NON-PENETRATING INJURIES OF THE HEART

BY

R. G. ANDERSON, M.D., M.R.C.P.

Honorary Physician, Cheltenham General Hospital

Although it has been recognized since the days of Hippocrates that penetrating wounds of the heart are usually fatal little has been written in the medical literature about the effects of non-penetrating injuries. The reports that have appeared have consisted almost entirely of anatomical descriptions of injuries of the heart found at necropsy. Until about five years ago the possibility that such trauma could produce functional or organic changes in the heart which were not necessarily fatal had been almost completely ignored. Bright and Beck (1935) collected from the literature since 1826 records of 168 cases of non-penetrating heart injury. Of these 156 were fatal, death in all but four being due to rupture of the heart. From these figures it might be assumed that recovery is a rare event, but that this conclusion would be fallacious is suggested by animal experiments carried out by the same authors. In twenty-five dogs the hearts were exposed and traumatized. Only five of the dogs died—one of rupture of the heart, two of ventricular fibrillation at the time of the trauma, and two of myocardial failure, one a few hours afterwards, and the other on the following day. From these experiments it appears that the heart can tolerate a great deal of trauma and that rupture is a rare event. The authors conclude that the vast majority of non-penetrating wounds of the heart are not recognized clinically, the explanation undoubtedly being that the possibility is not considered, especially if, as is usually the case, the patient is in a surgical ward on account of other injuries. Since correct treatment may prevent rupture of the heart during the days or weeks following an injury, and since minor degrees of injury may be followed by prolonged or permanent incapacity, early diagnosis of this condition is not unimportant. Chest injuries are common in civilian practice and will become more common in war-time. It is interesting that during the war of 1914-18, when chest injuries must have been numerous, only one case of non-penetrating injury of the heart was reported (Déjerine and Gascuel, 1914). Cardiac symptoms following a chest injury may be the grounds for a claim for compensation and may wrongly be ascribed to cardiac neurosis. For these reasons it is imperative that in every case in which symptoms of circulatory embarrassment follow an injury to the chest the possibility of cardiac involvement should be carefully considered. The presence of such an involvement may be quite obvious, or it may necessitate a thorough clinical investigation, with radiological and, most important of all, electrocardiographic examinations. The fact that cardiac symptoms do not follow immediately on the injury does not necessarily exclude the possibility of traumatic heart disease. Out of fifty-one cases collected by Warburg (1938) in which injury to the heart was proved by post-mortem examination there was a latent period after the accident in fifteen cases. This latent period varied from two days to as long as eighteen years.

It has usually been considered that the heart, enclosed in its bony cage, is well protected from injury. Yet such is the resilience of the ribs, especially in the young, that there can be sufficient distortion of the thorax to injure the heart without fracture of the ribs or sternum. In the published cases fracture of the ribs is the exception rather than the rule. Moreover, a portion of the heart lies in a very vulnerable position, between the sternum anteriorly and the bodies of the vertebrae posteriorly. Antero-posterior compression of the thorax or a severe blow on the sternum may compress the heart between these bony structures and rupture it.

Morbid Anatomy

Lesions of Pericardium.—In Warburg's (1938) series of sixty cases of subacute and chronic traumatic heart disease which came to necropsy fibrinous pericarditis was found in seven and purulent pericarditis in five. Adherent pericardium was observed in a child who died six months after the trauma. Warburg describes a case of his own which developed Pick's disease with calcifications in the pericardium two years after the injury. Clinically, pericardial friction has often been reported, this sign first being noted at periods varying from a few hours to a month after the injury. It seems not unreasonable to suggest that the milk spots seen so often on the pericardium may result from localized patches of fibrinous pericarditis following mild degrees of trauma. Haemopericardium is found when the heart wall has ruptured. Bright and Beck (1935) reported that a blood-stained pericardial effusion developed in half their experiments in which the exposed hearts of dogs were traumatized.

Lesions of Myocardium.—The injury may lead to immediate rupture of the heart or to contusion of the heart wall—that is, tearing of a few fibres with haemorrhage into the middle of the wall or under the endocardium or pericardium. The rent may involve the endocardium and extend part of the way through the myocardium. Contusion tends to be followed by softening of the area involved, producing a lesion closely resembling a cardiac infarct, and, as in the case of a cardiac infarct, this softened area may give way. The softening appears to reach its maximum during the second week after the injury, for it has been found that if the patient survives the first few hours this is the period during which rupture is most likely to occur (Bright and Beck, 1935). Should the patient survive the first few weeks the area of contusion becomes stronger owing to the development of scar tissue. This scar tissue may stretch, and the formation of a cardiac aneurysm has been described by Vaquez (1928) some months after the trauma and by Joachim and Mays (1927) thirteen years afterwards.

The frequency with which the various parts of the heart wall tend to be ruptured is shown in an analysis

by Bright and Beck (1935) of 152 cases, which gave the following results: left ventricle thirty-seven, right auricle thirty-six, right ventricle thirty-one, left auricle thirty, more than one chamber thirteen, interventricular septum eleven, interauricular septum one. As regards vulnerability there is therefore no striking difference between the four chambers of the heart.

Lesions of Endocardium.—Small subendocardial haemorrhages are often found in experimental animals which survive trauma to the heart. Rupture of valves and tearing of chordae tendineae or papillary muscles did not occur in any of thirty-two dogs whose hearts were exposed and injured by blows (Moritz and Atkins, 1938). Rupture of a healthy valve in a human being appears to be a very rare event. Theoretically it is possible that a small tear or contusion of the valve might heal, and in so doing lead to stenosis of the valve. A well-authenticated case in which mitral stenosis developed in this way is described by Barber (1938).

From the experimental studies of Schlomka (1934) and of Bright and Beck (1935), and from the case reports which have been mainly studied by Bright and Beck (1935) and Warburg (1938), the following clinical picture of non-penetrating heart injury can be built up.

Initial Symptoms of Heart Injury

The blow may be almost immediately fatal as a result of ventricular fibrillation or a large tear in the wall of the heart. In cases which are not rapidly fatal the blow is, as a rule, immediately followed by a transient state of collapse which may be due to the cardiac contusion or to shock or to the emotional effect of the accident. Loss of consciousness is unusual. On recovery the patient complains of symptoms referable to the circulatory system—precordial pain, a sensation of tightness in the chest, dyspnoea, palpitations, or faintness. The precordial pain has no special characteristics and does not as a rule radiate down the arms. It is difficult to differentiate it from the pain caused by injury to the chest wall. It is important to remember that the initial shock may be followed by a symptom-free period during which the patient feels well and can take exercise normally. This latent period varies from a few days to a month, and in cases in which an aneurysm of the heart has developed the patient may remain apparently well for many years and then die as a direct but late result of the injury.

Physical Signs of Heart Injury

Acute traumatic dilatation of the heart occurs experimentally in the majority of cases but has not been definitely established in the human being. An increase in the area of cardiac dullness following injury may result from rupture of a valve or of the interventricular septum or from the development of a pericardial effusion.

The appearance of a thrill in a case in which it was known to be absent before the injury would point to rupture of a valve or septum. Similarly a fresh murmur would suggest one of the above injuries, a laceration of the endocardial lining of one of the chambers, or acute dilatation of the heart.

In cardiac contusion the heart sounds may be weak. Beck (1935) noted that in experimental animals the sounds had a characteristic "tick-tick" quality, and the recognition of this in a man who had received an injury from the steering-wheel of a car enabled him to make the diagnosis during life. With the development of a pericardial effusion the sounds become muffled. Pericardial friction is frequently observed, and such a finding gives definite proof of cardiac injury. Another suggestive finding after a chest injury is persistent tachycardia with a normal temperature. A few days after the injury the

temperature may rise and there may be a slight leucocytosis. Abnormalities of rhythm commonly occur. The occurrence of extrasystoles cannot be used as evidence of cardiac injury since they may appear in many circumstances with a normal heart. Attacks of paroxysmal tachycardia are not in themselves of great diagnostic significance. Auricular flutter or fibrillation may be transient or permanent. They probably indicate some damage to the auricular wall or subendocardial haemorrhage in that situation. Partial or complete heart-block may result from damage to the bundle of His.

Contusion of the heart muscle may be followed by:

(a) *Complete Recovery.*—Recovery is probably more frequent than has been thought in the past, as the lesser degrees of contusion have not been diagnosed.

(b) *Reduced Cardiac Capacity.*—When shortness of breath on exertion first appears after a chest injury or becomes much more incapacitating after the injury than it was before, the possibility of cardiac contusion must be borne in mind.

(c) *Angina of Effort.*—Several cases have been recorded of typical angina of effort starting shortly after chest trauma in patients who were too young to be suffering from coronary artery disease. Where the coronary arteries are already narrowed trauma may precipitate the onset of symptoms.

(d) *Congestive Heart Failure.*—This follows soon after the injury when it is due directly to contusion of the heart muscle. When, however, the injury leads to prolonged paroxysmal tachycardia or auricular fibrillation or flutter there may be an interval of weeks or months before signs of failure make their appearance.

(e) *Rupture of the Heart.*—This complication may occur very rapidly after the injury, but if the tear has not penetrated the whole thickness of the heart wall blood may gradually force its way through the wall, tearing the muscle in front of it so that the final perforation may be delayed for some hours. On the other hand, perforation may not occur until a contused area of heart muscle has had time to soften. This softening is comparable to that which takes place in a cardiac infarct and, as in the case of a cardiac infarct, it is during the second week that perforation is most likely to occur. It is apparent from the clinical course that the leak is often a slow one. The pericardial sac fills with blood, and since the parietal pericardium is a tough and inelastic structure there soon comes a time when any further increase in the effusion compresses the heart and prevents diastolic filling, so that the circulation rapidly fails. The clinical manifestations of this condition are briefly as follows:

There is sudden collapse and frequently complaint of precordial pain. The pulse may be slow at first, but as the compression of the heart increases it becomes more rapid and feeble. As the arterial pressure falls the venous pressure rises, so that the cervical veins become distended. There may or may not be a demonstrable increase in the area of cardiac dullness, but the heart sounds become gradually more distant and muffled. The patient may vomit. He becomes increasingly restless and distressed. As the circulation finally fails pallor ensues and consciousness is lost. In cases of doubt a definite diagnosis may be made by inserting a needle into the pericardial sac.

Electrocardiographic Evidence of Heart Injury

There have been some thirty cases of heart injury in which abnormalities of the electrocardiogram have been described. From these one can draw the following conclusions, which correspond closely with the findings in experimental animals.

The most frequent abnormalities affect the T waves, which may be unusually large or inverted. The RS-T complex may be elevated above the iso-electric line or depressed below it. Typical coronary T waves may occur. Such T waves are also found in myocardial infarction and in cases of pericarditis with or without effusion. They have been described, too, in cases of haemopericardium and of stab wounds of the heart

Electrocardiographic record of case. The first four tracings were taken with the Both portable electrocardiograph.

which have been surgically repaired. The Q waves may be abnormally deep, and various forms of arrhythmia and various grades of heart-block may be shown. The QRS complexes may be slurred or notched. In considering the diagnosis of cardiac trauma from evidence furnished by the electrocardiogram it is necessary that one should be reasonably certain that it was normal before the injury. The occurrence of transient changes in the electrocardiogram after an injury is therefore valuable evidence of cardiac involvement. The fact that the electrocardiogram may closely resemble that seen in cardiac infarction may be due simply to the presence of an area of contused muscle and haemorrhage in the wall of the heart. On the other hand, Schlomka (1934) has suggested that the functional derangements which follow an injury may be due to the fact that the trauma produces not only a muscle lesion directly but also, what is much more important, coronary artery spasm of a degree and duration sufficient to produce actual infarction of the myocardium.

Treatment

The early diagnosis of cardiac contusion is important because correct treatment may prevent the occurrence of perforation. The treatment of cardiac contusion is essentially similar to that of cardiac infarction—that is, absolute rest in bed for six weeks, morphine for the relief of pain, digitalis if signs of congestive heart failure should appear, and quinidine for certain cases of auricular flutter or fibrillation. A pericardial effusion large enough to embarrass the heart's action will require paracentesis.

No case has ever been recorded in which a perforation of the wall of the heart in a human being following a non-penetrating injury has been sutured. This does not, however, appear to be a surgical impossibility. Bright and Beck (1935) collected thirty such cases in which the patient survived the accident for more than an hour. In

each case the opening into the cardiac chamber was small and there was little or no bruising of the myocardium. As death is by no means immediate even after rupture has occurred it appears that prompt surgical intervention might have saved the lives of some of these patients. It is therefore probably advisable, once the diagnosis of myocardial contusion has been made, to have a surgeon always close at hand. After perforation has occurred, and before the operation is begun, the patient might be kept alive by needling the pericardium and allowing blood to escape so as to prevent the development of cardiac tamponade.

Case Report

An amateur steeplechase rider aged 30 was first seen in consultation with Dr. D. P. Gray of Cirencester on April 14, 1939. Three days previously he had been thrown in a hurdle race and stepped on by a horse that was following. One hoof was said to have hit him over the heart and the other in the epigastric region. He was "winded" but did not lose consciousness. On recovering in a few minutes he felt pain all over his body, but was not particularly short of breath. He coughed up a little bright blood on two occasions. Since the accident he had been in bed, where his breathing was fairly comfortable, but he was dyspnoeic on getting out of bed and standing up. He complained of pain in the praecordium, the right scapular region, and the epigastrium, and of a sensation of tightness across the upper part of the sternum on taking a breath. He could feel his heart beating, and since the accident had been able to hear a noise synchronous with the heart-beat. He had had an evening temperature up to 100.4°.

Past History.—At the age of 9 he had had pneumonia and a left-sided empyema, for which rib resection was performed. Nine years before the accident he had been accepted for life assurance, and two years previously, when he had had slight jaundice, his heart had been examined and no comment made. Otherwise he had had no illnesses and was exceptionally fit, riding and playing strenuous games without difficulty. It

may therefore be assumed that his heart was normal before the accident.

Condition on Examination.—The patient was pale but not distressed. There were some abrasions and a slight swelling in the precordial region. The cardiac impulse was weak and there was a faint systolic thrill to be felt over the fourth intercostal space just to the left of the sternum. The apex beat was situated one-quarter of an inch internal to the nipple line in the fifth space. The area of cardiac dullness was not enlarged. There was a very loud blowing systolic murmur audible all over the praecordium and conducted round to the left base. It was maximum in the fourth space just to the left of the sternum. No second sound was audible except in the pulmonary area, where it could just be detected. The pulse was regular, 92 per minute, and of poor value, the blood pressure being 88/74. The lungs were clear. The electrocardiogram showed the RS-T complex elevated above the base line in all three leads—the elevation being maximum in Lead II—and a deep Q wave and inverted T wave in Lead III.

He was kept at complete rest in bed and was given tab. digitalis folia 1/2 grain t.d.s. During the following week he ran a slight irregular temperature and had some cough. On April 21 the signs in the heart were unchanged, but the blood pressure had risen to 114/76. There was an area of impaired percussion note and diminished air entry at the right base and rales were heard at both bases. The liver edge could be felt one inch below the right lower costal margin in the nipple line, but there was no oedema. The electrocardiogram now showed that T_1 was of low voltage and that T_2 was becoming inverted. The dose of digitalis was increased to 1 grain t.d.s. On April 24 radiographs (Dr. S. L. Mucklow) showed that the heart shadow was greatly enlarged in all diameters, the shape of the shadow suggesting enlargement of the heart rather than pericardial effusion. On April 26 the patient had two syncopal attacks. The blood pressure had fallen to 100/64 and pericardial friction was heard over the apex of the heart. On April 28 T_2 was definitely of coronary type. On May 5 pericardial friction could be heard over the whole of the lower half of the praecordium. Although his blood pressure was only 96/64 his general condition had improved, and from that time he continued to make steady progress, although he still had occasional syncopal attacks after meals. On May 12 the pericardial friction was no longer audible. After eight weeks in bed he was allowed up. His blood pressure was still low—97/70—but he was able gradually to increase his exercise. On October 31 radiographs showed that there was only slight enlargement of the left ventricle. On April 3, 1940 (a year after the accident), it was reported that he was taking all ordinary exercise without shortness of breath or discomfort. The heart signs were unchanged, except that no systolic thrill could now be detected. The blood pressure had risen to 115/85 and exercise tolerance was good. The electrocardiogram was approaching normal, although the RS-T complex in Lead II was still slightly elevated, T_1 was large and peaked, and T_3 was still inverted.

Discussion of Case

There seems little doubt that this accident resulted in more than one lesion of the heart. The appearance of a systolic thrill and a loud systolic murmur after the accident might be accounted for by a rupture of the mitral valve or a perforation of the interventricular septum. It is impossible to say with certainty which of these two lesions was actually present. The marked dilatation of the heart which occurred might have resulted from one of these lesions or might correspond to the acute dilatation which has been observed in animals after trauma to the heart. It is interesting that the clinical findings did not lead one to expect the great dilatation which the radiographs revealed.

The finding of electrocardiographic changes resembling those of coronary thrombosis suggested that there was contusion of the myocardium in addition. It is true that coronary changes of T_2 type (in which the elevation of the RS-T segment and later the inversion of T are

maximum in Lead II) are found in association with pericarditis; but pericarditis was probably not the cause of the changes in this case, as they were found as early as the third day after the accident, whereas pericardial friction was not heard until twelve days later.

Summary

Injury to the heart due to non-penetrating blows is commoner than has been supposed and is not necessarily fatal. From a consideration of the cases already published and of animal experiments a brief description of the morbid anatomy, symptoms, signs, and treatment of the condition is given. A new case is recorded.

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INSULIN TREATMENT OF SCHIZOPHRENIA IN WARTIME

BY

JAMES S. MCGREGOR, M.B., Ch.B.

Senior Assistant Medical Officer

AND

R. A. SANDISON, B.Sc., M.R.C.S., L.R.C.P.

House-physician

Warlingham Park Hospital

Recent correspondence in the columns of the *British Medical Journal* has raised two questions in connexion with the continuation of the insulin treatment of schizophrenia in wartime. The first of these throws some doubt on the value of the method as a cure, and the second points out that it requires the use of large amounts of both sugar and insulin. This, it has been said, is not justified in view of the shortage of sugar and the difficulties that may be encountered in importing the raw materials required for the manufacture of insulin.

It is not our intention to discuss the former question in this paper. The last word has yet to be said on this subject, but we do not doubt its value in certain cases of this disease, for we have seen patients respond to insulin treatment who had previously failed to respond either to cardiazol or to electrical convulsion therapy. Furthermore, we have seen cases in which combined insulin and convulsion therapy met with complete success after failure of either alone to influence the course of the psychosis. With the latter question we concerned ourselves at an early date after the introduction of sugar rationing, for it was obvious to us that the allowance of half a pound of sugar a week would in no manner meet the requirements of patients undergoing this form of therapy. With the problem of economy in the amounts of insulin used we did not concern ourselves until a later date.

The technique of insulin treatment adopted at most hospitals is somewhat as follows. The patient is given intramuscular injections of insulin. Starting at 15 to 20 units, the dose is increased daily until it induces hypoglycaemic coma. If there are no contraindications the patient is allowed to remain in coma for one and a half to two hours, after which time the coma is terminated by feeding seven ounces of sucrose in solution into the stomach through a nasal tube. Recovery usually takes