FIVE CASES OF SPONTANEOUS RUPTURE OF THE HEART.

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INTRODUCTION.

The increasing interest in coronary disease during recent years has led to a better understanding of one of its most dramatic complications, spontaneous rupture of the heart. By reason of the tragic nature of the clinical picture and the interest attached to the post-mortem findings the condition has been the subject of frequent report. The most important of the early contributions to our knowledge of the subject is the thesis by Elleaume (Essai sur les ruptures du coeur, 42 p.p. 8°, Paris 1857). Since this paper various comprehensive studies have been made, including Quain (Med. Chir. Trans., London, 1871, 33, 121), Barth (Arch. gén. de Med., 1871, 65), Robin and Nicolli (De la rupture du Cœur, 152 pp., Ruff & Cie, Paris, 1895), Houchard (Maladies du Cœur, 1899, 1, p. 228), Minet and LeClercq (Clinique, Paris, 1910, v. 273), Wearn (on coronary disease) (Am Jour. Med. Sc., 1923, clxv, 250), and Krumbhaar and Crowell (Am. Jour. Med. Sc., 1925, clxx, 828). The last mentioned authors report 22 personal cases and have assembled 632 cases from the literature. In spite of this large series of published cases, however, the condition must be considered as a very rare one. Krumbhaar and Crowell give figures showing an incidence of only 23 cases among 37,000 autopsies. At the Massachusetts General Hospital there was one case in 4,076 autopsies and at the Boston City Hospital five cases in 5,800 autopsies. The combined figures show but 29 cases in 46,876 necropsies, or six one-hundredths of one per cent.

The five cases observed at the Boston City Hospital are summarized below.

CASE 1.

Man of 61. Excellent general health. Never any serious illness. Nocturia (2-3) for past two years. Frequent severe pain

in small of back for several years. No cardiac symptoms until past 2 or 3 months when he has had severe attacks about once each week of sudden intense dyspnoea, palpitation, weakness, vertigo, pallor, numbness in hands and great distress in epigastrium. They usually follow exertion and last approximately five minutes. Their severity has gradually increased. Physical examination showed moderate cyanosis, rapid respiration, evident dyspnoea and signs of double hydrothorax. Pulse elevated, equal, regular, fair volume and tension. Peripheral arteries stiff and tortuous. Heart moderately increased in lateral area of dullness, action regular, sounds fairly clear and normal, fair volume and tension; occasional reduplication of first sound at the apex. No murmurs. No oedema. Abdomen negative. The day after entrance the patient seemed more comfortable but on the following day had a sudden attack of intense dyspnoea with great precordial distress and became deeply cyanosed. Moderate relief from opiates. At the end of one or two hours a second attack, with death following almost instantaneously.

Autopsy No. 02-83. Each pleural cavity contains 1500 cc. of clear straw colored fluid. Pleural surfaces normal. Pericardial cavity contains bloody serum and a currant jelly clot which presents as an envelope to the heart. The clot peels readily from the epicardium, revealing on the anterior aspect of the left ventricle about one-half way down and within a centimeter of interventricular septum, two ruptures. These ruptures are slit-like, 4 to 5 mm. in length, 1 to 2 mm. broad. The ruptures contain clotted blood, and on gentle pressure no fluid escapes from them. Heart weight 540 gms. Measurement of valve orifices: tricuspid 13 cm., pulmonary 8.5 cm., mitral 12 cm., aortic 9 cm., left ventricle wall 2 cm., right ventricle wall .6 cm. Tricuspid, pulmonary and mitral valves normal. Slight diffuse thickening of the aortic cusps and some calcification at the insertion of the mitral valve. The wall of the left ventricle in the region of distribution of the anterior descending coronary is considerably thinned, presenting a marked concavity towards the ventricle and some outward bulging. The endocardium in this region is overlaid by considerable granular clot. On section of the heart wall in this region, it is found to be made up of pale, opaque, striated tissue alternating with lenticular areas of clotted blood. Such lenticular areas communi-

cate with the ruptures described on the surface of the heart and the clotted blood in the ventricles. The coronary artery supplying this region of the heart wall shows marked thickening of its wall and at one point, 2 cm. from the auricular ventricular groove, a roughened area to which a thrombus is adherent. At this point the lumen of the artery is completely occluded. This occlusion lies just outside of the ischaemic area of the myocardium. Elsewhere the wall of the left ventricle is considerably hypertrophied. The other coronary arteries show marked sclerosis, but are nowhere occluded.

Anatomical Diagnoses. Hemopericardium. Thrombosis of left descending coronary with infarction and rupture of the heart. Mural thrombosis. Coronary sclerosis. Chronic aortic endocarditis. Hypertrophy of left ventricle. General arteriorsclerosis. Hydrothorax (double). Edema of the lungs. Cholelithiasis. Chronic diffuse nephritis. Chronic interstitial orchitis.

Microscopic Examination. Heart. Scattered through the section are small islands of myocardium. The muscle fibers show various degrees of degeneration. One island seems to be one of the columnae carneae. This consists of a central area of comparatively normal muscle fibers surrounded by a narrow zone in which the fibers are in part degenerated and in part replaced by fat. The whole area is surrounded almost completely by a layer of fibrous tissue. The remaining portion of the section is made up of blood clot in which the fibrin network figures prominently. In some places the polynuclear leucocytes are present in large numbers.

CASE 2.

Man of 72. Carpenter, who for two years has had rather severe attacks of precordial pain radiating to the shoulder and down the left arm. These attacks are brought on by any form of exertion, but especially by walking. The present attack began at 11 o'clock in the morning while walking, when he had an attack of pain of great intensity in the region of the precordium, which radiated to the middle of the back and down both arms. He perspired freely and was exceedingly weak, but had no dyspnoea or respiratory symptoms. On examination when admitted to the hospital the patient was evidently in great agony from precordial pain. The heart area and position

seemed normal, but the sounds were very weak and intermittent. No definite murmurs made out. In spite of the weak heart sounds the pulse seemed to be of high tension. After a few hours the patient's pain was relieved and he seemed very comfortable for twenty-four hours, except for fairly frequent vomiting and some pain and tenderness in the epigastrium. On the second day the patient did not seem so well, and during the afternoon, while in a sitting posture talking to his wife, was seized with a sharp pain in the mid chest and radiating down the arms. He fell back on the pillows, dying instantly.

Autopsy No. 06-166. Entire outer surface of right lung bound to chest wall by old, soft, stringy adhesions. The pericardial cavity is distended with over 300 cc. of fluid and clotted blood. Surfaces smooth and glistening. A visible line of rupture in left ventricle reveals source of bloody effusion. Heart weight 320 gms. Epicardium well laden with fat. Muscle is soft and flabby. On surface of left veitricle, 7 cm. from apex and 4 cm. to left of upper margin of right ventricle, is a nearly vertical line of rupture 2 cm. in length. Edges are well approximated, but on separation are distinctly ragged in outline. On section this rupture is seen to communicate directly with cavity of left ventricle. A ragged vertical line of rupture 5 cm. in length is situated behind and between leaflets of mitral valve and 1 cm. below mitral ring. On section muscle at this point is soft, pinkish gray in color, and easily fragmented. Remaining muscle is quite soft and flabby. Aortic leaflets are slightly retracted and bases contain calcified plates with numerous small raised yellowish areas. Remaining valves are normal. Each coronary contains numerous irregular yellowish raised calcified placques. These are more marked in the upper third, in places nearly occluding arteries, and involve the orifices of the branches, especially those leading toward point of rupture. Measurements of valve orifices: tricuspid 11 cm., pulmonary 7.5 cm., mitral 9.5 cm., aortic 7 cm., left ventricle wall 1.7 cm., right ventricle wall .4 cm.

Anatomical Diagnoses. Spontaneous rupture left ventricle with hemopericardium. Advanced arteriosclerosis, involving coronaries. Myocarditis. Healed tuberculosis, both apices. Fibrous pleuritis.

Microscopic Examination. Heart. Several areas show extensive

necrosis of muscle fibers with heavy infiltration of polymorphonuclear leucocytes. Cultures of heart's blood: no growth.

CASE 3.

Man of 75. Only very meager history obtained. Chancre at 16; gonorrheal infection at 18. Apparently a rather prolonged history of dyspnoea and cough. Patient states that he was perfectly well until three months before admission, when he caught cold, since which time he has had a cough and pains in his left shoulder and at times throughout the thorax. During this period dyspnoea with slight exertion has been rather extreme. He has also had a good deal of intermittent pain in the upper right abdomen. On examination there is evidence of extreme arteriosclerosis. The heart does not seem enlarged, and the apex beat is neither seen nor felt. Regular rhythm. No murmurs. Sounds rather faint. Patient seemed very comfortable in the hospital, but on the third day, without warning, died instantly.

Autopsy No. 21-41. The peritoneal cavity contains about 500 cc. of amber colored fluid. Many firm adhesions binding the liver to all adjacent structures. The left pleural cavity contains about 1000 cc. of straw colored fluid, with several adhesions at the apex. Right pleural cavity obliterated, and both layers of pleura have been very intimately glued together by adhesions. The pericardium is tensely stretched by dark red blood, in the midst of which are several large clots. Over the anterior surface of the apex, for an area about 4 cm. in diameter, the pericardium, which is thickened to 4 mm. in this location, is so firmly adherent to the heart that it cannot be detached. Heart weight 505 gms. Larger than normal, soft and flabby. Apparently there has been a primary hypertrophy followed by thinning out of the lower two-thirds of the left ventricular wall, with rupture of this wall as cause of sudden death. The ventricular wall below the mitral valve is 1.6 cm. thick; it gradually thins out toward the apex, where it is only 2 to 3 mm. thick. A firm brown clot up to 1.3 cm. in thickness lines the lower portion of the ventricular cavity; it is an old clot and is detached with difficulty. Two perforations present themselves in this aneurysmal wall, one irregular in outline and large enough to admit the index finger, the other a linear rupture about 1 cm. in length. Valves are negative except the aortic, two of whose

leaflets have numerous small, hard, stony, pale yellow vegetations; the aortic orifice is coincidentally stenosed. Coronaries are sclerosed, the walls of vessels feeding the left ventricular wall are thick, yellow, and the vessel stands out rigidly, with its lumen practically occluded. Measurements of valve orifices: tricuspid 13.1 cm., pulmonary 8 cm., mitral 11.3 cm., aortic 6 cm., left ventricle wall variable, right ventricle wall .6 cm.

Anatomical Diagnoses. Aneurism of left ventricular wall with rupture. Healed aortic valvulitis with stenosis. Hemopericardium. Healed pericarditis. Healed obliterative pleurisy (right). Pleurisy with effusion (left). Oedema and congestion of lung (right). Chronic bronchitis. Chronic passive congestion. Healed perihepatitis. Ascites. Healed tubercles of spleen. Chronic diffuse nephritis (arteriosclerotic). Simple cyst of kidney (left). Healed infarcts of kidney (right). Arteriosclerosis.

Microscopic Examination. Considerable interstitial sclerosis of the heart, with pigment in the muscle cells. One slide shows epicardium with large amount of fat, and wall of left ventricle with adherent organizing thrombus. The aneurysmal wall consists of connective tissue, fibroblasts, occasional cardiac muscle bundles, many small blood vessels with more or less diffuse infiltration of erythrocytes, polymorphonuclear leucocytes, many endothelial leucocytes laden with blood pigment, lymphocites and a moderate amount of fibrin. The fibroblasts are growing into the blood clot on the endocardial wall.

CASE 4.

A man of 74 was brought to the hospital in an unconscious condition, having collapsed on an elevated train. He soon regained consciousness and seemed fairly rational, but with very feeble memory. Story obtained that for at least two years he has been very short of breath and has had considerable number of attacks of syncopy of very short duration. At entrance the patient was cyanosed, pulseless, and no heart sounds could be heard. With stimulation temporary improvement resulted, but later in the day the patient became very restless and evidently suffered intensely from cardiac distress. Death followed at the end of a few hours.

Autopsy No. 07-21. There is 100 cc. of clear, straw colored fluid in the peritoneal cavity. Firm fibrous bands unite the two layers of the right pleura. The pericardial cavity contains 75 cc. of fluid blood. About the heart there is a layer of clotted blood which anteriorly measures about 1.5 cm. and posteriorly about 2.5 cm. in thickness; not adherent to the heart. Over the left ventricle there is a small slit in the pericardial cavity. Heart weight 400 gms. In the left ventricle, 4.5 cm. from the interventricular septum, there is a slit through the pericardium and the myocardium connecting the left ventricle and the pericardial cavity. This slit measures 1.5 cm. in length and 3 mm. in breadth, and runs downward in the direction of the apex. There is a loose blood clot within the left ventricle which extends into this opening for a short distance. On section for 2 to 3 cm. about this rupture in the heart wall, the myocardium shows marked evidence of degeneration. Just adjacent to the rupture its color is light red, and it is quite soft. Beyond this point its color has a distinct yellowish tint, and it is much softer than normal. Coronaries are thickened in places. The branch passing downward in the direction of the rupture shows several thickened areas, one of which almost completely occludes the vessel. Measurement of the valve orifices: tricuspid 12.5 cm., pulmonary 6.5 cm., mitral 9 cm., aortic 7.5 cm., left ventricle wall 1.7 cm.

Anatomical Diagnoses. Rupture of the heart. Hemopericardium. Softening of the myocardium. Myocarditis. Chronic nephritis. General arteriosclerosis. Hypertrophy of the heart. Congestion and oedema of the lungs. Chronic passive congestion of the liver and spleen. Chronic adhesive pleurisy. Right hydrocele. Chronic adhesive pericarditis. Oedema of brain. Arteriosclerosis of the circle of Willis.

Microscopic Examination. Heart. Irregularly distributed areas of varying size show numerous fine fat droplets throughout the myocardium. In places these fine drops are so closely packed that the striation of the muscle cannot be seen. In the area of rupture there is quite marked fatty infiltration of muscle, interstitial leucocytic infiltration and intramyocardial leucocytic infiltration. In both positions there is marked fatty infiltration of the leucocytes. The amount of the individual muscle fiber which has been infiltrated with polymor-

phonuclear leucocytes varies from a small area to most of the fiber. Cultures of heart's blood: sterile.

CASE 5.

Man of 60. Engineer. Previous health excellent. Entered the hospital with a story of shortness of breath with exertion of rather sudden onset one week previously. Constant sensation of being choked. Examination showed heart very much increased in lateral diameter, sounds regular but rather weak, soft systolic murmur with maximum at the apex. Patient's general appearance and symptoms did not indicate that he was critically ill. During the first day seemed very comfortable. Had a good night and awoke in morning feeling in good condition. About 8.45 A.M., however, while sitting quietly in bed, suddenly collapsed, became cyanosed and died within a few seconds.

Autopsy No. 07-151. Pericardial cavity considerably larger than normal. Fluctuation on palpation. It contains a large blood clot, thickest in dependent part and 300 cc. fluid blood. A clot completely surrounds the heart, but is nowhere adherent to pericardium. A slit entirely through the left ventricle connects the pericardial cavity and left ventricle. Heart weight 450 gms. There is a slit in the lower portion of the left ventricle 2 cm. from the interventricular septum. This slit is situated posteriorly, measures 4 cm. in length and contains a clot which connects with the clot in the pericardium and left ventricle. About the rupture the myocardium is of dark color and softer than myocardium elsewhere. The remainder of the myocardium, most marked throughout the left ventricle, shows quite numerous, fairly well-outlined, yellowish areas. As a whole the myocardium is softer than normal. The endocardium is smooth and glistening, save about the bases of the aortic valves, where there is some thickening. The coronaries show considerable sclerosis, most marked in localized areas. The coronary passing toward the region of the rupture is at one point almost occluded by a small, calcareous area. Measurement of valves normal. Thickness of left ventricle 2 to 2.5 cm., right ventricle .7 cm.

Anatomical Diagnoses. Hemopericardium with rupture of heart. Hypertrophy of heart. Fatty heart. General arteriosclerosis and of

basal vessels. Chronic nephritis. Subpial oedema. Hypertrophy of prostate.

Microscopic Examination. Heart. Shows irregular areas with fat in fine drops, which vary in amount within the myocardium. Most marked in and immediately about areas of sclerosis. Some connective tissue cells in sclerotic areas show fat drops. Phagocytic rather than degenerative process. Atrophy of muscle fibers. Increase of lipochrome. *Aorta.* Marked fatty change, mostly in the thickened intima. Areas of diffuse fatty infiltration and areas of fat in very fine drops. Latter most marked in the innermost layers of the adventitia. Cultures of pericardial blood sterile.

Symptoms and Course.

The clinical picture of this condition, though in some respects extremely uniform in type, is not sufficiently unique and characteristic to warrant a sharp differentiation from several other cardiac emergencies and especially coronary disease. Indeed, strictly speaking, the symptoms of spontaneous rupture of the heart are in a vast majority of cases those of occlusion of the coronary arteries with infarction, the former being merely the final stage in the cardiac failure. An individual with myocardial disease suddenly succumbs without the development of new symptoms, and at necropsy a rupture of the heart wall is found. This is strikingly true of each of the five cases given above. In cases 1 and 2 death occurred suddenly in the course of a typical attack of angina pectoris to which the patient had been subject. The three remaining cases presented a typical picture of severe coronary sclerosis without angina. In each there was sudden collapse and death, cases 3 and 5 dying instantly, case 4 only after an interval of a few hours.

In a considerable number of the published cases there have been no very definite symptoms of cardiac disease until the final attack with immediate death. The usual picture in such cases is that of very great precordial distress, intense dyspnoea, cyanosis, collapse and death, with no preliminary or warning symptoms of the cardiac accident. In the general run of cases, on the other hand, the terminal attack follows only after a shorter or longer period of intermittent

coronary symptoms, such as those noted in the cases forming the basis of this paper.

The true symptoms of rupture, then, may be regarded as those occurring with the terminal attack, and these present a fairly uniform complex. The onset is very abrupt and almost invariably with pain in some form as the outstanding feature. Without warning the victim is seized with an overwhelming pain in the region of the heart, which may or may not radiate to the back or shoulders and arms. Less commonly it is localized in the epigastrium. Its type is variously described as "tearing," "boring," "great pressure," etc. The agony evidenced by these cases is of a very extreme type. The next most frequent and outstanding symptom is dyspnoea, which is almost always of an extreme grade and often associated with a choking sensation. Cyanosis is usual, often intense. Nausea and vomiting may be present. Pallor, vertigo, cold sweats, vomiting, diarrhea, and faintness often form a part of the picture of collapse. In a few instances severe convulsions have been observed. Oedema of the lungs may develop. Fever and leucocytosis very frequently appear if the patient survives the initial attack. Rebattu and Josserand (Lyon Med., 1923, cxxxii, 41) report a case which is quite unique in that the patient died instantly without symptoms of any kind. At necropsy a large jagged hole in the anterior wall of the left ventricle was found, giving the impression that the infarct had literally dropped out.

Physical signs are primarily those of collapse as described above. Death supervenes so quickly in most cases that there is no opportunity for a careful study of the heart. In the few cases where an examination was made the signs do not seem to have been exceptional in any way. The heart may or may not be enlarged, the action is rapid and tumultuous, often with irregular rhythm and sounds of poor quality. Reznikoff (Jour. Am. Med. Assn., 1922, lxxviii, 1926) reports a case with rupture of the left ventricle in whom, on auscultation just before death, "a continuous, muffled, low pitched rushing rumble" was heard over the precordium. The signs of hydropericardium have been described, but it is difficult to conceive of such a picture ordinarily, since death occurs simultaneously with the filling of the pericardium. Furthermore, the amount of blood usually found

in the sac is not sufficient to give very striking signs of pericordial effusion.

Clinical Course. Sufficient has been said above to indicate that as a rule death occurs within a few seconds following the onset of the first symptoms. Krumbhaar and Crowell found this to be true in 72 per cent of all recorded cases, while in another 7.7 per cent exitus was delayed only ten minutes. In the remaining 20 per cent of cases the interval was from ten minutes to several days. The onset and the intensity of the symptoms in this last mentioned group are the same, but instead of collapse and death the patient gradually improves, to be stricken again with immediately fatal results. Case 4 furnishes an excellent example of this type. The patient was brought to the hospital in collapse and lived several hours after entrance, then dying very suddenly in an attack with precordial pain. It must be admitted that in this last mentioned type of case we have no proof that rupture develops with the onset of the early symptoms. Precisely such a course is characteristic of coronary thrombosis without rupture of the myocardium. In many instances of this condition death is also instantaneous, while in others there is a varying interval between the onset of symptoms and death. The mode of death is likewise the same, both as regards the symptoms observed and their severity.

Pathology and Etiology.

Peculiar interest attaches to the discussion of the pathology of this condition for the reason that a necropsy has been done on all published cases. It is unfortunate, however, that so few of the early cases were studied microscopically, and that so many of the case reports are very incomplete.

In a previous article by the author (Medical Clinics of North America, Phil., 1925, viii, 1669) the figures with regard to location of the rupture in 259 published cases were given. This group of cases showed 79.9 per cent of the lesions in the left and 10.1 per cent in the right ventricle, figures which are almost identical with those of Krumbhaar and Crowell for their larger group of 654 cases. The remaining 10 per cent are about equally distributed between the auricles and other miscellaneous locations in the heart. The inter-

ventricular septum is occasionally the site of rupture. Among the 80 per cent of cases with rupture in the left ventricle, nearly all occurred in the anterior wall, the favorite site in order of frequency being the lower, the middle, and the upper third. At whatever level of the ventricular wall, the tear is apt to be near the interventricular ridge. In all five cases reported in this paper the opening was in the anterior wall of the left ventricle. Only a few instances are recorded of rupture in the posterior wall of the left ventricle. The overwhelming frequency of the lesion in the anterior wall of this heart chamber is readily explained by the great prevalence of sclerotic changes in the descending branch of the left coronary artery, with resulting occlusion and infarcts of the lower anterior two-thirds of the left ventricle.

The tear is almost invariably single, although there are a few instances of multiple openings, as in cases 1 and 3 of our series, in each of which there were two openings into the pericardium. The character of the tear in the heart muscle is of quite varied form, but usually of a linear type, varying in length up to a maximum of 7 or 8 cm., and in width seldom more than a few mm. It is most often closed tightly, but the edges may be separated and plugged firmly by a clot. Occasionally the opening is very clean cut, as if done with a sharp knife, at other times it is irregular, jagged, angular, or even radiate. A few specimens have shown simply a ragged hole in the ventricle wall. Observations have frequently been made that the external opening is considerably larger than the inner, which may be very small and hidden among the papillary muscles. The course of the canal through the ventricle wall is sometimes direct, but more often very irregular as in the common zigzag type.

The outward appearances and condition of the pericardial sac are constant except for slight variations. It is greatly distended, tense, fluctuant, and of a dull, congested color. These appearances are due to the presence of blood clots and serosanguineous fluid to the amount of a few hundred cubic centimeters. A larger quantity of blood is not generally found, due, it would seem, to the fact that the normal pericardial sac is very resistant to sudden distention. A very striking exception to this rule is to be found in Sturrock's unique case (Brit. Med. Jour., London, 1906, i, 500), where, notwithstand-

ing the fact that death occurred in four minutes after the onset of symptoms, the pericardium contained 29 ounces of clotted and fluid blood. Very commonly the heart is entirely encased in a continuous clot to which it may be in part densely adherent. Since death follows so quickly, changes in the pericardium are insignificant.

The heart muscle presents at the same time the most interesting and significant changes. Unfortunately, many of the autopsy reports are so meager in their description of the condition of the myocardium as to be of little or no value. In a very large number of cases, for example, the condition of the coronaries is not even recorded. A careful study of the reports regarding the morbid anatomy of the condition under consideration therefore is disappointing because of the inconclusive evidence which it yields. There are still some unsettled questions of very vital moment regarding the pathogenesis. In the main, however, the changes in the heart muscle which are characteristic of the disease can be quite definitely described.

In his classic treatise entitled "On Fatty Diseases of the Heart," referred to earlier, Quain discusses with considerable thoroughness the symptomology and pathogenesis of spontaneous rupture of the heart. He lays the greatest stress on fatty degeneration of the myocardium, as did many of the early writers on the subject, largely overlooking the rôle played by disease of the coronary arteries. Judgment regarding the condition of the heart muscle was formed by these older writers largely from the gross appearances rather than from careful histological examination, as has been brought out by Krumbhaar and Crowell. In 30 per cent of the cases tabulated by these authors the cause of the rupture was attributed to "fatty degeneration and infiltration." It appears very significant that in the pathological reports of recent years the description of such alterations in the heart muscle as fatty degeneration have been given very little prominence, the condition being regarded as one form of muscle degeneration resulting from coronary sclerosis with occlusions. These changes are apt to be especially evident just beneath the epicardium.

In accordance with v. Jürgensen's law, spontaneous rupture occurs only in the severely diseased muscle. Most commonly the lesion is in the center of an area of infarction. The area comprising the infarct is of dark color, soft, and on histological examination

found to show the type of degeneration common to local anemia, and actual necrosis if the occlusion is complete. The altered tissue is often intensely hemorrhagic, suggesting that the blood has been forced into the softened area, as in a dissecting aneurism. In this severe type, rupture is apt to occur early and with immediate death. Such a condition is shown in case 1, where thrombosis of the descending branch of the left coronary artery has led to a typical infarct and rupture in the middle anterior part of the left ventricle near the septum. In this case the heart muscle is pale, somewhat opaque, soft, and with areas of clotted blood. Microscopically the muscle fibers show various degrees of degeneration with a large amount of fatty infiltration. The central portion of the infarct is composed of blood clot with a rich fibrin network. Polynuclear leucocyte infiltration is extensive.

If rupture does not take place early or if the occlusion of the coronary artery is incomplete or slow in development, a somewhat different condition is produced, namely, a greater degree of general fibrosis with atrophy of the muscle fibers. It is this form which occasionally goes on to the formation of aneurismal dilatation, and as a final stage sometimes to perforation also. An excellent example of this is found in case 3, in which the lower two-thirds of the left ventricle is thinned to barely 2 or 3 mm. and takes on the form of a definite aneurism in the center of which are two perforations. The coronaries are sclerosed, the lumen of the vessel supplying the left ventricle being almost occluded. The microscopic examination of the aneurismal wall shows mainly connective tissue diffusely infiltrated with erythrocytes, polymorphonuclear and endothelial leucocytes. Many fibroblasts and small blood vessels are present, but only an occasional muscle bundle. Cases 2, 4 and 5 seem to be intermediate between the condition of true infarct with rupture in an early stage (case 1) and the slow fibrosis with aneurism and final rupture (case 3). The coronaries in all three show extensive sclerosis with almost complete occlusion of the lumen of the descending branch of the left and moderately extensive myocardial degeneration of the wall of the left ventricle, but no infarct. The degree and extent of degenerative changes in cases 2 and 5, however, are much less than in case 1 and show but little fibrosis as compared with case 3. The gross

changes in case 4 are the same as cases 2 and 5, but are much more extensive and striking. Degeneration, fatty infiltration and leucocytic infiltration, both interstitial and intramyocardial, are very marked.

Whether the process is an acute myomalacia from sudden and complete coronary occlusion or a more slowly developing process of fibrosis and degeneration from less abrupt interruption of the blood supply to the heart muscle, the result is the same, namely, a much weakened heart wall over a localized area. The diseased muscle is usually found to be surrounded by relatively healthy muscle, which furnishes ideal conditions for rupture of the heart wall during systole. With but few exceptions the heart shows some degree of hypertrophy. In the cases forming the basis of this paper the hearts weighed respectively 540, 320, 505, 400, and 450 grams.

While there seems every reason to assume that in a vast majority of cases the pathogenesis of spontaneous heart rupture is as given above, it must be admitted that in a very small percentage of cases other conditions may lead to rupture of the heart wall. The most important of these are syphilitic invasion of the myocardium, infectious myocarditis, embolic abscess of the heart wall, and ulcerative endocarditis with invasion of the myocardium. Tuberculosis, tumors and cysts are less frequent causes. A very striking fact is the considerable number of aged insane among the reported cases of rupture, but its significance is not clear.

Certain so-called secondary causes merit passing consideration, since they appear very definitely to play some part, though a minor one, in the production of the rupture in a portion of the cases. It would be natural to consider such factors as any extreme physical exertion as favoring rupture through sudden and powerful contraction of the heart. Indeed, a significant association between rupture and such severe efforts as carrying heavy burdens, intense exertion, violent laughing or coughing, convulsions, vomiting, difficult defecation, coitus, etc., seems to have been shown in many cases. Probably great excitement and intense emotional upsets should also be included in the same category. Minet and Leclercq lay great stress on the part which these "determining causes" play in the actual production of rupture. On the other hand, in a much larger percentage rupture has not occurred following a sudden increase of intracardiac pressure, but

while the patient was lying absolutely quiet in bed. This was true of all five of our cases. A considerable number have actually died during sleep.

In the main, the mechanism of cardiac rupture is fully explained on the basis of the foregoing considerations. The exact relation of symptoms to the stage of the process, however, is less clear and it may well be that no close correlation is possible. It seems evident that if the tear is complete and of sufficient size death must follow almost instantly. What happens in those cases where death follows only after a varying interval of hours or days? It is conceivable that an incomplete or very small break in the muscle wall might permit a very small leak only and death be postponed for a short time. It is rather generally contended that the first sudden onset of pain and dyspnoea marks the first stage of the accident, the final collapse, and death the real breaking through of the rent into the pericardium. Ramond and Baudoin (Medicine, Paris, 1921-22, ii, 426) suggest the possibility that in certain cases of prolonged evolution with each contraction of the heart the edges of the wound are forced tightly together, thus preventing the rapid development of hemopericardium. In those cases of long delay in the fatal outcome it seems more reasonable to assume, as stated earlier in this paper, that the sudden initial symptoms are due to an accident to the coronary vessels and at the end of some days the final and fatal attack, the evidence of rupture of the heart muscle. Or may it not happen occasionally that the blood slowly bores its way through the diseased muscle?

The cause of death has been the subject of much speculation and of some experimental study. It seems obvious that the pressure of the blood forced into the pericardium through the opening in the heart wall must have some bearing on the heart action, but that increased pericardial pressure alone is the cause of death, as so often assumed, is not entirely reasonable. Hopkins (Proc. New York Path. Soc., 1910-1911, n.s., x, 200) gives an excellent discussion of the subject and presents authoritative experimental evidence to show that an increase in intrapericardial pressure in animals of only moderate degree, if sudden, will cause death. This author expresses his conviction that when the pressure in the pericardium becomes equal to that in the right auricle, blood no longer enters the heart from the

systemic veins. That this factor is not the cause of death in all cases can be easily proved by the fact that a few cases are on record where but very little or no blood was found in the pericardium, *i.e.*, not a sufficient quantity to alter the pressure significantly. There is a good deal which might be said in favor of the theory advocated by many that death is due to reflex causes. Those fatal cases without hemorrhage into the pericardium are very reasonably explained on this hypothesis.

Is the rupture always fatal? The post-mortem findings of extensive fibrosis marking the site of earlier infarct without rupture indicate positively that healing under these circumstances is not unusual. A few cases are on record in which scars have been found which the observer believes represent healing of a true rupture. The evidence is by no means convincing, and on theoretic grounds healing is impossible.

Rupture of the heart is a disease of old age. In a total of 602 cases, tabulated by Krumbhaar and Crowell, 72.5 per cent were sixty or over. They further show that the relatively few cases occurring before middle life and without coronary diseases are the ones due to the miscellaneous causes mentioned above.

Males predominate somewhat over females. Habits, occupation, diet, general hygiene and other similar factors are of only very minor if any significance in the consideration of etiology.

An ante-mortem diagnosis of spontaneous rupture of the heart is practically impossible. One can only consider its possibility under certain conditions in cases of sudden death. In an individual over sixty who dies suddenly with symptoms of great dyspnoea and intense precordial or epigastric pain of anginal type, there is a small chance that a rupture of the heart is the cause of death. Even a reasonably certain differentiation between this lesion and angina pectoris or coronary disease cannot be made. In those cases of slow evolution a careful physical examination is usually possible and may rarely give some data suggestive of rupture.