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Recommended Citation

Harry W. Hale Jr. & J. Walter Martin, Non-Penetrating Wounds of the Chest, 7 Clev.-Marshall L. Rev. 439 (1958)

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Non-Penetrating Wounds of the Chest

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SUMMARY: The heart may be seriously injured by compression of the chest by a steering wheel in automobile accidents or in other injuries by non-penetrating blows to the chest. These injuries vary from a simple bruise of the heart to actual laceration of heart muscle and heart valves. The changes may be identified by electrocardiograph and changes in heart rhythm.

Attorneys should make certain that any injury by a blow to the chest is studied by a cardiologist, using not one but a series of electrocardiographs, as even seemingly harmless blows to the chest may result in injuries equivalent to heart attacks.

DURING AND FOLLOWING WORLD WAR II, the surgical literature has contained many reports of the treatment of penetrating or perforating wounds of the heart. Furthermore, dramatic advances have been made in the management of such open wounds of the heart. However, there is a more subtle and less spectacular group of cardiac injuries which are much more common in civilian practice and which are attaining increasing frequency and importance in this day of high-speed automobile accidents. This group of injuries comprises those resulting from blunt trauma to the chest where no open wound is incurred.

Perhaps the commonest type of such injuries is that known as myocardial contusion. It is felt that this injury is not uncommon and often goes unrecognized because the surgeon is distracted by other more obvious injuries and fails to look for the characteristic findings.

Myocardial contusion was first recognized two centuries ago, but very few reports have been made of this injury in the last 150 years. The reports that were made were usually of nonpenetrating wounds that ended fatally. However, in 1942, Leinoff,¹ presented 16 cases which showed the relation between chest trauma and changes in the heart itself.

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¹ Leinoff, H. D.: Acute coronary thrombosis in industry. Direct nonpenetrating injuries, with report of cases. 70 Arch. Int. Med. 33 (1942).

Within the last 20 years, the pathological picture of cardiac contusion has been well documented by Kissane,² Bright and Beck,³ and Osborn.⁴ Beck,⁵ and Moritz and Atkins⁶ were responsible for experimental studies of the effects of blunt trauma to the heart in dogs. Similar studies have been done showing changes in the electrocardiogram relative to these injuries.⁷

Anatomically, the heart hangs freely in the chest cage, suspended from one end and lying between the sternum in front and the vertebral column behind. In young individuals the chest cage exhibits a high degree of elasticity. It is capable of squeezing the heart against the vertebral column without actually suffering any break in its bony structure. This type of injury occurs in the typical "steering-wheel" injury. In addition, the heart can be injured by a sudden acceleration or deceleration which causes the heart to be thrust against the inner surface of the chest wall. The heart muscle may be bruised, or its covering, the pericardium, may be torn. At times, the great vessels which lead to and from the heart may be torn. Violent changes in the intrathoracic pressure, caused by sudden massive abdominal compression, for example, may tear the heart valves.

The cardiac injuries which may be properly classified under the heading of "myocardial contusion" vary considerably in extent and type. The simplest of these is the small bruise beneath the inner or outer lining of the heart and termed respectively, a sub-endocardial or sub-epicardial ecchymosis. In this injury, there is a small localized hemorrhage beneath the lining membrane with disruption of a few muscle fibers. If the injury is more severe a larger area of the heart wall is involved with destruction of more muscle fibers. This destruction may involve the full thickness of the heart muscle over a considerable area. In the latter case, the symptoms, physical findings, and electrocardiographic changes may be identical with a myocardial in-

² Kissane, R. W.: Traumatic heart disease, nonpenetrating injuries. 6 Circulation 421 (1952); Traumatic heart diseases, especially myocardial contusion. 15 Postgrad. Med. 114 (1954).

³ Bright, E. F., and Beck, C. S.: Nonpenetrating wounds of the heart: a clinical and experimental study. 10 Ann. Heart J. 293 (1935).

⁴ Osborn, G. R.: Findings in 262 fatal accidents. 2 Lancet 277 (1943).

⁵ Beck, C. S.: Contusions of the heart. 104 J. A. M. A. 109 (1935).

⁶ Moritz, A. R., and Atkins, J. P.: Cardiac contusion: an experimental and pathologic study. 25 Arch. Path. 445 (1938).

⁷ Kissane, R. W., Fidler, B. A., and Koons, R. A.: Electrocardiographic changes following external chest injury to dogs. 11 Ann. Int. Med. 907 (1937).

farction due to a coronary occlusion. These larger areas undergo softening due to necrosis of muscle and, if the patient lives, heal by replacement with scar tissue, whereas the smaller areas described above heal with little or no detectable scar formation.

Actual thrombosis of a coronary artery may occur due to a blow to the heart, and the changes consequent to this are the same as those following a coronary thrombosis due to any other cause.

Many of these injuries may be accompanied by inflammation of or collections of fluid in the pericardial sac surrounding the heart.

Blunt injuries to the heart may produce changes in the regular rhythm of the heart beat. Changes in heart rhythm, including sudden arrest of cardiac activity, may be present without demonstrable anatomical changes in the heart. This has been shown very well on experimental animals and in autopsy examinations of individuals dying suddenly after trauma to the chest, apparently of sudden cardiac arrest, and in whom no gross anatomical defect is found.

Clinically, the picture of myocardial contusion varies with the extent of the injury. The patient almost invariably complains of left chest pain or pain beneath the sternum which begins immediately or within a few hours after injury. The pain may radiate down the inner aspect of the left arm. There is usually an increase in the heart rate and at times a lowering of the blood pressure. The patient is frequently short of breath. In mild injuries, the signs and symptoms may be absent or transient. In the more severe injuries all the clinical findings of coronary thrombosis may be found, including severe, crushing chest pain radiating to the neck and arm, pallor, apprehension, rapid feeble pulse, low blood pressure, elevation of the temperature, white blood count, blood sedimentation rate, and changes in the electrocardiogram indicative of considerable damage to heart muscle.

Changes in the electrocardiogram may reflect inflammatory disease in the sac covering the heart or areas of focal damage in the heart muscle. Serial electrocardiograms are important, as they show an active inflammatory process in the injured heart which is the natural reaction of these tissues to the injury.

In a period of three years, nine patients were seen in the Edward J. Meyer Memorial Hospital who were thought to have myocardial contusion. Seven of these were injured in automobile accidents and two fell from a height. Five of them suffered the typical "steering-wheel" type of injury.

Five of the patients in this group were under forty years of age and none gave any history of previous heart disease. Four patients had fractures of one or more ribs, but none had other severe intrathoracic injuries such as pneumothorax or hemothorax.

The severity of symptoms varied, but all complained of chest pain except one patient. Four patients showed temporary depression of the blood pressure and seven showed marked increase in the pulse rate, which in one patient persisted for many weeks.

All the patients in this study showed electrocardiographic abnormalities which indicated damage of heart muscle.

In making the diagnosis of myocardial contusion the electrocardiogram is of great importance and in instances where the initial electrocardiographic findings are equivocal and the type of trauma, symptoms or physical findings suggest this diagnosis, the electrocardiogram should be repeated daily for several days in order to detect any change in the pattern which may confirm the diagnosis. The surgeon should seek the assistance of the cardiologist in interpreting questionable electrocardiographic changes, and weigh them along with all the other clinical evidence. Abnormalities of the electrocardiographic tracing which indicate damage of muscle or irregularities of rhythm are suggestive of this injury, particularly if they persist for a time and then improve under treatment. One must be on guard not to misinterpret as due to myocardial contusion those non-specific electrocardiographic aberrations which may be caused by shock, by hemorrhage, or by interference with proper respiration. Those changes, when due to the conditions mentioned, usually promptly revert to normal when the causative disturbance is corrected. Another electrocardiographic abnormality noted in our experience was a rather dramatic change in the pattern, which was interpreted as being characteristic of an abnormally high level of potassium in the blood. This patient had extensive damage to thigh muscles, and it was believed that this damage had caused release of large amounts of potassium which were absorbed into the blood stream. The electrocardiogram returned to normal promptly as might be expected from this condition. and at no time did the patient exhibit any clinical evidence of cardiac abnormality.

The treatment of myocardial contusion is similar to that for coronary occlusion except that anticoagulants are not used because of the danger of aggravating hemorrhage into the heart muscle which is a feature of this injury. In mild cases the patient is kept at rest until symptoms subside and the electrocardiogram returns to normal. In more severe cases complete bed rest is maintained for six weeks and then gradual return to activity as tolerated. Oxygen, sedatives for pain, and digitalis may be needed.

The prognosis in most cases of myocardial contusion is good since most of the injuries are mild and heal promptly with little or no scarring and no disability. At the other extreme, however, occasional patients are killed outright or may die suddenly about ten days after injury when the area of softened muscle in the heart wall ruptures. Occasional patients with severe contusions recover but are left with a considerable area of scarring of the heart muscle, which gives rise to more or less limitation of physical activity.