Traumatic Heart Disease
Nonpenetrating Injuries

By R. W. Kissane, M.D.

In 1764 Mark Akenside reported the first unquestionable case of myocardial contusion due to a nonpenetrating chest injury. He treated a 14 year old boy who was struck between the ribs by a plate. The boy died six months after the blow; the autopsy revealed an area of necrosis in the left ventricle the size of a half crown extending nearly through the heart wall and covered by adherent pericardium. During almost 200 years, from 1676 to 1868, only 27 cases of traumatic heart disease were reported in the literature. In 1868 Fisher published an impressive description of 76 cases, of which 69 dealt with traumatic rupture and only seven with myocardial contusion. From 1900 to 1937, 108 cases of nonpenetrating injuries to the heart were reported at which time Urbach, in a review of 1000 autopsies of contusion of the chest, found 185 instances of injury to the heart. In 1940 Leinoff reported 50 nonselected consecutive cases of fatal automobile accidents of which eight (16 per cent) showed macroscopic evidence of cardiac damage. In 1936 Glendy and White reported a case of a nonpenetrating wound of the heart, and stated that among 7600 autopsies at the Massachusetts General Hospital not a single case of cardiac trauma of any type was recorded. However, Osborn, in a pathologic study of 202 accidents, found 19 cases of nonpenetrating traumatic lesions of the heart.

Since the year 1930, I have collected 158 autopsied cases of traumatic heart disease due to nonpenetrating injuries. No statement as to general incidence can be made since a large number of these cases or specimens were sent to me because of my interest in the subject. There is nothing to indicate a wide fluctuation in the incidence of myocardial contusion over the centuries, although an increase would be expected with the advent of the automobile and increase in traffic accidents. The reported incidence of traumatic heart disease due to nonpenetrating injuries undoubtedly has been influenced by the general opinion of the medical profession which progressed from considering the condition as impossible or extremely rare to finally accepting it as a possibility in about 15 per cent of all fatal chest injuries.

Experimentally, tears, pericardial and epicardial hemorrhages, were the most frequent injuries produced by blows to the anesthetized dog. They were usually due to moderate blows uncomplicated by myocardial contusion. The anatomic relationship, attachments and structure appear to predispose the pericardium to injury. Similar conditions prevailed in 58 autopsied cases with mild to moderate chest trauma, but with severe, fatal, extracardiac injury; there was subpericardial hemorrhage in 55, pericardial hemorrhage in 21, and small pericardial or epicardial tears in 10. All cases died within three weeks. The subpericardial hemorrhages were usually anterior and varied in size from that of small petechiae to large bruised areas. Subpericardial fibrosis was a frequent finding in a similar group of patients who lived 6 to 18 months after the accident. Uncomplicated hemorrhages of the pericardium and subpericardium, while infrequently found.
at autopsy due to the tendency of patients to recover, probably produce temporary symptoms and electrocardiographic changes.\(^8\)

In the milder instances of trauma the pericardial tears vary from small slits in the visceral pericardium to rents in the pericardial sac. The lesions are generally related to the point of compression against the spinal column due to anterior thoracic trauma. In severe trauma from crushing or falls from heights, the compression force is transmitted more widely and lesions due to bursting may occur some distance from the line of force. There is also a bursting from within due to compression of the great vessels and re-expanding tension of the fluid mass which may rupture the myocardium as well as the pericardium. When cardiac bursting occurs, the wound is invariably larger on the outside with sharp, clean-cut edges, and is lengthwise in the ventricles. Violent displacement of the heart, usually as a result of a fall from high places or other forcible impact, appears to cause tears at the entrance or exit of the great vessels.

Pericardial rupture may be complicated by infection or pneumopericardium due to communication with adjacent injured air containing organs, or by hemopericardium due to myocardial rupture. While herniation of the heart through the torn pericardium is rare, strangulation and sudden death from this cause are observed.

Healing of the minor injuries is usually without adhesion formation. In the more extensive injuries, however, adhesions range from a single fibrous band to adhesive pericarditis with complete obliteration of the sac. Hemorrhage into the posterior mediastinal space frequently is overlooked, but is important because it may cause constriction of the superior vena cava or its branches, the trachea, the bronchi, the nerves in this space, the esophagus, or fixation of the heart in the chest.

There is close similarity between the lesions of myocardial infarction and myocardial contusion.\(^9\) If there is a question of trauma they cannot be differentiated with absolute assurance even by demonstrating an arteriosclerotic lesion at the site of a recent occlusion of a coronary artery, since injury to the vessel wall can also cause thrombosis. Experimental and clinical instances of recent myocardial contusions or injury to vessels are characterized by hemorrhage between the muscle fibers which crowd them aside. A small subendocardial or epicardial hemorrhage is very often the apex of a large triangular area of myocardial contusion. Frequently, fragmentation of the muscle fibers and tears in the papillary muscles are seen. The changes in areas of old hemorrhage produced by severe chest injury are striking. A wall of polymorphonuclear leukocytes intermingled with granules surrounds the area. Muscle fibers partly lose their transverse striations and are swollen with a granular or lumpy protoplasm. These lesions are usually found deep in the fresh specimen and show up very well after fixation. When there is a difference of opinion concerning pathology it is usually in regard to the old lesions. Observation of such healed lesions in dogs and autopsy specimens from persons who had lived six, but not more than 18 months, showed that the muscle fibers which had undergone anemic necrosis were replaced by a gradual absorption by leukocytes and by connective tissue. This led to thinning of the wall and aneurysmal formation.

Subendocardial hemorrhage was found in 90 per cent of the severe, and 50 per cent of the moderate instances of chest trauma. The next most frequent lesion was small endocardial tears with or without tears of the myocardium. The larger hemorrhages had the appearance of a hematoma; sometimes the underlying myocardium was necrotic. The contraction of the scar tissue occasionally causes incompetence of a valve.

Tears due to trauma may occur in normal or diseased valves and great care must be taken to differentiate them from spontaneous rupture. Frequently the tears of a valve cusp do not heal, leaving fragmentation of the free edge and rupture of the chordae tendineae. Healing occurs when the tear is along the base or commissure. However, complete healing with obliteration of the rupture does not occur although the fragments may grow together or become attached to the heart wall. The
tears are only partial because when the rupture occurs, pressure on the cusps is relieved. The passage of a cardiac catheter through a valve against the blood flow frequently will injure or rupture the cusps while passage with the blood stream or retrieving seems to have no effect.

The mitral valve and its large, broad thick cusps supported by chordae tendineae and papillary muscles rather than the cusps, are most frequently ruptured. When the chordae tendineae hold fast, however, the overbending of the cusps during compression of the heart may produce jagged tears in the cusps. Ruptures and tears of tricuspid and pulmonary valves are rare. A traumatized valve is apparently vulnerable to infection which may produce stenosis by the subsequent scarring, thickening, contraction, and adhesion of the cusps. It would be an extraordinary coincidence in which trauma to the heart occurred at exactly the same time that bacteria were in the blood stream; injury to other parts of the body, however, may afford entrance later.

**Symptomatology and Diagnosis**

More cases of traumatic heart disease are probably overlooked than diagnosed erroneously. The most persistent symptom is precordial pain. The onset may be delayed for six to eight hours. Painful breathing with dyspnea and orthopnea may be observed. Palpitation and tenderness over the precordium are more frequent and may persist for a considerable period of time. The pain of traumatic pericarditis differs from that of myocardial infarction. The latter usually subsides within a short period of time and is accompanied by the clinical picture of coronary occlusion. When a valve is ruptured, the murmur occurs immediately, is loud, constant, and can frequently be heard at a distance from the body. There are also the expected signs of valvular insufficiency. The vibration of a ruptured chordae tendineae produces a sound like a Jew’s harp which disappears when the broken ends become entangled or adhere to the side of the heart cavity.

Symptomless myocardial contusions occur but most frequently the characteristic signs are not observed because the patient is unconscious or attention is distracted by severe injuries to other parts of the body. Close questioning frequently discloses that there are two different types of pain present when myocardial contusion is complicated by fractures of the thoracic bony cage. The chief symptom is pain which is identical in character, location and radiation with that of angina pectoris and coronary artery occlusion. There may be a latent period of from a few hours to a few days before the pain develops. Angina of effort frequently develops with dyspnea, and signs of myocardial weakness with congestive failure. All arrhythmias with their characteristic signs and symptoms have been observed. The occurrence of hemopericardium should be suspected if the heart sounds become distant or if, in the presence of shock, the neck veins are distended. Pericardial tap should be done immediately if hemopericardium is suspected. Roentgenographic examination is important not only of the heart but also of the anterior and posterior mediastinal spaces to determine the presence of hemorrhage. Kymoroentgenography and electrokymography offer not only a means of diagnosis, but also a possibility of locating the lesion and its extent.

It may be difficult or even impossible to differentiate myocardial contusion from myocardial infarction because the former with hemorrhage and fragmentation and the latter with myomalacia produce the same signs and symptoms. Much depends upon a careful history. These considerations also apply to angina pectoris; every effort should be made to ascertain the condition of the heart prior to the injury. There is always the possibility of the coincidental occurrence of coronary artery occlusion but too many cases of chest injury have been followed by this condition to call them all coincidental. It is also well to remember that contusion can occur to a heart already damaged by disease or arterial damage.

Almost every arrhythmia has been produced experimentally by nonpenetrating chest injury. The location of the contusion influences the type of arrhythmia with its characteristic signs, symptoms, and electrocardiographic changes. Myocardial, pericardial, and endo-
Cardiac injury produce changes in the RS-T component and T waves similar to those produced by disease, and may be identical with those of coronary artery occlusion. The unipolar leads are useful in locating the position of the injury.

Cardiac rupture is accompanied by pain and sudden collapse. As the circulation fails, marked pallor, cyanosis, and finally unconsciousness develop. The pulse and heart rate may be slow at first but as tamponade develops the pulse becomes very rapid and weak. The heart sounds are distant and muffled. The arterial pressure falls and the venous pressure rises. There is an increase in the area of cardiac dullness on percussion and of the cardiac silhouette on roentgenographic examination. Pericardial tap and withdrawal of blood is a definite aid in establishing the diagnosis; however, tears of pericardial vessels may also cause hemopericardium. Rupture of the cardiac septum occasionally occurs as the result of necrosis.

**MANAGEMENT AND TREATMENT**

The general program of treatment of myocardial contusion is similar to that of coronary artery occlusion. The period of bed rest should be from two to four weeks. Patients with severe myocardial contusion should be given oxygen continuously for the first four to seven days to alleviate pain, dyspnea, orthopnea and cyanosis. The anginal pain is not affected by any of the coronary vasodilators. The anxiety usually requires a sedative or narcotic. Digitalis is of benefit in auricular fibrillation and congestive failure but does not control the usual tachycardia. Digitalis should not be used routinely because it increases the frequency of ectopic rhythms, suggesting that the irritability of damaged myocardium is increased by digitalis.

The arrhythmias have a tendency to revert spontaneously to normal rhythm, but auricular fibrillation frequently is persistent and requires quinidine therapy. It is advisable to give Pronestyl or quinidine for the first few days for prevention of the more serious cardiac irregularities. When pulmonary edema occurs it should be treated by the usual accepted methods; venesection is not advisable because pulmonary edema is almost invariably the result of pulmonary trauma.

Many patients with serious cardiac injuries arrive at the hospital unconscious and in extremis, but the possibility of survival should not be doubted. Beck has advised repeated pericardial tapping in cases with tears and rupture in order to relieve cardiac tamponade until operation can be performed. If cardiac standstill occurs, the heart should not be massaged because of the danger of increasing the area of contusion and hemorrhage. Although massage is frequently successful, the results are usually only temporary; it is safer to use electrical stimulation or intracardiac injection. Occasionally adhesions, the result of hemorrhage, or a decrease in the anterior-posterior chest diameter from fractures of the bony cage, require surgical correction.

**PROGNOSIS**

Myocardial contusions tend to heal when they are not multiple or extensive and when only one layer of the heart wall is involved. Immediate death does not always occur even from cardiac rupture, especially when located in the septum. Sudden fatal arrhythmias have been produced experimentally in dogs by non-penetrating chest injury in the absence of cardiac pathologic changes at autopsy. This emphasizes the importance of injuries which involve the intrinsic nervous mechanism of the heart. Anderson summarized the possibilities following myocardial contusion as complete recovery, reduced cardiac capacity, pain on effort, and congestive failure or rupture. Slight epicardial injuries heal more readily than those of the endocardium probably because blood does not enter the tear and cause dissection. Extensive pericardial lesions may produce adhesions, obliterate the pericardial sac and result in myocardial insufficiency and failure. As with complete rupture, cardiac tamponade can result from pericardial tears and hemorrhage. The clot formation in the sac may embarrass the heart or the blood may escape into the pleural cavity.

Small endocardial lesions, in spite of the possibility of dissection and mural clot forma-
TRAUMATIC HEART DISEASE

Traumatic heart disease, usually recover completely. The possibility of emboli is always present. Valvular rupture is not associated with sudden death but results in permanent damage. The duration of life is short following rupture of the aortic valve because of the difficulty of the left ventricle to compensate for sudden aortic insufficiency. Rupture of the mitral and other valves causes cardiac hypertrophy and predisposes to congestive failure.

REFERENCES

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