

lesion is readily overlooked. A sharp first sound, evidence of enlargement of the right heart, sometimes a gallop rhythm, together with a small pulse should suggest stenosis, and with recovery the thrill and murmur often reappear.

**Diagnosis.**—A presystolic murmur is occasionally heard in conditions other than mitral stenosis.

The Flint murmur has already been referred to under aortic incompetence. In an adherent pericardium and in vegetative endocarditis of the mitral valve, a murmur of this character is sometimes found, usually of the low rumbling variety rather than of the loud and rough character. Ewart and Rutherford have recorded a case in which all the typical signs of stenosis were present and in which the autopsy showed a fibrous polyp hanging from the auricle through the mitral orifice.

**TRICUSPID REGURGITATION.**—Regurgitation at this orifice is very generally admitted to occur in health, resulting from conditions, such as violent exercise, which temporarily raise the blood pressure in the pulmonary circuit. This is often spoken of as the safety-valve action, preventing the lungs from becoming unduly congested.

In the great majority of instances, tricuspid incompetence is the result of dilatation of the right ventricle, inducing relative incompetence at this orifice; in only a comparatively few cases does the lesion depend on organic changes in the valves.

Dilatation of the right ventricle is always a secondary process. It constantly occurs in old-standing mitral lesions, and as the result of obstruction in the pulmonary circuit, from emphysema or fibrosis of the lungs. In dilatation of the left ventricle, from whatever cause, the right ventricle is commonly included in the morbid process.

The valve may be affected by endocarditis, acute or chronic, or by degenerative changes. Under these circumstances the valves of the left heart are almost invariably involved by similar lesions. In malignant endocarditis, the valve is occasionally affected alone. In thirty-nine instances, Pitt found ten in which this valve was alone attacked.

**Symptoms.**—These are similar to those found in mitral disease, and consist in breathlessness, with evidence of obstruction in both the systemic and the portal venous systems.

**Physical Signs.**—A systolic bruit is sometimes heard in the lower sternal region, having its maximum intensity just to the right of the sternum about the fourth and fifth ribs, and occasionally transmitted to the right as far as the parasternal line. It is not heard at the apex, although it may be difficult to differentiate it from a co-existing mitral murmur. In some instances a difference in timbre may be noticed. Owing to a debilitated condition of the right ventricle the murmur may be absent even in well-marked cases, and again, it may disappear under the influence of rest and digitalis. The enlargement of the right ventricle causes increased pulsation in the epigastric and lower sternal regions. Dulness extends to the right border of the sternum or for two or three finger-breadths beyond, although it is frequently masked by emphysema.

Pulsation in the veins, accompanying each systole of the heart, forms important evidence of the condition under discussion. It depends on a reflux of blood in the dilated veins after their valves have become incompetent. It is seen in the external and internal jugulars, and sometimes even in more distant veins such as the brachial. Care must be taken not to confuse true venous, or, as it is sometimes termed, positive pulsation, with certain undulatory movements of these vessels. If the vein is emptied from below, positive pulsation continues.

Venous pulsation is also observed in the liver. The enlarged organ, grasped between the hands, presents a true expansive pulsation, and not merely a transmitted epigastric impulse from an enlarged right ventricle.

**TRICUSPID STENOSIS.**—This condition, although rare, is more frequent than are lesions at the pulmonary orifice. Pitt found it 87 times in 12,000 autopsies.

Congenital stenosis is not uncommon in still-born children, or in those who have lived a few days. It is frequently associated with other cardiac abnormalities or defects.

It has been asserted that almost all cases are of congenital origin, but this view is not borne out by statistics. In Pitt's series of 87 cases, only one patient died under ten, and in Herrick's tabulation of 40 cases the youngest patient was twelve years old, with a history of acute rheumatism two years previously, facts which strongly suggest an acquired origin. In the great majority of cases, tricuspid stenosis is associated with a similar process in the mitral, and often with disease of the aortic segments as well. In Leudet's series of 114 cases the tricuspid valve was affected alone in only one instance, and in Herrick's once in 40 cases. Females are affected about four times as often as males. A history of acute rheumatism was present in 10 or 25 per cent. of Herrick's series, chorea in 2, doubtful rheumatism in 4, no rheumatism in 9, and in 15 there was no record. In Pitt's cases, however, rheumatism played a predominant rôle in the etiology.

A fatal issue usually results in patients who are under thirty, and in only 12 cases out of 151 (Leudet's and Herrick's series) did the patient reach the age of fifty. The symptoms are those of mitral stenosis of the most pronounced form. Dyspnoea and cyanosis are often extreme, and Broadbent regards pronounced dropsy as evidence of the disease. The diagnosis has been made in a few instances during life, but in the great majority of cases it has been overlooked.

A presystolic murmur and snapping first sound heard at the ensiform cartilage or a little to the right, and fading off and again becoming marked at the mitral, has been correctly interpreted as indicating stenosis of both orifices, and in a few cases a thrill has been detected in the tricuspid region. The murmurs in the tricuspid and mitral regions have also been noted as having a different character, but the characters of the cardiac sounds are often rendered difficult of interpretation owing to rapid or irregular cardiac action. The heart is enlarged to the right as in mitral stenosis, whilst both the venous and the hepatic forms of pulsation are often present owing to associated tricuspid regurgitation.

**PULMONARY ORIFICE.**—Disease of the pulmonary valves is usually congenital, and is then commonly associated with other cardiac anomalies. Only a few of these cases reach adult life.

The majority of acquired cases are due to malignant endocarditis. Congenital absence of one of the cusps may lead to sclerotic processes in later years, as in aortic disease.

Acute rheumatism is not a frequent cause, being present in only seven of twenty-nine cases collected by Gerhardt.

Obstructive disease is difficult to recognize owing to the frequent occurrence of systolic murmurs in the pulmonary area from other causes. A distinct thrill, a harsh murmur, and evidence of enlargement of the right ventricle are the signs which may be found, but similar signs may be caused by the pressure of an aneurism or tumor on the trunk of the vessel.

Incompetence gives rise to a diastolic murmur in the pulmonary region, often louder during expiration, and transmitted down the left sternal border. It is not heard toward the apex or in the vessels of the neck.

Enlargement of the right ventricle is usually considerable and the apex is pushed to the left. There is an absence of the arterial phenomena of aortic disease, but Gerhardt has shown that graphic representations of the pulmonary pulsations show analogous changes. By introducing a manometer into one nostril, closing the opposite one by pressure, and directing the patient to cease breathing, he obtained a tracing showing larger excursions of the lever, and a more sudden rise and fall than is obtained either in health or in aortic incompetence.

Cyanosis, venous enlargement, and pulmonary embolism have also been noticed in disease of this orifice.

Tuberculosis is not an uncommon mode of death, and is attributed to the anæmic condition of the lungs.

**COMBINED VALVULAR LESIONS.**—In a large proportion of cases more than one valve is affected, or there may be both incompetence and obstruction at one orifice. The general features and signs usually point to predominant disease of one valve. Aortic and mitral incompetence are often associated, whilst mitral stenosis is frequently accompanied by slight sclerosis of the aortic segments, having no clinical significance.

**PROGNOSIS OF VALVULAR DISEASE.**—The prognosis is based partly on an estimate of the extent and character of the valvular lesion, partly on the general health and habits of the patient.

Aortic incompetence is usually regarded as the most serious of valvular affections. The arterio-sclerotic cases, owing to the frequency with which the coronary vessels are involved, are more serious than stationary rheumatic conditions. Of the valvular affections, aortic incompetence is the one in which sudden death is most apt to occur. Favorable features are a moderate degree of cardiac enlargement, a pulse not showing the collapsing character to a high degree, a distinct second sound in the arteries of the neck, and an absence of cardiac symptoms. According to N. S. Davis the average duration of life after compensation begins to break down is 3.8 years, a period which closely corresponds to Balfour's estimate of four years.

Aortic stenosis is usually regarded as the least serious of valvular lesions. In moderate grades of the disease but little disturbance of function takes place. More serious, however, are the cases with evidence of a high degree of stenosis, as shown particularly by the pulse. Arterio-sclerosis always suggests the possibility of disease of the coronary arteries, and anginal attacks convert such a suggestion to a certainty.

Sudden death is rare, although it may occur from disease of the coronary vessels, and has also resulted from occlusion of an extremely narrowed orifice.

Mitral lesions are on the whole less serious than aortic regurgitation. Stenosis is usually regarded as a more serious affection than incompetence. Osler regards the latter as almost as serious as the former, whilst N. S. Davis reverses the opinion usually held.

In many cases in which a mitral systolic murmur is heard at the apex, this lesion shows no tendency to shorten life, and in the absence of cardiac symptoms, and of other signs of cardiac disease, its presence may be regarded with but slight apprehension. More serious are the cases in which a considerable degree of regurgitation is revealed by marked enlargement of the ventricle, but even here the patient may enjoy a long period of good health, provided that symptoms of cardiac disturbance are absent.

A cardio-pulmonary murmur is not uncommon at the apex, and is heard best or exclusively at the end of inspiration. It is frequent in pulmonary tuberculosis, and in the absence of other evidence of cardiac disease may be disregarded.

The mere loudness of a murmur is in itself no index to the severity of the lesion. Very soft murmurs are often heard in failing hearts, whilst loud rasping murmurs, although indicative of valvular damage, may be present with perfect compensation.

Mitral stenosis is not incompatible with longevity, but in most cases there is a marked shortening of life. Broadbent found the average age at death to be thirty-three years in males and thirty-eight in females, and other estimates agree closely with this. Of other influences affecting the duration of cardiac disease experience shows that children bear these lesions badly, and such cases frequently terminate fatally at or shortly after puberty. Females bear valvular disease better than males, a fact which is probably to be attributed to their less active habits and to their less frequent indulgence in alcoholic excess. An exception to this rule is sometimes seen as the result of pregnancy, in which the increased strain occasionally results in serious and even fatal cardiac failure.

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Laborious occupations are unfavorable, as are also sexual and alcoholic excess, and the free use of tobacco.

Finally poor general health, anæmia, recurring attacks of bronchitis, hepatic and renal engorgement, and pulmonary embolism are all unfavorable signs.

Recurring attacks of rheumatism often exert a most unfavorable influence owing to fresh attacks of endocarditis, and in children rheumatic inflammations occasionally progress to a fatal issue within a few weeks or months.

Acute disease, particularly pneumonia, is often ill borne, and if survived is sometimes followed by loss of compensation. I have known several cases of mitral stenosis date their symptoms from an attack of pneumonia.

Finally, poor general health, anæmia, hepatic or renal engorgement, and pulmonary embolism are all unfavorable symptoms.

**TREATMENT.**—During the period of compensation no very active treatment should be directed toward the heart.

It is important in this stage to keep the general health up to the best possible point. For this purpose the diet should be plain and nourishing, and the coarser vegetables should be avoided as tending toward flatulence. Alcohol is unnecessary in young people, and if used at all should be taken in strict moderation with food. Tobacco is injurious, whilst tea and coffee must be avoided should they cause any cardiac palpitation or uneasiness.

In rheumatic cases special care should be taken to see that the patient is suitably clad, and even with slight rheumatic manifestations he should be confined to bed or the house.

Exercise and athletics are beneficial provided they cause no undue fatigue or are not pushed to the point of inducing dyspnoea. The more violent forms of exercise such as football or even tennis should be forbidden, but golf, mild bicycling, or rowing and walking are safer, inasmuch as they throw much less strain on the heart. In cases in which for any reason it is judged unwise to allow even easy exercise, rest in the open air is beneficial.

Medicinally, an occasional aperient; tonic doses of iron or arsenic, particularly when anæmia is present, or strychnine are valuable. Small doses of digitalis (℥ v. to x.) may be administered should any tendency to dyspnoea or palpitation exist, and the use of this drug is sometimes continued for lengthened periods with decided benefit.

When compensation begins to fail, and dyspnoea, dropsy, and palpitation become marked, a more active plan of treatment is required.

In most instances of uncomplicated valve lesions failure of compensation is due to dilatation, brought about for the most part by excessive muscular work, although in some cases it is induced by severe mental strain. The first object is therefore to secure physical and sometimes mental rest. When the symptoms are at all severe, absolute rest in bed must be enjoined; and treatment by this method alone is sometimes sufficient to relieve dyspnoea and remove dropsy. It is, however, advisable in most instances to use medicinal or other agents, which hasten recovery and alleviate distress.

Of the cardiac stimulants by far the most valuable is digitalis. This remedy slows and strengthens the muscular contraction of the heart, whilst the pulse rate is reduced and becomes more regular, the peripheral arteries are contracted, the renal arteries relax, and a large volume of blood passes through the kidneys as shown by the free diuresis.

In aortic incompetence the objection to digitalis is sometimes raised that it prolongs diastole and hence allows of freer regurgitation. This objection is largely theoretical, and many cases receive a marked degree of benefit from the increased muscular action, dyspnoea is relieved, and in favorable cases compensation is completely restored. It is in mitral disease, particularly when dropsy and dyspnoea are present, that digitalis induces the most brilliant results. Dyspnoea is relieved,

free diuresis is set up, and dropsy lessens and disappears.

In mitral incompetence, not only is the muscle stimulated, but the regurgitation is lessened owing to the more perfect closure of the auriculo-ventricular ring. In stenosis the lengthened diastolic period allows more time for blood to pass from the engorged auricle through the narrowed valve.

The action of digitalis is somewhat slow and it is not until the third day that the full effects of the drug are seen. When marked slowing of the pulse to 60 or under, or a fall in the quantity of urine occurs, the dose should be lessened or stopped, owing to the danger of toxic manifestations, which reveal themselves in the form of nausea and vomiting, weakness and irregularity of the pulse, or a double beat of the heart to one of the pulse.

The drug should be given freely for from forty-eight to seventy-two hours when the symptoms are at all urgent—say in doses of  $\mathfrak{m}$  xv. of the tincture or 3 iv. of the infusion every three or four hours. Smaller doses are usually sufficient ( $\mathfrak{m}$  x.-xv. t.i.d.) in the absence of severe symptoms. Nausea and vomiting are sometimes produced by even moderate doses, and in such cases rectal administration of one or more large doses of the fluid extract are often extremely beneficial. Five or ten minims ( $\mathfrak{m}$  i.-gr. i. digitalis) administered with starch in this way often act rapidly, and I have seen the pulse slowed and dyspnoea relieved in the course of a few hours. An old and reliable preparation is Guy's pill, consisting of gr. i. each of digitalis, squills, and pil. hydrarg.

Strophanthus is a valuable substitute for digitalis. It acts on the heart like digitalis, but has not the same tonic effects on the arteries. It is consequently sometimes preferred in arterio-sclerotic cases with increased arterial tension. It is inferior to digitalis in removing dropsy, but is sometimes of value in alleviating cardiac symptoms when this drug fails. Sparteine acts in a similar manner to strophanthus, but as a rule is inferior to it.

Caffeine is occasionally useful as a substitute for digitalis. It sometimes induces restlessness and insomnia.

Suprarenal extract has recently come into use as a cardiac tonic; it acts on the muscular substance of the heart. Some of the clinical reports are very encouraging and it seems worthy of further trial.

Strychnine is a valuable adjunct to digitalis and may be administered in doses of gr.  $\frac{1}{16}$  three to six times daily.

Diffusible stimulants such as alcohol, ether, and ammonia are of value in emergencies, and until the more slowly acting stimulants affect the heart.

A dose of calomel, gr. v. with gr. x. of bicarbonate of soda, is often administered with benefit preceding the course of digitalis. It relieves portal congestion and engorgement of the right heart, and increases the efficacy of digitalis.

In arterio-sclerotic cases the long-continued administration of iodides often proves of much value in controlling palpitation and other symptoms, possibly by lowering peripheral resistance. Nitroglycerin is also a valuable agent in lessening peripheral resistance and is often advantageously given with digitalis in doses of  $\mathfrak{m}$  i. of the one-per-cent. solution.

Obstinate or persistent symptoms often require special measures for their mitigation or relief.

Dyspnoea continuing after the use of digitalis, and especially when nocturnal in character, is usually relieved in a remarkable manner by a hypodermic injection of morphine, and in advanced cases this drug is invaluable.

Sleeplessness sometimes yields to paraldehyde, trional, or sulphonal, but, as it is often dependent on dyspnoea, these remedies are much inferior to morphine.

Pain over the cardiac region is often alleviated by the iodides, especially in arterial cases, or by nitroglycerin.

Dropsy.—Dropsy may be diminished by free purgation. For this purpose calomel, salines, and hydragogues, especially compound jalap powder, are most serviceable.

Needle puncture of the distended limb with strict aseptic precautions allows considerable quantities of the fluid

to drain away. Southey's tubes are also very efficacious, but there is considerable danger of inflammatory changes taking place in the subcutaneous tissues.

Theobromine in doses of gr. lx. to gr. xc. daily may be used with occasional benefit as a diuretic when the urinary secretion is persistently small.

When considerable quantities of fluid collect in the abdominal or the pleural cavity paracentesis is necessary and may be repeated as often as required.

Palpitation is much relieved by an ice-bag over the precordial region. A diffusible stimulant, or strychnine in full doses often proves of signal service. If the attack is severe, a hypodermic injection of morphine may be given. Strict attention to diet together with quiet and rest is also essential.

Vomiting.—Food must sometimes be withheld for some hours, or replaced by sips of hot water, brandy, or champagne. After the vomiting has yielded or lessened, liquid food may be cautiously tried. Milk may be given freely diluted with lime water, Vichy or effervescing waters, or peptonized beef essences are often tolerated when other foods are rejected. In obstinate cases rectal alimentation may become requisite. The frequent action of digitalis in inducing vomiting must be remembered in this connection. It must often be withheld for a period, but should not be given up without good reason.

Venesection is occasionally required when marked cyanosis or dyspnoea is present, and ten, twenty, or even thirty ounces of blood may be abstracted from the arm. A valuable measure which has largely fallen into disuse is the application of a few leeches to the precordial region followed by hot fomentations to encourage bleeding.

During the past few years much has been written on the Nauheim methods, consisting in bathing in aerated water, and movements with resistance. The baths lessen the peripheral resistance, and the movements tend to empty the veins. Percussion and skiagraphs show a lessening of cardiac dilatation after application of these methods. They seem to be more successful at the natural springs than when applied in hospitals or other localities.

Frederick G. Finley.

**HEART, RUPTURES OF.**—These are divided into two classes: *First*, Those due to degeneration of the myocardium, the so-called spontaneous ruptures; and *second*, those due to traumatism.

Instances of the first class, while not frequent, are more often observed than are those of traumatic origin.

**MORBID ANATOMY.**—Spontaneous rupture of the heart generally occurs in the substance of the left ventricle owing to the greater frequency of degenerative changes in this locality, and to the greater strain which this part of the heart has to bear in lifting the blood through the systemic circulation.

The anterior face of the ventricular wall somewhat above the apex is usually the site of the tear. Although it is said that the tear usually begins from within and proceeds to the outer face, the reverse may happen, and it is impossible to tell from inspection upon which surface the lesion originated. The sites of the rupture in 55 cases collected by Elleaume were as follows: 43 in the left ventricle, 7 in the right, 3 in the right auricle, and 2 in the left auricle.

The tear may be complete or incomplete, and may vary in extent from a centimetre to the length of the ventricle. It is usually single, but as many as five have been noted, sometimes intercommunicating. The rent is generally ragged, irregular, and sometimes echymosed at the edges. Its course through the ventricular wall may be tortuous so that the endocardial and epicardial openings do not correspond. Usually the fissure runs parallel to the course of the muscular fibres. Those ruptures which are due to an abscess of the heart wall or to hemorrhagic softening more frequently are in the form of an ulceration or perforation. The immediate result of this accident is the distention of the pericardial sac with blood which generally forms clots, leaving the serum in the pericardium.

Cases have been noted in which a plug of fibrin has more or less completely occluded the tear in the heart wall.

**ETIOLOGY.**—This form of rupture is always the result of disease of the myocardium, softening, fatty degeneration, fatty infiltration, etc., dependent generally upon occlusion or fibrosis of the coronary arteries. This degeneration may be local or general, and may result primarily in an aneurism of the heart or aorta, which may burst. Very rarely we have abscess of the heart wall, which bursts and leads to rupture. The *exciting* cause of the lesion may be a sudden and perhaps an unusual motion like stumbling or falling, lifting or straining. A comparatively frequent cause is running for a train. Anger, excitement, and mental or physical shock may determine the accident. Not infrequently it occurs while the patient is in perfect rest. He may be asleep in bed. The male sex is considerably more liable to this accident than the female. It seldom occurs before the fiftieth year, more often after the sixtieth.

**SYMPTOMS.**—Seldom are the premonitory signs of this disaster distinctive. There would naturally be signs of a diseased and feeble heart. Comparatively few diseased hearts, however, go on to rupture. On the occurrence of the lesion, death is usually instantaneous; the patient not infrequently being found dead in bed with the clothes not even disturbed. In case the rupture is oblique in direction and small in extent, the patient may survive several hours, or even days.

Dr. Langman has recently reported a case of a man who had two ruptures of the heart wall, one having happened two weeks before the other. He died four hours after the occurrence of the second, which was in the left ventricle, the first was in the right. The heart walls were thin, but not fatty.

The special symptoms denoting approaching dissolution are intense precordial pain, restlessness, dyspnoea, rapid, feeble, and fluttering pulse; vomiting, cyanosis, loss of consciousness, and convulsions.

Hampeln has observed that there is greater pain in rupture of the pericardial portion of the aorta than in that of the heart substance proper.

The physical signs, even when the patient's life is sufficiently prolonged, are more or less indefinite. The pulse is weak and intermittent. The heart sounds are muffled, distant, and imperfectly developed, with probably some increase of pericardial dullness.

**DIAGNOSIS.**—This is so uncertain that it cannot as a rule be relied upon, although in some cases the condition has been made out ante mortem. A diagnosis ought certainly to be made in those cases in which, in addition to the rational symptoms already detailed, percussion affords evidence that the pericardial area has recently undergone enlargement.

**PROGNOSIS.**—This is uniformly unfavorable; no case of spontaneous heart rupture having ever been known to recover, although life may be prolonged for several days and even weeks, by judicious treatment and nursing.

**TREATMENT.**—This is mainly palliative and can have no effect on the diseased condition of the heart. Cold applications, morphine hypodermatically, a strict maintenance of the recumbent posture, perfect quiet, and warmth to the extremities may prolong life for a limited period. Stimulants are contraindicated.

**Traumatic Ruptures of the Heart** are quite rare. This organ from its shape and position, its mobility and the protection afforded it by the elasticity of its walls, and by its overlying and surrounding structures, is seldom ruptured—except in cases in which the entire chest wall is crushed in. A certain number of cases of rupture of the heart walls have occurred with comparatively little injury to the chest wall, even without penetration of the pericardial sac. The writer was able, in 1899, to collect forty-five cases of heart rupture, not due to stab or gunshot wounds, and generally speaking caused by an injury from the effects of which the heart walls would be expected to escape scot free. In the majority of the cases there was evidence of disease or abnormality of the organ. But in a few cases the heart muscle was healthy.

The writer reported the case of a vigorous man of twenty-eight who was riding his bicycle rapidly, when the forward wheel struck a hose pipe which was filled with water and which lay across the road. As a result of this sudden strain the bicycle broke at the "head," and the rider was thrown to the ground with great violence, the detached handle-bar post interposing itself between his body and the hard road. This impact fractured the sixth costal cartilage somewhat to the left of the sternum and drove the distal end attached to the rib into the pericardial sac. The sac did not tear; but the force of the blow was so great that the right auricle was torn entirely through. A triangular flap (see accompanying photograph, Fig. 2596) of the heart wall had been lifted up,

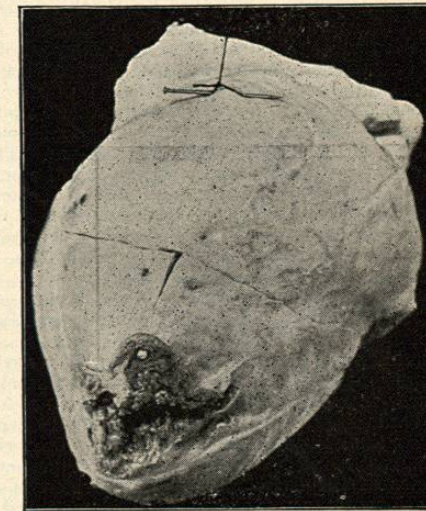


FIG. 2596.—Showing Traumatic Rupture of Heart at Apex of Right Ventricle.

and when it was turned back the ventricular cavity was exposed. The heart weighed eleven and one-quarter ounces, and its valves and muscular substance were healthy and competent. The man lived for an hour and a half after the accident and partly recovered from shock. The cause of death was apparently the distention of the pericardium with blood, of which from eight to ten ounces, mostly coagulated, was found in the sac.

Of traumatic ruptures a majority occur in the right ventricle near its apex. Of Gamgee's 28 cases, in 9 there was no fracture of ribs or sternum and either no bruise of the parietes or a very slight one. The pericardium was intact in at least one-half of the cases, and in 22 in which the principal seat of the injury was noted, the right ventricle was torn in 8; the left in 3. The left auricle was injured in 7 and the right in 4. The apex of the right ventricle is a favorite spot for traumatic rupture, first, on account of its exposed position as it comes in contact with the chest wall in systole, and second, because the myocardium is thinnest at this point. Ruptures of the muscular fibres most frequently occur in systole when they are tense and firm and offer the greatest resistance to the impact of the opposing force. If struck in diastole when the ventricles are filled with blood, their walls being flaccid, the seat of rupture will be in the resisting valves, or on the interventricular septum.

So far as ascertained the longest time that a person has survived after traumatic heart rupture is seventeen hours. The symptoms of intense pericardial pain, weak, fluttering or intermittent pulse, dyspnoea, lividity, cold extremities, sweating, and convulsions, are much the same as in cases of spontaneous rupture. With the history of a blow upon the chest, especially if the ribs or their car-