidity of death. A fortuitous examination may reveal an unusual murmur or sudden cardiac tamponade (case 15) immediately prior to death. In contrast, the diagnosis of septal rupture can frequently be established or at least suspected ante mortem on the basis of a new loud systolic murmur and progressive congestive failure complicating the course of acute myocardial infarction. Patients may survive for days or even months following a complete septal tear; survival for five years has been reported. Intraventricular block has been observed in septal rupture, but the disturbance in conduction may have been due to the infarction per se rather than the septal perforation. Complete atrioventricular dissociation has not been reported and might not be expected to result from rupture, which usually involves the lower portion of the septum (fig. 1).

Angina pectoris, previous healed myocardial infarction and congestive failure all have been observed prior to the terminal infarction in patients with rupture. The comparative frequency and significance of these symptoms, however, have not been clearly analyzed. Since the hearts of patients with rupture revealed little old coronary artery obstruction and slight myocardial fibrosis, one might expect to find minimal evidence of angina pectoris, old infarction or congestive failure among these patients. All 20 patients with rupture met these expectations in that none showed a healed infarction and only one had congestive failure. In this one instance (case 13) the congestive failure was probably due to hypertensive heart disease since pathologic examination showed cardiac hypertrophy, very little myocardial fibrosis and no valvular disease. Angina pectoris, however, preceded the terminal infarction in half of the group. This incidence of angina was the same as that in the control group, although in the latter there was a significantly greater amount of coronary artery obstruction. Although angina pectoris was an expression of pre-existing heart disease, it resulted in the group with rupture from the combination of relatively little coronary disease and arterial hypertension. Therefore, angina was not a useful indication in this series of the extent of coronary obstruction or the likelihood of rupture.

Careful study of the selected microscopic sections in the ruptured hearts emphasized two facts: first, an acute myocardial infarct continues to change in extent for some time after its inception; and second, rupture ordinarily occurs through areas of more recent spread. Although the clinical picture indicates the onset of a myocardial infarction at a particular time, the entire infarct is not all of that same age. Infarction is a continuous and progressive process with the simultaneous repair of old areas, the development of fresh necrosis and the recovery of ischemic muscle. The occurrence of rupture through the more recently infarcted myocardium, which is continuously evolving, offers a basis for the wide time interval of three weeks after acute myocardial infarction during which rupture may occur.

Paradoxically, patients who die of rupture have even less evidence of serious heart disease, aside from hypertension, than patients who survive such an attack. Congestive failure and major, complicating pathologic states are both uncommon among patients with rupture. The catastrophe that abruptly terminates this relatively favorable picture may be anticipated in part by the ominous signs of antecedent and persistent hypertension, unusual effort and
the absence of previous infarction that would have caused the development of collateral circulation and fibrous tissue to buttress the walls of the heart. Patients such as those who develop rupture are often regarded as mild cases who are doing well and, therefore, do not need strict limitation of activities. It is in just this type of mild uncomplicated case in whom the blood pressure does not drop appreciably from the usual level and in whom rest is not rigorously enforced that rupture is likely to occur. The “sick coronary” with marked depression of the blood pressure level, with carefully supervised and prolonged bed rest because of complicating illnesses or gross congestive heart failure and with effective sedation for restlessness, dyspnea or recurrent chest pain is less likely to develop cardiac rupture and more prone to die of extensive infarction.

The hypothesis that a heightened intraventricular pressure is necessary to produce rupture of a necrotic myocardium is susceptible to experimental study in laboratory animals. Spontaneous cardiac rupture in animals is rare. Cardiac rupture after experimental ligation of a coronary artery is also quite unusual.

In a recent study involving injection of the coronary arteries and preparation of multiple microscopic sections, acute myocardial infarction was produced by acute coronary artery ligation in 31 dogs. Although most of these hearts showed the pathologic substrate for rupture, myocardial rupture did not occur. These dogs were permitted the routine activities of the animal farm but were not exercised or subjected to special strain postoperatively. Attempts are now being undertaken in this laboratory to produce rupture by inducing hypertension and subjecting animals to other types of strain following acute coronary artery ligation.

Persistent hypotension secondary to acute myocardial infarction is generally accepted as an ominous event that is at times treated successfully with pressor agents. Similarly, hypertension persisting during an acute infarct may eventually be considered a threat to life serious enough to justify the use of pharmacologic agents that depress the blood pressure to normotensive levels. Although such therapy cannot be recommended at this time, a precedent for its use may be found in the successful treatment of hypertensive crises and toxemias of pregnancy with the veratrum alkaloids.

For the present, rest and sedation in all patients with acute myocardial infarction will reduce cardiac work, perhaps lessen the extent of necrosis in ischemic areas and diminish the height of the intraventricular pressure. Avoidance of sudden or excessive exertion can be accomplished by cooperation of the patient, meticulous nursing care and conservative medical judgment. Since cardiac rupture accounts for almost one tenth of all deaths from acute myocardial infarction and occurs particularly in otherwise favorable cases, efforts to prevent rupture may increase appreciably the number of patients who make a good recovery from an initial myocardial infarction.

**SUMMARY AND CONCLUSIONS**

1. Twenty ruptured hearts were studied by a special technic consisting of injection and dissection of the coronary arteries and examination of multiple, carefully localized histologic sections of the myocardium. All 20 ruptures occurred through areas of acute myocardial infarction. A control series of 104 nonruptured hearts with acute infarction studied in the same way and the clinical records of 104 additional patients who survived an episode of acute myocardial infarction were examined for purposes of comparison. In addition, reports in the literature on cardiac rupture were reviewed.

2. The reported incidence of rupture among patients dying with acute myocardial infarction in general hospitals is approximately 9 per cent.

3. There is a uniform pathologic substrate common to hearts with spontaneous myocardial rupture. This substrate consists of an acute coronary artery occlusion and a recent myocardial infarction which is transmural, poorly supplied by collateral circulation and in which fibrosis is entirely absent in at least one area. These pathologic criteria select hearts which are totally unprotected by scar or anastomosis at the site of an acute myocardial infarction.

4. Concomitantly, there is significantly less
coronary artery occlusion and myocardial fibrosis among patients with acute infarction who die with rupture than among those who die without rupture.

5. There is likewise a "clinical profile" for the type of patient in whom rupture is most likely to occur. Rupture usually develops between the fourth and eleventh day of an acute myocardial infarction in a hypertensive patient with no previous history of old myocardial infarction or congestive failure. Persisting hypertension or excessive effort usually precede rupture.

6. Among patients with acute myocardial infarction rupture is relatively more common in women than men. This is due in part to the lesser extent of coronary disease and the greater incidence of hypertension among women.

7. Hearts that show the pathologic substrate for rupture may not perforate unless hypertension or excessive effort occurs during acute infarction; conversely, hypertension and excessive effort do not produce rupture if the appropriate cardiac pathology is absent.

8. Most acute myocardial infarctions change continuously in extent for some time after their inception, with simultaneous repair of old areas and involvement of new sites. The usual location of rupture through the more recently infarcted myocardium explains, in part, why rupture may occur almost any time during the first three weeks after the onset of necrosis.

9. Increased intraventricular pressure appears to be a critical factor in the development of cardiac rupture. Strenuous measures may, therefore, be justified to avoid or reduce arterial hypertension and effort in patients during acute myocardial infarction.

REFERENCES


3. —: Relation of anatomic pattern to pathologic conditions of the coronary arteries. Arch. Path. 30: 403, 1940.


22. Brandt, B.: Spontanruptur der tuberkulösen


40 —: A note on the musculature of the human heart as illustrated by pathological processes. M. J. Australia 1: 826, 1940.


54 Martland, H. S.: Sudden deaths with reference
to their prevention. Proc. New England Heart Assoc. 1940, P. 42.


86 BOAS, E. P.: Angina pectoris and cardiac infarction from trauma or unusual effort with a consideration of certain medicolegal aspects. J.A.M.A. 112: 1887, 1939.


92 SEGALL, H. N.: Rupture of ventricular myocard-


15 —: III. Diseases, etc., of the organs of circulation. IV. Spontaneous rupture of the heart. Tr. Path. Soc. London 50: 49, 1899.


17 BRUCE, J.: Case of rupture of the left ventricle of the heart. J. Ment. Sc. 38: 85, 1892.


32 BLAND, E. F.: Personal communication.

33 FOORD, A. G.: Personal communication.

34 SELZER, A.: Personal communication.

35 MACDONALD, G. E.: Personal communication.


37 LODGE-PATCH, I.: Personal communication.


The Pathogenesis of Spontaneous Cardiac Rupture
STANFORD WESSLER, PAUL M. ZOLL and MONROE J. SCHLESINGER

Circulation. 1952;6:334-351
doi: 10.1161/01.CIR.6.3.334
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1952 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/6/3/334

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/