Atrial Rupture of the Heart

Report of Case following Atrial Infarction and Summary of 79 Cases Collected from the Literature

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This paper reports a case of atrial rupture of the heart and reviews 79 other cases collected from the literature proven by autopsy. Incidence, etiology, pathologic physiology, diagnosis and possible treatment are discussed. Infarction of the atrial wall due to an oblitative endarteritis or thrombotic occlusion is the most common cause of spontaneous rupture of the atrium. With a high index of suspicion of atrial rupture in a patient with atrial infarction or cardiac tamponade, earlier diagnosis and surgical treatment may become possible.

Atrial rupture of the heart is a dramatic event which leads inevitably to death. This paper reports the only case of atrial rupture among 5,900 autopsies at the Albany Hospital and summarizes the pertinent literature on atrial rupture as to etiology, diagnosis, pathologic physiology, sex, age, and treatment. A total of 79 cases of atrial rupture has been collected from the literature, classified and studied. The present report brings the total number of recorded instances of rupture of the atrium up to 80.

The term "atrial rupture" denotes a complete, acquired defect of the wall of either atrium through which the blood may leak into the pericardial sac and produce a fatal pericardial tamponade. This condition, never yet known to be diagnosed ante mortem, and suspected only once in 80 cases, is frequently confused with ventricular rupture.

Ventricular rupture occurs in 8 to 10 per cent of patients who develop myocardial infarction with a high incidence among insane and hypertensive patients. Atrial rupture, by contrast, is a much rarer condition. In the 710 combined cases of spontaneous rupture of the heart reported by Krumbhaar and Crowell and by Davenport, atrial rupture occurred in 51 patients. The right atrium was involved in 38 patients and the left in 13. Bright and Beck, in a review of 152 cases of traumatic heart rupture, found the right atrium damaged in 36 patients and the left in 30. One had a perforation of the interatrial septum. Since Bright and Beck's review in 1935, only sporadic cases of atrial rupture have been reported.

Case Report

J. H., AH 74724, a 64 year old white woman, was admitted to the Medical Service of the Albany Hospital on Sept. 9, 1940. She complained of weakness, vomiting, and lancinating precordial pain of one hour's duration. For the two preceding years, the patient had suffered from angina pectoris and dyspnea during exertion. One year prior to admission, she had chest pains attributed to pleurisy. Previous operations included oophorectomy, renal lithotomy, appendectomy, cholecystectomy, and herniorrhaphy.

On physical examination, the patient was conscious but appeared rather ill, and was evidently suffering severe precordial pain. She vomited occasionally. The blood pressure was 80/60; the pulse rate was 80; the heart sounds were distant and weak and the heart's action slightly irregular. No murmur or friction rub was audible.

Urinalysis showed 2 plus albumin and some leukocytes. The erythrocyte count was 4,800,000; the leukocyte count 13,800 per cubic millimeter, with 80 per cent polymorphonuclear cells and 20 per cent lymphocytes. The Wassermann test was negative. The blood nonprotein nitrogen was 45.4 mg. per 100 cc.

The patient's temperature, elevated to 101 F. on the second hospital day, returned to normal by the sixth hospital day. Her blood pressure rose to 120/80 within the first 24 hours. On the fourth hospital day, her pulse rate suddenly increased to 240 per minute and an electrocardiogram revealed paroxysmal auricular fibrillation (fig. 1). Sinus rhythm returned rather promptly after the administration of 0.3 Gm. of quinidine. Electrocardiograms before and after this episode showed regular sinus rhythm. The patient improved until the thirty-fifth hospital...
day when she complained again of precordial pain. The temperature rose to 101 F. The blood pressure dropped to 90/70; the heart rhythm alternated between paroxysmal auricular fibrillation and regular sinus rhythm despite the administration of digitalis and quinidine. The severe chest pain persisted for several days. Shortly after awakening on the forty-first hospital day, the patient asked to be raised in bed. Before this could be done, she suddenly died. The clinical diagnoses recorded ante mortem were recent and remote infarction of the left ventricle.

Postmortem Examination. An autopsy revealed an almost imperceptible perforation on the anterior surface of the right atrium near the coronary sinus which had resulted in a hemopericardium, consisting of 500 cc. of clotted blood and 200 cc. of serous fluid. The heart weighed 240 Gm. Calcified pulmonary valves and fusion of two aortic cusps with calcifications were noted. The walls of the coronary arteries were thickened but no occlusion was found.

Incidental findings included atherosclerosis of the aorta, generalized arteriosclerosis, acute congestion of the spleen and kidneys, and a large cyst in the right lobe of the liver.

Microscopically, the heart revealed evidence of old pericarditis with necrosis of some myocardial fibers in the region of the atrial rupture. The main coronary vessels with intact intima were patent. Some endarteritis was present. The small coronary branches showed lymphocytic cuffing, splitting of the muscle layer, desquamation of endothelial cells, and partial occlusion of the lumina by granulation tissue.

Pathologic diagnoses: (1) infarction of the right atrium, anterior wall, with softening, rupture and hemothorax; (2) pericarditis, old, without scar tissue formation; (3) endarteritis of coronary vessels with narrowing of smaller branches; (4) congestion of spleen, kidneys and liver; (5) atherosclerosis of aorta; (6) valvulitis, aortic, atherosclerotic, old, with calcification; (7) cysts of right lobe of liver, and (8) generalized arteriosclerosis.

**DISCUSSION**

**Review of Literature**

Few of the reported cases contain adequate microscopic descriptions of the pathologic findings, and complete clinical data are often lacking. Electrocardiographic tracings are rarely published since many of the cases were recorded before the introduction of the electrocardiograph some two decades ago.

Rutherford recorded the first case of atrial rupture in 1828.33 His patient, a 24 year old female, subject to “deep mental inquietude,” had occasionally suffered from palpitation of the heart for about four years prior to her death.

Complaining of pains in the chest, she was bled by leeches and blood letting. After 22 ounces of blood were removed, “She did not show the slightest disposition to faint after the bleeding, but on lying down she suddenly exclaimed 'Oh dear, my heart! it will certainly burst: my feet feel so strange, they are quite dead: pray, put your hand on my heart, sir, it will come out.' I went immediately round to her bedside when she expired in my arms in a state similar to fainting.’” Autopsy demonstrated a rupture of the right atrium near the superior vena cava.

Subsequently, sporadic reports of atrial rupture have appeared in the world literature. Table 1* classifies these according to etiology.

* At the request of the Editor, table 1 is being omitted. In this table all reported cases of atrial rupture are summarized and classified as to etiology. Table 1 will be furnished on request.
Etiology of Atrial Rupture

Atrial rupture is almost always associated with or preceded by one of the following conditions: trauma, atrial infarction, fatty degeneration of the muscle, valvular heart disease, tumor of the heart or aneurysm of the atrium (table 1*). Several cases, whose etiology cannot be ascertained because of inadequate description, are listed under the heading of “miscellaneous.” Trauma is the prime cause of atrial rupture. Atrial infarction is next.

A. Atrial infarction (10 cases, table 1, cases 1 to 10 inclusive*). It has been observed that atrial infarction per se does not necessarily lead to atrial rupture. In fact, most patients do not develop atrial rupture after atrial infarction. However, in the collected series of 10 cases, which does include our own, atrial infarction actually preceded atrial rupture in every instance (table 1, cases 1 to 10).* In two of these patients, there was an associated infarction of the left ventricle. In the other eight cases, atrial infarction occurred without ventricular infarction.

In the 10 cases under discussion, the right atrium was involved more frequently than the left. Rupture of the right atrium occurred in seven; rupture of the left atrium, in three. The rupture was usually a single one, multiple ruptures being found in the same atrium in only one instance.

Atrial infarction may occur without thrombosis of a major artery. Hyaline changes and narrowing of the smaller coronary vessels without thrombosis produced the infarction in our case. In Cushing’s series of atrial infarctions without rupture, only 1 of 31 cases demonstrated a gross occlusion of a coronary artery. The gross recognition of atrial infarction may be difficult. In questionable cases, many tissue blocks must be taken for microscopic examination. Atrial thrombi, when present, often serve to call attention to the possibility of underlying atrial infarction.65

Before the work of Cushing and his associates in 1942, the true incidence of atrial infarction was not realized. In 182 patients dying of myocardial infarction of the ventricle, these authors carefully searched for evidence of infarction in the auricles and found it present in 31 or 17 per cent. Söderström, in 1948, independently confirmed and extended the preceding studies of Cushing in a most comprehensive and classic monograph. The material studied comprised 192 autopsy cases with mural thrombi in the cardiac atria. Atrial infarcts were practically impossible to recognize with the naked eye. Realization of the value of mural atrial thrombosis as an indicator led to the discovery of an increased number of underlying atrial infarctions, but only a fraction of mural thromboses yielded infarcts. Forty-seven examples of true infarcts were found with minor atrial lesions in 33 other cases. Clinical signs referable to atrial infarcts proved to be few and of little practical import. Electrocardiographic changes were not considered reliable indexes of infarction. There were no instances of atrial rupture, secondary to infarction, in either Cushing’s or Söderström’s series.

B. “Fatty degeneration of the heart” (7 cases, table 1, cases 11 to 17 inclusive*). Fatty degeneration and brown atrophy of the heart muscle are descriptive terms applied to cases of atrial rupture reported many years ago which, today, would be classified as infarction. Quain, for example, as early as 1850 pointed out that many instances of fatty degeneration and brown atrophy also manifested disease of the nutrient arteries.63

For most of their cases, the older authors gave as the cause of rupture either fatty degeneration of the heart or myocarditis. They contented themselves with the examination of the heart muscle only. In the second half of the nineteenth century, attention was focussed on the atheromatous changes in the coronary arteries. From that time on, an increasing number of observers indicated that the softening of the heart muscle was in the large majority of cases due to disease of the nutrient arteries of the heart.

Askey and Edwardes state that it has not been established whether fatty changes are greater than normal in the heart which has been ruptured. Before ischemic necrosis resulting from coronary occlusion was recognized,
the myomalacia was usually ascribed to fatty degeneration. When the true nature of the necrosis was realized, fatty changes were generally rejected by pathologists as having little if any etiologic significance. This same attitude applied not only to fatty degeneration but to fatty infiltration as well. It is now a fairly well accepted fact that fatty degeneration occurs no more frequently in the hearts of myocardial infarction than in the hearts of apparently normal persons. Beresford and Earl suggest that increased fatty infiltration may be a factor in inducing rupture. It is argued that ischemia produces necrosis of fat with extreme softening, almost to the point of liquefaction. The softened intramural fat, it is thus believed, facilitates the extension of a tear through the cardiac wall and hemorrhage, once initiated, forces its way into the epicardium.

It is rather significant that no case of atrial rupture has been ascribed to fatty degeneration since 1905, which fact offers convincing proof of a decided change in our concept as to the mechanism of cardiac rupture.

C. Trauma (19 cases, table 1, cases 18 to 36 inclusive*). Various types of trauma may lead to rupture of any of the heart chambers. In this review, the nonpenetrating type of trauma leading to atrial rupture is the sole variety of injury considered. Only a single chamber is, as a rule, involved. Most cases of atrial rupture have been produced by direct blows to the chest, but several instances have been reported which were associated with trauma to the head, leg or abdomen only.

Three theories have been advanced to explain the production mechanisms of atrial rupture by nonpenetrating trauma. In the first mechanism, direct compression of the heart between the sternum and the dorsal vertebrae is believed to be capable of producing atrial rupture. This is the most plausible explanation in patients who die from atrial rupture immediately after an accident.

In the second mechanism, contusion of the heart with subsequent necrosis of the injured myocardium is thought to lead to rupture of the atrium. An example of this type is that of a 9 year old boy (case 23) who died suddenly seven days after being assaulted by a group of boys. Autopsy demonstrated a slit in the left atrium and a clot in the pericardial sac of several days duration. Similarly, a five year old girl (case 25) fell two feet from a porch without evidence of injury being found. Seven days later, she died in her sleep. Autopsy showed rupture of the left atrium.

In the third and less clearly established mechanism, trauma to the head, leg, or abdomen appears to have led in some instances to atrial rupture, by increasing the intracardiac pressure. Cases 19 and 34 are good examples of this. Experimental support for this concept is furnished by the work of Bright and Beck. They tried to produce cardiac rupture in dogs by indirect trauma applied to the legs and abdomens of the animals, and found that the right atrial pressure rose in their animals. They were also able to produce cardiac dilatation and heart failure together with small epicardial and myocardial hemorrhages, but no actual rupture of the heart occurred in their dogs.

The condition of the myocardium at the time of injury definitely influences the production of atrial rupture by nonpenetrating injury. Atrial rupture is more likely to occur when trauma is inflicted upon a previously damaged myocardium.

D. Valvular heart disease (5 cases, presumably all chronic rheumatic heart disease. table 1, cases 37 to 41 inclusive*). Five cases of atrial rupture associated with valvular heart disease have been reported. Four had involvement of both mitral and aortic valves; the fifth case (case 41) showed only mitral stenosis. The right atrium was involved in four instances; the left, in one. Two of these cases (cases 39 and 40) also had subacute bacterial endocarditis which may have caused additional damage to the myocardium.

In this group, it seems reasonable to believe that elevated atrial pressures produced by the valvular lesions contributed to the rupture and that the myocardium, already weakened by disease, gave way to the strain of the elevated atrial pressure. In one patient (case 41),

* See footnote page 222.
pulmonary emboli also may well have contributed to an elevated pressure in the right atrium.

E. Tumors (3 cases, table 1, cases 42 to 44 inclusive*). Tumors were associated with rupture of the left atrium in three instances. One patient had a primary rhabdomyosarcoma of the heart; the second, a metastatic lesion to the heart from carcinoma of the duodenum; and the third, infiltration of the myocardium by leukemic cells of the myeloid type.

F. Aneurysm of Atrium (3 cases, table 1, cases 45 to 47 inclusive*). Three cases of atrial rupture associated with aneurysm of the atrial wall were reported between 1903 and 1908.

G. Miscellaneous (33 cases, table 1, cases 48 to 80 inclusive*). This category includes 33 atrial ruptures of uncertain etiology which cannot be classified because of incompletely recorded information. Three patients had coronary artery disease without infarction of the atrium. In the first patient (case 77), the unusual exertion of cranking a Diesel engine produced atrial rupture despite apparent good health. After working for an hour, he complained of something "going wrong" in his stomach and died suddenly. Autopsy revealed multiple ruptures of the right atrium without necrosis in the region of the ruptures. The right coronary artery contained multiple foci of medial necrosis with calcification, atheroma formation, intimal thickening, and splitting of the elastica.

In the second patient (case 66), a 26 year old man with acute nephritis, atrial rupture was attributed to "degeneration of the heart" from Bright's disease. Atheromatous changes of the atria and of the aorta were present.

The third patient (case 75), a 55 year old woman, died suddenly in a mental institution. Autopsy revealed atherosclerosis of the coronary arteries and rupture of the right atrium.

Unusual exertion is not a common antecedent of atrial rupture. In only four cases was unusual exertion mentioned, such as rowing a boat, cranking a Diesel engine, drawing a truck, and carrying a weight of 20 pounds. One patient was stricken while straining at stool and one, while attempting to vomit.

Several patients with syphilis also developed atrial rupture but there is insufficient evidence to implicate syphilis as an etiologic factor.

Diagnosis of Atrial Rupture

There is no instance of atrial rupture diagnosed ante mortem in the 80 cases comprising this study. Di Ilesi and associates99 suspected it and came nearer to making an actual ante mortem diagnosis in 1949 than anyone. A 69 year old white man, "suffered an acute myocardial infarction involving the posterior wall of the left ventricle, right auricle and inter-ventricular septum. The elevation of the P-Ta segment in leads II and III offered a clue to the presence of a recent infarction involving the posterior wall of one of the auricles." The development of symptoms and signs varies with the length of the survival time of the patient. Symptoms, in general, closely resemble those of myocardial infarction of the ventricle. Severe excruciating chest pain, often radiating to the back, is the commonest complaint of patients who survive the initial episode. Weakness, dyspnea, and occasionally, syncope from a fall in blood pressure are prone to occur.

Patients surviving the atrial rupture for a number of hours or days may develop such signs of cardiac tamponade as a small weak pulse, distant heart sounds, an increased area of cardiac dullness, and distended neck veins. Cardiac arrhythmias are common. Although there are no characteristic auscultatory findings, in patient 65 a splashing sound was audible during systole. In patient 53, the examiner heard a rushing sound at the presumptive time of rupture and a previously audible bruit disappeared. Because of the paucity of characteristic diagnostic physical signs recorded in such cases, it is of interest to quote briefly a portion of the author's original description of the findings on auscultation in one of these two instances where an unusual bruit was heard.

Case 65, reported by H. B. Smith in 1881, deals with a 37 year old man first seen in April 1881 with pain in the region of the heart. A

* See foot note page 222.
constant ache had been present for the preceding six months. Examination was negative. Several months later, while stooping over after having built a fire, he fell unconscious to the floor. Examined 20 minutes later, he was found to be conscious, breathing hurriedly and deeply, with a terrified expression of countenance, writhing and tossing in his bed, calling to be raised up one minute and turned over the next, asking to be rubbed on the extremities, back and chest, while great drops of sweat stood in his face. He asked frequently for water; the stomach seemed distended with gas. "I was able to make but a very slight examination because of his writhing and tossing about. He was pulseless at the wrist and the extremities were cold to the knees and elbows. On placing my ear to the precordia, I could faintly hear a muffled splashing sound of the heart, during the systole only. I could hear no second sound." No pain was present, only a sense of smothering. Death occurred one and a quarter hours after the attack. Postmortem examination showed pericardium filled to bursting with blood. There was a three fourths inch jagged rupture in the wall of the left atrium.

In estimating the survival period after atrial rupture, one is handicapped by difficulty in differentiating the symptoms of the underlying condition from those of actual rupture. Nevertheless, pathologic evidence such as pericardial reaction, and organization of the pericardial clot, indicate that some patients with atrial rupture have survived up to nine weeks. This condition, however, is always fatal, with 61 per cent of the patients dying instantaneously after the rupture.

Barnes states that atrial rupture usually leads to more rapid death than does ventricular rupture, since extravasation occurs more frequently in the former condition. Our data, however, indicate that death is less rapid after atrial rupture than after ventricular rupture, since 15 per cent of patients with atrial rupture have survived over 24 hours. In ventricular rupture, less than 2 per cent of patients live 24 hours. Cases of traumatic atrial rupture usually have the longest survival period. The case reported by Rixford (case 36), is a striking example of this; the patient was kicked in the chest by a cow and survived nine weeks. During this period, three pericardial taps yielded two quarts of old blood.

The electrocardiogram may aid in the diagnosis of atrial rupture by indicating atrial infarction prior to the rupture and pericarditis after the rupture. In a series of 31 patients with atrial infarctions without rupture, Cushing and co-workers noted atrial extrasystoles, atrial fibrillation, wandering pacemaker, and nodal rhythm in 23 who were subjected to electrocardiographic study. Occasionally, they found depression of the P-Q segment. Similar arrhythmias, except for atrial fibrillation, occurred in experimental infarction of the atrium in dogs. In Söderström's series of 47 cases of atrial infarction, atrial fibrillation developed in one-third of the patients with regular sinus rhythm prior to the infarction. Rapid auricular fibrillation also was recorded in the electrocardiogram of our patient (fig. 1).

Although elevated or depressed P-Ta segments, especially in leads II and III, are diagnostic of atrial infarction, these changes are difficult to see unless complete heart block is present. Depression of the P-Ta segment in leads II and III indicates infarction of the anterolateral portion of the right atrium. It occurs more frequently than P-Ta elevation, which indicates involvement of the posterior wall of the atria. Söderström suggests that an abnormally short P-R (in the absence of a Wolff-Parkinson-White syndrome) with upright P waves may be a sign of infarction of the right atrium.

The electrocardiographic pattern of pericarditis may occur after atrial rupture because of blood in the pericardial sac. Randles, Gorham and Dresbach described S-T segment elevation in all limb leads of dogs in which atrial rupture was produced experimentally. This pattern was undoubtedly the result of pericarditis. Similar S-T elevations occurred in the electrocardiogram of the patient with atrial rupture reported by Clowe, Kellert, and Gorham.

A roentgenogram of the heart after atrial
rupture may indicate pericardial effusion by an enlarged heart shadow. Aspiration of blood from the pericardial sac will also confirm the diagnosis of cardiac tamponade.

Pathologic Physiology of Atrial Rupture

Two theories have been advanced to explain the production of atrial rupture. The first assumes that an increased intra-atrial pressure leads to a bursting of the wall of the chamber. The second postulates a direct tearing of diseased atrial musculature by the force of its own contraction. Probably a combination of both factors is involved in atrial rupture. In patients with atrial fibrillation who develop atrial rupture, the increased intracavity pressure is perhaps more important than the muscular contraction factor, although Prinzmetal and his co-workers have demonstrated small muscular contractions in fibrillating atria.

Variations in the anatomy and intraluminal pressure of the atria may influence the development of atrial rupture. Söderström points out that the endocardium of the trabeculated portion of the right atrium is extremely thin, consisting merely of endothelium in contact with perimysial connective tissue of the myocardium. In the smooth-walled parts of the atria, the myocardium normally varies from 1 to 3 mm. in thickness. Some portions of the right atrium actually lack myocardial tissue between endocardium and pericardium. The fact that the right atrium is generally thinner and less muscular than the left probably accounts for the increased incidence of rupture in the right atrium.

Although the atrial blood supply exhibits considerable anatomic variations, branches of the right coronary artery arising near its origin customarily nourish the wall of the right atrium. Branches of the left circumflex artery supply the left atrial wall. In addition, extracoronary anastomoses are present between the atria and the vasae vasorum of the venae cavae and the pulmonary veins. It has also been suggested that the atria may obtain nutrition directly from their lumina.

Cushing and associates ascribe the higher incidence of right atrial infarction to the lower oxygen content of the blood in the right atrium. A second but more important fact is that thrombosis of the right coronary artery usually occurs in the proximal 2 or 3 cm. which also occludes the branches supplying the right atrium. This is more likely the reason for the higher incidence of right atrial rupture than a difference in the oxygen content of blood in the two atria.

An increase in the intraluminal pressure also appears significant in any rupture of the heart. The lower incidence of atrial rupture, as compared with ventricular rupture, seems closely related to the much lower intraluminal pressure of the atria which, in the right atrium, varies from −2 to +2 mm. Hg and, in the left atrium, is only slightly higher. Considerably higher atrial pressures may be encountered in patients with pulmonary disease or valvular heart disease, especially if they have congestive heart failure or atrial fibrillation.

In the presence of lower atrial pressures, some ruptures may be accompanied by “slow leaks” which may, occasionally, even seal off with a clot. Examples of this are to be found especially among traumatic cases: Case 23, 7 days; case 25, 7 days; case 34, 12 days and case 36, 9 weeks. In experimental atrial rupture produced in dogs, it has been clearly shown that clot formation and healing are so rapid that a saw-string suture is necessary to insure bleeding after 24 hours. On the other hand, all five instances of atrial rupture recorded in patients with valvular heart disease (and, presumably higher atrial pressures) ended in sudden death.

Several attempts have been made to calculate mathematically the pressures influencing rupture. These studies suffer from oversimplification, since this proves to be a highly complex problem with many interdependent variables.

Sex and Age Factors

Atrial rupture, in contrast to ventricular rupture, occurs about twice as frequently among males as females in almost all groups listed in table 2; 68.6 per cent of patients with
TABLE 2.—Incidence of Atrial Rupture According to Sex

<table>
<thead>
<tr>
<th>Sex</th>
<th>Infarct</th>
<th>&quot;Fatty Degeneration&quot;</th>
<th>Trauma</th>
<th>Valvular</th>
<th>Tumor</th>
<th>Aneurysm</th>
<th>Others</th>
<th>Total</th>
<th>%</th>
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<td>17</td>
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<td>2</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>11</td>
<td>22</td>
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<tr>
<td>Totals</td>
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<td>19</td>
<td>5</td>
<td>3</td>
<td>3</td>
<td>23</td>
<td>70</td>
<td>100.0</td>
</tr>
</tbody>
</table>

* May be considered as infarct.  † 10 cases recorded without notation as to age.

TABLE 3.—Incidence of Atrial Rupture According to Age

<table>
<thead>
<tr>
<th>Age</th>
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<th>&quot;Fatty Degeneration&quot;</th>
<th>Trauma</th>
<th>Valvular</th>
<th>Tumor</th>
<th>Aneurysm</th>
<th>Others</th>
<th>Total</th>
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<td>—</td>
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<td>—</td>
<td>1</td>
<td>2</td>
<td>3.3</td>
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<td>60-69</td>
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<td>—</td>
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<td>2</td>
<td>13</td>
<td>18.6</td>
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<td>6</td>
<td>10.0</td>
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<td>1</td>
<td>5</td>
<td>—</td>
<td>—</td>
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<td>3</td>
<td>11</td>
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<td>30-39</td>
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<td>4</td>
<td>3</td>
<td>—</td>
<td>—</td>
<td>5</td>
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<td>3</td>
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* May be considered as infarct.  † 20 cases with inadequate data reported.

Atrial rupture were males and 31.4 per cent were females. On the other hand, Oblath and co-workers found 58.75 per cent of their 80 patients with ventricular rupture were women and only 41.25 per cent were men.28a

It is also of interest that the collected series of 17 atrial ruptures following atrial infarction includes five patients below the age of 50 while no patient was below 50 years of age in the series of ventricular ruptures reported by Oblath and colleagues.28a The reasons for the sex and age differences between atrial and ventricular ruptures are not readily explained. The small numbers involved may not be statistically significant.

Tables 2 and 3 are necessarily incomplete because of inadequate reporting of the earlier cases in the literature.

Treatment

No treatment of atrial rupture has been of avail. With the advent of modern cardiac surgery, repair of the atrial rupture becomes feasible if the diagnosis is made in time and the leak is a slow one.11 A high index of suspicion of atrial rupture in a patient with atrial infarction or cardiac tamponade may conceivably some day lead to an earlier diagnosis and possible surgical treatment. Aspiration of blood from the pericardial sac to relieve cardiac tamponade is of little value unless surgical repair follows.

Summary

1. An additional case of atrial rupture in a 64 year old woman is reported.
2. Seventy-nine cases of atrial rupture have been collected, classified and studied, making a total of 80, up to Jan. 1, 1953.
3. Atrial infarction and trauma are the chief causes of atrial rupture, accounting for 17 and 19 cases, respectively.
4. Obliterative endarteritis of the atrial branches of the coronary arteries rather than thrombotic occlusion is often the inciting factor of atrial infarction and will be found more often if sought for at autopsy.
5. In contrast to ventricular rupture, atrial rupture occurs more frequently in men than in women. Although sudden death is the rule, survival up to nine weeks has been observed. Contrary to a previously published
6. The diagnostic features and pathologic physiology of atrial rupture are discussed.

7. No case of atrial rupture in which the correct diagnosis was made ante mortem has thus far been recorded. Electrocardiographic changes occasionally occur which should arouse suspicion of this diagnosis. Only one instance of such suspicion was found in the literature.

8. A high index of suspicion of atrial rupture in a patient with atrial infarction or cardiac tamponade may conceivably some day lead to an earlier diagnosis and possible surgical treatment.

**ACKNOWLEDGMENTS**

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