Nonpenetrating Traumatic Injury of the Heart


The misconception that nonpenetrating trauma to the heart is relatively rare, is primarily due to the fact that myocardial contusion or traumatic pericardial lesions are usually well tolerated and the clinical findings transient and often difficult to recognize. However, the sequelae of this type of cardiac trauma may be serious. Therefore, a careful evaluation of every traumatized individual for cardiovascular injury is essential if the more serious complications are to be recognized and treated effectively. The most commonly encountered cardiac lesion at necropsy was myocardial rupture of a septum or a chamber wall. Now that surgical therapy is available for certain types of cardiac rupture, early diagnosis is essential. Thrombosis of a major coronary artery as a direct result of nonpenetrating trauma was not found in this study and is considered rare. Nevertheless, a previously diseased heart appears to be more vulnerable to trauma. Under these circumstances, it is often difficult to assess properly the extent to which trauma may aggravate a pre-existent disease.

In the present era of high-speed transportation and mechanization of industry, injuries that result in trauma to the heart and great vessels are on the increase. While direct injuries of the penetrating type are usually readily recognized and appropriately handled, the etiologic relation of nonpenetrating and indirect injuries to cardiac trauma is often overlooked, either at the time of injury or when complications are latent. This, despite the fact that there are many excellent reports1-7 on nonpenetrating cardiac injuries, Warburg's contributions8,9 being the most comprehensive. The true incidence of nonpenetrating traumatic injuries to the heart and the morbidity and mortality rates still are not well established. As a consequence, medical and legal opinions in such matters may be arbitrary and conflicting.

It is the purpose of this paper to discuss the causation and to present pathologic data on 546 autopsy cases of nonpenetrating traumatic injury to the heart, and to review our clinical experience with the problem by presenting selected case studies. The 546 cases from the files of the Armed Forces Institute of Pathology represent 0.1 per cent of a total of 207,548 autopsy cases that were available for study. This low incidence is comparable to that reported by others.10,11

CAUSE

The mechanisms whereby nonpenetrating injuries to the heart may be produced have been described.1,2,12,13,14 The forces that cause these injuries may be placed empirically in 7 broad categories: (1) direct, (2) indirect, (3) bidirectional or compressive, (4) decelerative, (5) blast, (6) concussive, and (7) combined. Only a few of these warrant discussion.

Indirect force produces an increase of intravascular hydrostatic pressure such as occurs with sudden compression or crushing of the abdomen and extremities. This mechanism of trauma has been demonstrated experimentally by Beck and Bright.15 The most frequently cited clinical example of cardiac rupture due to indirect forces is the case reported by Kellert.15 A similar case is included in our series.

Blast forces resulting in visceral injury have been well defined.14 Several instances of rupture of a ventricle, interventricular septum, or both, caused by blast trauma were found in our series.

Concussion of the heart, as Beck2 admitted, is an empirical term. Nevertheless, it is descriptive and may be used to indicate forces
TABLE 1.—Lesions Found in 546 Autopsy Cases of Nonpenetrating Trauma to the Heart Tabulated According to the Predominant Lesion and Showing in Parentheses the Number Whose More Significant Associated Cardiac Injury Was Tabulated in Another Category

<table>
<thead>
<tr>
<th>Type and/or Site of Injury</th>
<th>Number of cases combined with</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rupture</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right ventricle</td>
<td>56</td>
<td>66</td>
</tr>
<tr>
<td>Left ventricle</td>
<td>46</td>
<td>59</td>
</tr>
<tr>
<td>Right atrium</td>
<td>35</td>
<td>41</td>
</tr>
<tr>
<td>Left atrium</td>
<td>24</td>
<td>26</td>
</tr>
<tr>
<td>I.V. septum</td>
<td>25(20*)</td>
<td>30</td>
</tr>
<tr>
<td>I.A. septum</td>
<td>18(10*)</td>
<td>25</td>
</tr>
<tr>
<td>Multiple chamber ruptures</td>
<td>69</td>
<td>106</td>
</tr>
<tr>
<td>Contusion/Laceration</td>
<td>105</td>
<td>129</td>
</tr>
<tr>
<td>Pericardial laceration</td>
<td>18</td>
<td>36</td>
</tr>
<tr>
<td>Hemopericardium</td>
<td>13</td>
<td>25</td>
</tr>
<tr>
<td>Valvular laceration/Rupture</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aortic</td>
<td>1(2†)</td>
<td>1</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>0(4†)</td>
<td>0</td>
</tr>
<tr>
<td>Tricuspid</td>
<td>0(8†)</td>
<td>0</td>
</tr>
<tr>
<td>Mitral</td>
<td>0(8†)</td>
<td>0</td>
</tr>
<tr>
<td>Mitral &amp; tricuspid</td>
<td>0(1†)</td>
<td>0</td>
</tr>
<tr>
<td>Coronary artery</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lac/Rupture</td>
<td>0(7†)</td>
<td>1</td>
</tr>
<tr>
<td>Papillary muscle</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lac/Rupture</td>
<td>1(23†)</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>411</td>
<td>546</td>
</tr>
</tbody>
</table>

*Associated with other sites of cardiac rupture.
†Combined with cardiac rupture or other cardiac injury.

that, although of no great magnitude, are sufficient to produce sudden jarring of the heart. Perhaps vagosympathetic responses or direct interference with the neuromuscular mechanisms of the heart result in cardiac standstill or ventricular arrhythmias in the more serious cases. Characteristically, if death is the outcome, significant lesions of the myocardium may not be found. Experimentally this was noted by Schlomka,18 who produced ventricular tachycardia and fibrillation in experimental animals when he traumatized the heart by direct blows, yet could not demonstrate significant anatomic changes in the myocardium at necropsy. Kissane and Koons4 duplicated these results. It was their impression that a region of increased irritability was produced whether or not hemorrhage could be demonstrated. Whatever the mechanism, clinical examples of injury of this type that resulted in sudden death have been reported,17 and there are 2 in our autopsy series. The first individual died a few minutes after he was hit in the chest by a stick; the second was struck in the precordium by a pitched ball, took a few steps forward and fell dead. On postmortem examination, neither showed evidence of cardiac injury other than a few petechiae, primarily in the epicardium, and no other pathologic changes to explain death.

INCIDENCE

The incidence of nonpenetrating traumatic injury to the heart can only be estimated, because the mortality rate is low and the morbidity generally minimal. The incidence reported in autopsy18-20 and clinical series21 has varied from 10 to 75 per cent in the presence of severe trauma, especially chest injury. When the lower percentage is used as an index, the incidence of cardiac injury from automobile accidents alone in the United States is in the range of 150,000 of the population annually. However, the cardiac injury is often overlooked by the clinician, for other bodily injury often masks the manifestations of cardiac trauma which, in most instances, must be searched for immediately after injury if they are to be discovered. Recognition of the early signs of cardiac injury is important, so that proper clinical safeguards can be instituted to minimize complications and to insure prompt and efficient treatment should the more serious sequelae develop.

PATHOLOGY

The principal lesions encountered in the autopsy series of 546 cases of nonpenetrating traumatic heart disease are presented in table 1. In the majority, the injury to extremities or other parts of the body was so severe that the patient would have died in the absence of cardiac lesions.

The results of the important experimental work of Moritz and Atkins,22 Bright and Beck,3 and Kissane and co-workers4,23 have been analyzed in an attempt to correlate them
with our observations on autopsy and clinical material. One of the more significant facts uncovered by these investigators was that direct nonpenetrating cardiac trauma seldom produces serious consequences unless the myocardium is ruptured or fatal arrhythmia develops. Study of the myocardium in these animals, even a few days after production of contusion, revealed little if any pathologic evidence of trauma. Lesions similar to those produced experimentally in animals have been demonstrated by many investigators, including ourselves, to be relatively incidental in fatally injured persons. If they had survived their other injuries, the cardiac lesions probably would have caused little morbidity, and healing would have been as complete as in the experimental animals.

Gross description of the lesions in our series, while ample in most cases, was brief and sketchy in some, leaving uncertainty as to extent and location. In many, records of the exact time of injury and death of the victim allowed computation of the interval during which the morphologic changes took place. Attempts to correlate these changes with the time sequence were not very fruitful because of the variety of causative forces, as well as the effect of other associated injuries, many of them severe. It was possible, however, to select from the material a series of lesions that differed in the extent to which they involved the heart and pericardium. While some of these lesions were single, often more than one was present, including associated cardiac lesions such as pericardial laceration with myocardial hemorrhage. The lesions were classified as follows:

- **Parietal pericardial hemorrhage**
- **Parietal pericardial lacerations**
- **Hemopericardium**
  - **Acute**
  - **Organizing**
- **Pericarditis**
- **Cardiac hemorrhage or contusion**
  - **Subepicardial**
  - **Myocardial**
  - **Subendocardial**
- **Lacerations and contusions**
- **Rupture of the heart**

**Parietal Pericardial Hemorrhage.** Hemorrhage into the parietal layer of the pericardium occurred as an isolated lesion or group of lesions. It was often associated with other traumatic lesions of the adjacent mediastinal structures and heart. Hemorrhagic manifestations varied from small punctate hemorrhages to large hematomas several centimeters in diameter. Grossly the small lesions must be distinguished from the so-called agonal hemorrhages frequently seen on the surface of the heart and pericardium in the absence of a history of trauma. Microscopic examination revealed hemorrhage into the fibrous connective tissue stroma of the pericardium, but the mesothelial lining of the inner surface often remained intact (fig. 1A and B).

**Parietal Pericardial Lacerations (249 Cases).** Lacerations of the parietal pericardium were either superficial or penetrating. The superficial lacerations involved the external or internal surface of the pericardium and sometimes were associated with hemopericardium or other evidence of injury to the heart. In 18 cases pericardial laceration was an isolated lesion (table 1). Lacerations were single or multiple, and varied in length from a few millimeters to 10 cm. or more. In some cases disruption of the pericardial sac was so extensive that the lesion was best described as rupture, the heart often being found in one of the pleural cavities. When descriptions of the site of the lesion were available, it was more often on the left side; but in many cases accurate localization was impossible.

**Hemopericardium.** Hemorrhage into the pericardial sac was frequently observed. It occurred as the only lesion in 13 cases (table 1). In the acute form associated with the fatal lesions, the pericardial sac was distended with blood, which often was partially clotted. The source of hemorrhage could usually, but not always, be demonstrated. The microscopic findings varied with the duration of the hemorrhage. If there had been a period of survival after the injury, both the parietal pericardium and the epicardial surface were coated with a layer of organizing hemorrhage (fig. 1C, D, and E).
PARMLEY, MANION, MATTINGLY

Fig. 1. A. AFIP Acc. 727325. Parietal pericardial hemorrhage. × 35. B. Higher power of part of the same field, showing the intact mesothelial surface and many extravasated red blood cells in the fibrous layer. × 220. C. AFIP Acc. 765838. Organizing hemopericardium. The patient had been struck in the chest by a truck tire he was changing. Organiz-

(Continued on opposite page)
Pericarditis. Occasionally evidence of pericarditis was noted on the surface of the heart at autopsy in the form of loss of luster, with thickening of the epicardium and focal fibrin deposition. Usually hemorrhage was minimal or absent and the pericardial fluid clear, although it sometimes contained flakes of fibrin. Microscopically, the epicardium was thickened with slight fibroblastic proliferation and focal fibrin deposition on the epicardial surface. Leukocytic infiltration, consisting chiefly of lymphocytes and neutrophils, was light and usually focal. When the cardiac injury was associated with lesions of the esophagus or adjacent mediastinal structures, the pericardial cavity sometimes became infected. With purulent pericarditis, the surface of the heart was covered with fibrin and vascularized granulation tissue in which many leukocytes, chiefly neutrophils, were present. In some instances, the leukocytic infiltration was more extensive and involved the superficial layers of the myocardium (fig. 1F). This condition has been designated traumatic myocarditis by some authors.

Cardiac Hemorrhage or Contusion. The cardiac hemorrhage or contusion was recognized at autopsy as a dark red hemorrhagic area in the wall of the heart. The lesion was observed alone or in association with others, especially lacerations or ruptures. In this series, there were 65 cases of cardiac hemorrhage in which the epicardial and endocardial surfaces were intact, and 64 in which hemorrhage was combined with a laceration or lacerations of one surface or the other. The lesion ranged from a few millimeters to 3 or more centimeters in length; in 12 instances the hemorrhage or contusion was on the right side, in 25 on the left, in 15 on both sides, and in 13 the site was not clearly described. The hemorrhage extended through the thickness of the wall of the heart in some instances, but in many others the location and extent of involvement of the wall made it possible to divide the hemorrhages into 3 groups: subepicardial, myocardial, and subendocardial. In some cases the hemorrhage was minimal and grossly appeared to involve only the subepicardial tissue, usually the region of, or adjacent to, the small surface blood vessels (fig. 1G). More extensive hemorrhages, primarily in the myocardium but extending to the subepicardium or endocardium, were grouped as myocardial hemorrhages (fig. 2A and B). Hemorrhages, limited chiefly to the subendocardial tissue, but sometimes involving the deeper layers of the myocardium or the connective tissue stroma of the valve leaflets, or both, were called subendocardial (fig. 2F). Hemorrhage of this type was not observed on gross inspection of the surface of the organ, but only when the heart was opened. Microscopically, the morphologic changes varied with the size, location, and age of the lesion. In lesions of recent origin, they consisted of extravasation of blood, chiefly erythrocytes, into the subepicardial tissue and myocardium, with separation and often disruption of the muscle fibers. In slightly older lesions, the regional blood vessels were dilated and congested, and leukocytes were seen in increased numbers, first in the blood vessels in which they were marginated, then in the hemorrhagic areas and adjacent tissues. With severe hemorrhages, the leukocytic infiltration was prominent and consisted chiefly of neutrophils with a few eosinophils and mononuclear cells. Mast cells were also present in many early but severe hemorrhagic lesions. Pigment-laden macrophages were seen in some

- Arrow indicates inner surface of pericardium. × 115. D. Same case. Coating of organizing hemorrhage on epicardial surface (indicated by arrow). × 15. E. Same case. Focus of fibrin deposition on epicardial surface. × 35. F. AFIP Acc. 331525. Epicardial deposition of fibrin with light infiltration of subepicardial tissue and superficial layers of the myocardium. × 35. G. AFIP Acc. 294749. Cardiac hemorrhage limited to the epicardial surface. × 12. H. AFIP Acc. 278608. Subepicardial hemorrhage with thrombosed surface vessel (arrow) associated with epicarditis, laceration of the heart, and mural thrombus formation due to injuries received in helicopter crash (duration 36 hours). × 8½.
FIG. 2. A. AFIP Acc. 335152. Cardiac hemorrhage or contusion of the myocardium as a result of an automobile accident. $\times 18$. B. Part of same field at higher power. $\times 48$. Extravasated blood with disruption and separation of the myocardial fibrils. Note the abundance of leukocytes in areas of hemorrhage. C. AFIP Acc. 205410. Cardiac hemorrhage with leukocytic infiltration and early necrosis due to contusion of the posterolateral wall of (Continued on opposite page)
cases with survival of a few hours or more, but iron stains were negative. Focal necrosis of the muscle, first evident as acidophilia and loss of striations, was a common feature. The necrotic areas were often limited to a portion of the muscle bundle, a characteristic that distinguishes them from the larger areas of necrosis in myocardial infarction (fig. 2C). Fibrin-like strands and masses, some in blood vessels, others in the interstitial tissue and between the muscle fibers, were noted in some cases, in one with a recorded survival time of only 20 minutes (fig. 2D and E). With longer survival, the necrosis became better differentiated and infiltration more intense. Fibroblastic proliferation was seen in the subepicardial fat and hemorrhagic areas. If the hemorrhage reached or involved the endocardium, even in the absence of laceration, another complication occasionally found was mural thrombus formation (fig. 2G and H). This occurred in both atria and ventricles. In cases of recent origin it appeared as an attached blood clot; in older cases early organization was evident. Definite scarring was not seen in our material.

Lacerations with Contusion. The 64 cases of contusion associated with laceration (but not rupture) were grouped separately. In these cases either of the atria or ventricles might be involved. The lesions were in the right side of the heart in 15 cases, the left side in 24 cases, and both sides in 18 cases. The site was not clearly stated in 7. Lacerations as well as contusions were single or multiple, epicardial, endocardial, or both. Some were superficial, being limited to either the epicardium or endocardium, or deep, involving the myocardium. They ranged in length from a few millimeters to 4 cm. or more. The edges of the lacerations were usually ragged and uneven, although linear lacerations were described. Those of the epicardial surface of the heart involved the surface blood vessels. Hemorrhage along the course of a major branch of a coronary artery was frequently noted. It may be, of course, that ruptures or lacerations of the coronary vessels are overlooked because of associated hemopericardium and gross evidence of laceration. Lacerations of the endocardial surface may occur in either of the atria or ventricles, or both, and involve the papillary muscles, the chordae tendineae, or the valve leaflets or cusps (table 1). Microscopically, the lesions varied according to the extent of the injury and the length of survival. In the immediately fatal lesions or those followed by brief survival, hemorrhage with extravasation of red blood cells that led to disruption and fragmentation of the heart muscle was the chief finding. With lengthening of the survival period to an hour or longer, mural thrombus formation occurred in some instances in which the endocardial surface was lacerated. Leukocytic infiltration, consisting chiefly of neutrophils and a few eosinophils, mast cells, and mononuclear cells, was present in the first few hours. Focal necrosis of muscle was frequent in early lesions, as evidenced by loss of striations and acidophilic staining of the affected fibers. In the older lesions, the leukocytic infiltration was heavier and macrophages were seen in the necrotic areas; sometimes thin strands and masses of fibrin were noted in the smaller blood vessels as well as in the interstitial tissue. Thrombosis of some of the smaller vessels had occurred in an occasional case, but thrombosis of the major coronary arteries or their branches was not encountered in this series (fig. 1H).

Rupture of the Heart. Rupture of the heart occurred in 353 cases of this series (table 1), and is perhaps best studied by gross inspec-

---

D. Same case, another view (× 70) showing an area of myocardial hemorrhage with fibrin strands (automobile accident, survival time 17 hours). E. AFIP Acc. 142447. Fibrin thrombi in vascular spaces of myocardium following blast injuries. × 100. F. AFIP Acc. 66-19313. Subendocardial hemorrhage as a result of an automobile accident. Hemorrhage in this case was extensive and involved the anterior and posterior walls of the left ventricle. G. AFIP Acc. 324481. Contusion of the right ventricle with mural thrombus. × 4¼. H. Same case. Focal necrosis of muscle at base of thrombus (arrow). × 50.
TABLE 2.—Tabulation of Survival Time and Type of Lesion in the 72 out of 546 Autopsy Cases of Nonpenetrating Trauma to the Heart Whose Injury Was Not Immediately Fatal

<table>
<thead>
<tr>
<th>Type and/or Site of Injury</th>
<th>Survival Time</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>30 min.</td>
</tr>
<tr>
<td>Rupture-cardiac</td>
<td></td>
</tr>
<tr>
<td>Right ventricular</td>
<td></td>
</tr>
<tr>
<td>Right atrium</td>
<td>6</td>
</tr>
<tr>
<td>Left atrium</td>
<td>1</td>
</tr>
<tr>
<td>Intervertricular septum</td>
<td>1*</td>
</tr>
<tr>
<td>Interastral septum</td>
<td></td>
</tr>
<tr>
<td>Papillary muscle</td>
<td></td>
</tr>
<tr>
<td>Contusion-laceration-cardiac</td>
<td>1</td>
</tr>
<tr>
<td>Laceration/rupture-pericardium</td>
<td>2(1‡)</td>
</tr>
<tr>
<td>Hemopericardium</td>
<td>1</td>
</tr>
<tr>
<td>Coronary Rupture</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>2§</td>
</tr>
</tbody>
</table>

*Associated with papillary muscle rupture.
†Associated with tricuspid valve rupture.
‡Associated with aortic rupture.
§Associated with cardiac contusion/laceration.

Cardiac contusion and laceration are the commonest lesions of nonpenetrating traumatic heart injury. Cardiac contusion has been reported as occurring in utero.24 Probably because such lesions are so seldom demonstrated at autopsy, traumatic injury has been considered a relatively rare form of heart disease. The current clinical impression regarding injuries of this type has been well expressed in Burchell’s25 couplet:

"And always with a heart contusion
Arise both doubt and much confusion."

In our autopsy material contusion or laceration was the only cardiac lesion in 105 instances (table 1). Thirty-eight of these persons lived for varying lengths of time after injury (table 2). Of the 67 who died as an immediate result of trauma, in only 12 was the primary cause of death considered to be the injury to the heart. Thus, in our series the number whose death was a direct result of cardiac contusion or laceration is relatively small.

Extensive and severe cardiac contusion and laceration from nonpenetrating trauma may occur without external evidence of chest injury;1,6,11 therefore, cardiac injury may not be suspected and the clinical evidence of it difficult to recognize. This is exemplified in the following case.

Case 1 (AFIP Accession 335152). A 20-year-old man, riding a motorcycle, collided with a truck. He was conscious and his blood pressure was 100/70. He had abrasions of the face and lower extremities, but no external evidence of chest injury. Within 30 minutes he became semicomatose, cyanotic, and began to cough. Blood pressure fell to 60/40, and the cardiac rate was 108 per minute. Chest X-ray was considered normal. He was given oxygen therapy and transfused with 500 ml of whole blood. He died suddenly 6 hours and 45 minutes after injury.

At autopsy the pericardium was intact and distended with 380 ml of blood. A laceration, 2 by 1.2 cm. and extending 0.5 cm. into the myoendium of the lateral aspect of the left ventricle at its mid portion, was surrounded by eeehymosis (fig. 3 Left). A branch of the anterior descending coronary artery was included in the laceration and was responsible for the hemopericardium. In addition, a depressed contused area, 2 by 1.2 cm. in a sagittal plane, was found on the posterior aspect of the left ventricle; when the heart was displaced to the right the depression corresponded to the convexity of the vertebral
NONPENETRATING TRAUMA TO THE HEART

Fig. 3 (Case 1, AFIP Acc. 335152). Left. Heart showing area of ecchymosis and contusion of the myocardium in the region of the lacerated branch of the anterior descending left coronary artery. Right. Heart opened, revealing laceration of the posterior papillary muscle of the mitral valve (arrow) and areas of contusion in the anterior and posterior walls of the left ventricular myocardium.

column. On opening the heart, it was noted that the posterior papillary muscle of the mitral valve was lacerated at its base (fig. 3 Right).

Clinically, significant signs or symptoms of cardiac contusion and laceration may be absent or masked by those of injury in other structures. They may also mimic in almost all respects the signs and symptoms of myocardial infarction resulting from coronary artery disease: the character and intensity of the pain, its radiation, and duration. With traumatic injury pain may not be immediate but may follow a latent period of several hours or days\(^5\),\(^{26}\),\(^{27}\) and often be more prolonged. Frequently associated chest injury makes it difficult to distinguish the symptoms of cardiac injury.

Occasionally the syndrome of angina pectoris may follow such an injury\(^6\),\(^8\),\(^{28}\). Usually the syndrome is transitory because of the rapid healing of the cardiac contusion without significant residual damage. If angina pectoris persists, the probability is that coexistent coronary disease, rather than traumatic injury, is responsible.

Electrocardiographic study may clarify such a clinical picture and, in the relatively asymptomatic case of cardiac contusion, may be the only means of arousing suspicion of cardiac injury. Conduction disturbances, arrhythmias, or QRS abnormalities, as immediate results of trauma, are the more important electrocardiographic indications of injury to the heart\(^8\). However, electrocardiograms in the majority of cases of suspected cardiac contusion reveal only transitory nonspecific abnormalities of the S-T segment and T wave. Serial electrocardiograms of a typical case are presented in figure 4. Many\(^7\),\(^{21}\),\(^{27}\),\(^{29}\)–\(^{31}\) have reported similar or more pronounced electrocardiographic abnormalities in clinical cases of traumatic cardiac injury. However, these electrocardiographic findings can hardly be considered justification for an unequivocal diagnosis of myocardial contusion, hemopericardium, or any other specific injury, since they may be produced by a multiplicity of noncardiac causes. This fact has been demonstrated in animals subjected to nonpenetrating cardiac contusion with production of similar electrocardiographic changes, when
FIG. 4. Serial electrocardiograms obtained on a patient who had sustained nonpenetrating chest injury as a result of an automobile accident. No auscultatory findings or roentgen evidence of cardiac or pericardial injury. Note the nonspecific S-T and T-wave abnormalities present on the day of injury, most evident in leads II, aVF, V₅, and V₆. Slight progression of these abnormalities developed during the subsequent week. All abnormalities disappeared by the second week after injury.

only mediastinal and pulmonary hemorrhage were demonstrated at necropsy. Thus, this type of electrocardiographic data should not be considered as a conclusive diagnostic criterion of cardiac trauma. Rather, the importance of these nonspecific abnormalities should be to alert one to the possibility of cardiac injury in the traumatized patient, so that careful evaluation may be carried out.

There are a number of late complications of cardiac contusion. Cardiac aneurysm may develop and calcification of the injured myocardium has been reported. More common is rupture of an extensively contused area of myocardium, hours, days, or even weeks after the injury. The second week is considered the most critical, although rupture may be longer delayed if aneurysm has
formed.11, 33 The high incidence of endocardial lesions with formation of mural thrombi favors thromboembolic complications, the cause of death in 2 patients of our series.

Myocardial failure may be either an early or relatively late sequel to myocardial contusion and is sometimes progressive. Extensive traumatic injury to the myocardium in experimental animals has not always caused myocardial failure.36, 37 As might be expected from these experimental studies, progressive myocardial failure secondary to contusion is relatively rare in man, even though it has been reported.3, 29, 38 It is indicative of severe and extensive myocardial injury. Early and fatal pulmonary edema, believed to be due to left ventricular failure resulting from severe cardiac contusion, developed in only one patient in our series; but, even in this one, pulmonary contusion was a complicating injury. Before pulmonary edema is attributed to myocardial failure, other possible etiologic factors must be considered, particularly associated pulmonary or cerebral injury. It is controversial whether chronic congestive failure can result from cardiac contusion. This complication was noted only once in our series and was believed to be the result of traumatic myocarditis.

Treatment of Cardiac Contusion

The principles of the management of cardiac contusion have been well presented.4, 26, 39 Our concepts differ little from those established. Most patients who have suffered cardiac contusion do not require specific treatment, but only immediate restriction of activity and continuous observation. The prognosis is good. Morbidity is slight and recovery usually complete. Bed rest will suffice until the acute cardiac symptoms subside and the danger of the more serious sequelae has passed. Usually this period of rest and observation need be no more than 2 weeks, but, if extensive contusion is suspected, it must be lengthened to 3 or even 4 weeks because of the possibility of delayed myocardial rupture. The precordial pain, which is an almost constant feature, is usually relieved by the milder analgesics, although narcotics may be required. Kissane and Koons,4 who have had the widest reported clinical experience with cases of this type, are of the opinion that oxygen therapy is almost specific in its ability to relieve the pain and reduce the dyspnea and cyanosis that may be features of the more serious injuries. An accompanying pulmonary injury may account for many of the symptoms in such cases and its response to oxygen is good. Associated thoracic injuries require prompt treatment, for they also produce serious hemodynamic alterations. Should true myocardial failure develop, the prognosis is poor. The accepted therapeutic measures, including digitalization, should be employed. Although thromboembolic complications are a threat to the injured patient, anticoagulant therapy is not indicated because of the danger of hemorrhage in traumatized areas, specifically increasing the threat of serious hemopericardium.

Cardiac Rupture

Cardiac rupture was by far the most common lesion and was encountered in 64 per cent of this autopsy series (table 1). It is also the traumatic cardiac lesion most often reported. In 1935 Bright and Beck3 collected 152 cases of traumatic cardiac rupture from the literature and analyzed the pathologic features. This has been the largest series reported until now. Our series includes data from 353 cases (table 1), only 2 of which, both instances of interventricular septal rupture, have previously been reported.

The 2 ventricular chambers rupture with about equal frequency. Rupture of the atria is less common, but the right atrium ruptures somewhat more frequently than the left. The trauma that produces rupture may, in some instances, seem disproportionate to the extent of cardiac injury. A blow on the chest by a fist40 and other minor trauma41 have been reported to have caused cardiac rupture, and similar observations were made on several cases in our series.

The cause of death in cases of traumatic cardiac rupture is exsanguination or cardiac
tamponade, unless other body injury is of sufficient severity to cause death, as it was in more than 50 per cent of the cases of myocardial rupture in our series. The myocardial rupture, either atrial or ventricular, ranged from an extremely small perforation to virtual cardiac destruction (figs. 5 Left and Right). It was not unusual for external evidence of chest trauma to be minimal or absent.

Survival of an individual who has suffered myocardial rupture is not expected but may occur. Although in our series 13 patients who had atrial rupture survived for a time, only one with ventricular rupture survived even temporarily and in this instance rupture probably was delayed (table 2).

Ventricular Rupture

Traumatic rupture of a ventricle is more common than that of an atrium and rarely permits even temporary survival. One patient in our series lived 4 hours after injury until massive hemorrhage into the thoracic cavity caused death. At autopsy extensive contusion of the right ventricular myocardium with a small rupture suggested that rupture was delayed or that dissection of the necrotic contused area by blood began shortly after injury.

Atrial Rupture

In Bright and Beck's series, 14 of 66 with atrial rupture survived temporarily. Recently Kohn et al. reviewed the literature and collected 80 cases of atrial rupture from various causes, 19 traumatic, in which death was not immediate. In our necropsy series there were 67 cases of atrial rupture, excluding those with multiple ruptures; 8 however, had associated aortic rupture (table 1). Thirteen, or 19 per cent, of this group of patients with atrial rupture survived the initial injury. In none of these was the diagnosis made before
death, and in only 1 was the possibility of atrial rupture entertained. The majority died in less than an hour, the range being 30 minutes to 58 hours. Clinical evaluation was understandably limited, but it was found that 6 of the 13 died as a result of hemopericardium with cardiac tamponade, and the others of hemorrhage or other severe bodily injury. Since the surgical repair of cardiac rupture has been reported, recognition of injury of this type has become important for other than academic reasons, since immediate pericardiocentesis and surgical treatment may be lifesaving.

**Rupture of Cardiac Septa**

Rupture of cardiac septa as a result of non-penetrating trauma deserves special attention because of the relatively high incidence of survival when it is the major cardiac lesion. In our autopsy series 30 instances of rupture of the interventricular septum and 25 of the interatrial septum were found (table 1), but septal rupture was the sole cardiac lesion in only 5 of the former and 8 of the latter. Brief survival was noted in 5 of the 8 cases of isolated interatrial septal rupture and in 3 of the 5 cases of the interventricular type (table 2). We have also studied clinically 2 additional patients with rupture of the interventricular septum caused by nonpenetrating injury.

No particular area of the interatrial septum seemed more susceptible to rupture, although it frequently extended to involve the border of the foramen ovale (fig. 6). We have not seen a patient that survived traumatic rupture of the interatrial septum for more than 2 days, nor are we familiar with any reported cases. The early diagnostic findings were not specific, and death could not be attributed to the creation of the defect.

Rupture of the interventricular septum was most often associated with rupture of the ventricles (table 1). The most common site of rupture was the muscular portion of the septum; second in frequency was rupture involving both the muscular and membranous portions of the septum, and last was rupture of the membranous portion only. Two of the 3 cases of traumatic interventricular septal rupture with temporary survival in our necropsy series have been reported in detail. In 1952, when Pollock et al. reported their cases, they were able to collect 12 of the same type from the literature. Since then at least 7 additional cases with survival have been reported; we have studied clinically, including catheterization of the right side of the heart. One is unique in that surgical repair of the interventricular defect was done by Pierce as an emergency procedure, with use of hypothermia and cardiotomy. Although
repair was not complete, the size of the defect was reduced so that survival of the patient was possible. Lilliehi, however, has successfully repaired such a defect by cardiotomy utilizing a biologic lung oxygenator. Surgical advances, combined with the technics of extracorporeal circulation and asystole, give promise that in the future these defects may be more frequently and thoroughly repaired.

Only severe necrosis and friability of the interventricular septum without an obvious gross defect could be demonstrated in a 3-year-old girl in our autopsy series, although the clinical and pathologic impression was that a left-to-right shunt had developed through this area. Initially a cardiac murmur was not heard, but by the second day after injury a grade IV systolic murmur became audible over the entire precordium and was associated with a systolic thrill. The electrocardiogram (fig. 7 Top) demonstrated right bundle-branch block. Death occurred suddenly on the third day, presumably the result of arrhythmia. This case demonstrated a probable frequent evolution of a traumatic interventricular septal defect from contusion to necrosis and consequent sloughing of the septal area involved.

Fatal arrhythmia or conduction disturbance may result from the creation of a traumatic interventricular septal defect; however, survival of the patient with such a lesion depends primarily upon the size of the defect and the hemodynamic consequences. The pulmonary vascular resistance that develops secondary to these hemodynamic alterations influences the course. A small defect will produce little alteration and the patient would be expected to survive. A large defect resulting in a left-to-right shunt of major magnitude may produce pulmonary edema and death relatively quickly. On the other hand, as in Pollock's case, enough pulmonary vascular resistance may develop to prolong the course with the production of pulmonary hypertension and eventual right ventricular hypertrophy, failure, and death.

The characteristic clinical manifestations of traumatic interventricular septal rupture may be evident immediately after injury, but that they often are delayed suggests that contusion of the septum may be the initial injury, followed by necrosis and finally rupture. The clinical findings are much like those in patients with a defect of the congenital type, but the electrocardiographic abnormalities are usually more pronounced and often demonstrate conduction disturbances, particularly bundle-branch block (fig. 7). Atrioventricular block has also been noted.

Coronary Artery Injury

The relation of nonpenetrating traumatic injury to the production of coronary artery lesions, particularly coronary thrombosis, has been a controversial subject. Warburg and Boas are proponents of the belief that nonpenetrating trauma may produce coronary thrombosis and that this sequence is more frequent than generally appreciated. Others are of the opinion that coronary thrombosis may occur but is rare except in cases of pre-existent coronary disease. We are in agreement with the latter view. In neither our clinical experience nor our autopsy material have we been able to establish the presence of thrombosis, due entirely to nonpenetrating trauma, in a major artery. On the contrary, 2 individuals in our series, one who lived 33 and the other 17 hours after injury, were found to have severe myocardial contusion of the area traversed by the anterior descending coronary artery. Extensive hemorrhage surrounded the artery along most of its course, but coronary thrombosis had not occurred. The demonstration that coronary thrombosis did not result even when there was evidence of severe traumatic injury along the course of the artery favors our impression that thrombosis of a major coronary artery as a result of nonpenetrating trauma is unusual.

On the other hand coronary artery injury in the form of laceration or rupture is not rare. Complete rupture of a major coronary artery was observed in 9 cases and an intimal laceration in a tenth case of our series. In the presence of cardiac maceration, coronary rupture was common, but these cases are not included in this tabulation. Of the 10 individuals, 8 died as an immediate consequence
of the injury: 6 with myocardial rupture, 1 with aortic rupture, and the eighth with contusion of the right ventricle. The 2 others lived for 2 hours and 6 hours, respectively. Both sustained rupture of the anterior descending branch of the left coronary artery at the site of severe contusion. In neither was myocardial infarction demonstrated. Both had marked hemopericardium and cardiac tamponade. These, rather than myocardial infarction, are the important sequelae of laceration of a coronary artery. Case 1 summarized the clinical and pathologic data from one of these patients.

**Valvular Lesions**

The occurrence of valvular lesions as a result of nonpenetrating trauma has been well known for many years. Most reports have been concerned with traumatic rupture of the aortic valve, and until recently surgical treatment was not considered feasible. Less frequently other valvular lesions have been attributed to nonpenetrating trauma, including 4 instances of mitral stenosis and 1 of mitral insufficiency. However, it is questionable that the etiologic relationship of trauma was established in these particular instances. Severe hemorrhage into a valve or adjacent area was found occasionally in our autopsy series and might conceivably have produced similar lesions if the individual had survived associated extensive endocardial laceration or myocardial rupture. The one instance of valvular rupture without associated cardiac injury was in a congenitally bicuspid aortic valve. It is of interest that of the 4 cases of aortic valve rupture in this series, 2 were of congenitally bicuspid valves. The atrioventricular valves were most frequently involved (table 1) but usually myocardial rupture was also present. Probably it is for this reason that traumatic rupture of an aortic valve is more commonly seen clinically. Only one individual in our series survived valvular rupture, and he lived 25 hours after complete separation of the anterior leaflet of the tricuspid valve. This lesion was only one of a number of severe endocardial lacerations in addition to a traumatic interatrial septal defect.

**Injuries of Papillary Muscles or Chordae Tendineae**

Traumatic lesions of the papillary muscles include contusion, laceration, and rupture.
NONPENETRATING TRAUMA TO THE HEART

Fig. 9. Case 2. Electrocardiogram demonstrating right bundle-branch block.

Involvement of chordae tendineae is almost exclusively that of rupture.

Rupture of the papillary muscles was encountered 24 times in our autopsy series and in all but one instance myocardial lesions, frequently myocardial rupture (table 1), were associated. Laceration or contusion of a papillary muscle, as demonstrated in case 1, was somewhat more frequent. No one particular papillary muscle appeared to be predisposed to injury. Three patients with rupture of a papillary muscle survived. Two of these lived 45 minutes to an hour and in both there was an associated traumatic interatrial septal defect. In one a papillary muscle of both the mitral and tricuspid valves had ruptured. The third case is sufficiently unique to warrant a detailed report.

Case 2.* This 28-year-old man was involved in an automobile accident and hospitalized 2 hours after injury. He was in shock. A chest roentgenogram revealed rib fractures and a probable left hemothorax. On left thoracentesis, 1,900 ml. of blood were recovered. Supportive treatment included transfusions of whole blood. Four days after injury a systolic murmur was first heard at the cardiac apex along with a pericardial friction rub. The patient responded to treatment. Four months later his only symptoms were moderate exertional dyspnea and fatigue. Examination at this time revealed a grade II systolic murmur along the left sternal border and a pulsating liver. Venous pressure was 132 mm. of water. The chest roentgenogram (fig. 9) was normal.

The electrocardiogram (fig. 9) demonstrated right bundle-branch block. Right heart catheterization study performed at Brooke Army Hospital revealed no evidence of shunt and normal right ventricular and pulmonary artery pressures.

Ventricularization of the right atrial pressure was the important finding (fig. 10). Tricuspid insufficiency due to traumatic rupture of a papillary muscle or tricuspid valve was considered the most probable diagnosis. Surgical correction was believed to be feasible and was performed by D. A. Cooley, M.D., assisted by T. H. Hewlitt, Lt. Col., MC, using technics of extracorporeal circulation. On cardiomyotomy it was found that the anterior papillary muscle was ruptured and partially regurgitated into the right atrium with each systole. Good repair of the ruptured papillary muscle was accomplished. An incidental finding was rupture of the pericardium. The immediate postoperative course was good, but massive hemorrhage occurred within 12 hours, apparently from the right atrial appendage, and the patient died.

The prognosis of patients with rupture of a papillary muscle has been considered grave, with death expected in a matter of hours or days after injury. This is not always the course. Rupture of a papillary muscle in the left side of the heart with its relatively high pressure would not be as well tolerated as rupture of one of the tricuspid papillary muscles in the low pressure side of the heart.

The clinical features thus depend upon the papillary muscle involved. If in the mitral valve, the findings of sudden severe mitral insufficiency with rapidly developing pulmonary edema may predominate. The signs and symptoms of tricuspid insufficiency that follow rupture of a papillary muscle of that valve may be less dramatic and, as in the re-

*The clinical data on this case were supplied through the courtesy of Weldon Walker, Col., MC, previously Chief of Cardiology, Brooke Army Hospital.
ported case, minimal. The murmurs that may result from rupture of a papillary muscle vary widely and have been reported as systolic, diastolic, or both. In 2 cases of traumatic rupture of papillary muscles, proved at autopsy, no murmurs were heard; neither were any recorded in 2 similar cases of our series, and in a third only a grade II systolic murmur of tricuspid insufficiency was heard.

The clinical findings in cases of rupture of the chordae tendineae depend upon the number ruptured and the valvular insufficiency produced. Significant hemodynamic alterations may be absent, and a murmur may be the only finding. The murmur, as in papillary muscle rupture, may vary in character and be inconstant. In one case it has been described as resembling the sound of a jew’s harp. If significant insufficiency of the tricuspid or mitral valve results, the hemodynamic consequences and the clinical picture would be identical to those of rupture of a papillary muscle.

ARRHYTHMIAS AND CONDUCTION DEFECTS

One of the more common consequences of myocardial injury is the production of arrhythmias, particularly those of supraventricular origin. Sinus tachycardia often accom-
On admission sinus tachycardia with a rate of 110 per minute was noted, blood pressure was 130/90, and there were distant heart sounds and a pericardial friction rub. The features of an x-ray of the chest were compatible with hemopericardium, mediastinal hemorrhage, and pleural effusion (fig. 11). Soon after admission atrial fibrillation (fig. 12 Top) ensued and was treated with Digoxin. During the next 7 days the atrial arrhythmias of fibrillation and flutter (fig. 12 Middle) appeared intermittently with sinus rhythm (fig. 12 Bottom). Twelve days after injury signs of cardiac tamponade due to hemopericardium developed and pericardial drainage was carried out through a partial pericardiectomy. Atrial fibrillation recurred in the immediate postoperative course but was soon converted to a normal sinus rhythm, which was maintained thereafter.

Conduction disturbances and intraventricular and atrioventricular conduction defects also complicate traumatic cardiac injury. The intraventricular conduction defect may be transient or permanent. Right or left bundle-branch block may occur (fig. 7), particularly if the contusion or the rupture involves the interventricular septum. Traumatic myocardial injury may result in varying degrees of A-V block, from prolonged P-R interval to complete block. In some cases this may be a transient phenomenon, in others, permanent. As in other cases of A-V block, a Stokes-Adams syndrome may occur. In our series only one instance of A-V block as the result of trauma was recorded and in this case it was manifested only by a prolonged P-R interval.

**Pericardial Injury**

The pericardium is frequently involved in nonpenetrating traumatic injuries of the heart and pericardial laceration, rupture, or hemopericardium may often be an isolated lesion (table 1).

Although rupture of the heart without associated pericardial rupture has been considered rare in our series it was found in 71 of the 161 cases of isolated atrial or ventricular rupture, but in only 11 in which there were multiple ruptures. Cardiac contusion or laceration frequently occurred without a pericardial lesion. Individuals subjected to apparently the same trauma would, in one instance, show extensive pericardial rupture and, in another, no evidence of injury.

If pericardial laceration or rupture is an isolated lesion, it is usually of no consequence unless complicated by hemorrhage from a lacerated pericardiophrenic artery. Herniation of the heart through the ruptured pericardium usually causes no difficulty but may result in strangulation and sudden death. Herniation without apparent ill effect occurred in 3 patients of this series. Pericardial laceration and rupture undoubtedly are more frequent in traumatized individuals than realized and may be easily overlooked or clinically suggest pericarditis.

A serofibrinous or purulent pericarditis may be a sequel of nonpenetrating trauma. Purulent pericarditis is rare and usually the result of combined lesions, including pericardial laceration and associated lesions of the esophagus or respiratory structures. Pneumopericardium occurring under similar circumstances is usually a benign complication, requiring no specific treatment. Mediastinitis and mediastinal emphysema may complicate the clinical picture.
Fig. 12. Case 3. Serial electrocardiograms recorded during the first week after injury demonstrating: Top, atrial fibrillation, Middle, atrial flutter, and Bottom, normal sinus rhythm. Nonspecific ST-T wave changes compatible with pericardial injury and digitalis effect.
Hemopericardium is common and in a mild form often accompanies epicardial hemorrhage or other minor myocardial injury. In many instances it is the cause of transient changes in the S-T segment and T waves seen in the electrocardiogram. Usually it resolves rapidly. Diagnostic pericardiocentesis is not needed and medical observation to insure against development of increasing hemopericardium and possible cardiac tamponade is the only requirement.

If evidence of cardiac tamponade appears, pericardiocentesis should be performed without delay. If the hemopericardium has progressed rapidly, the possibilities of laceration of one of the coronary vessels, severe myocardial laceration, or atrial rupture must be considered. If pericardiocentesis fails because of clots, or if hemopericardium rapidly recurs, surgical treatment is indicated to locate and control the site of bleeding if possible.

In cases in which hemopericardium shows evidence of progression, but not of such a degree as to produce signs of cardiac tamponade, it is problematical whether immediate pericardiocentesis is indicated. Hemopericardium may be accompanied by a serous pericardial effusion and, if left undisturbed, will often regress. If hemopericardium appears to be moderate in amount and shows no inclination to regress, pericardiocentesis is indicated. It is because of the possibility of the development of constrictive pericarditis that hemopericardium of this degree is best evacuated. In our autopsy series we had no proved instance of constrictive pericarditis nor have we seen this complication in any of our clinical cases. However, in one case of fatal left ventricular rupture incurred in an automobile accident, the pericardium was 1.5 cm. thick, apparently as the result of organization of hemopericardium suffered in an automobile accident 6 months previously.

Hemopericardium with pericardial effusion may develop so insidiously during the first few weeks after trauma that it may go unnoticed at early examinations. For this reason careful cardiovascular evaluation of the traumatized patient should continue for at least 2 weeks after injury. The following case demonstrates the massive proportions hemopericardium may reach following trauma, if repeated evaluations for possible cardiac injury are neglected and attention is focused on the more obvious injuries.

Case 4 (WRAH Reg. No. 85295). This 23-year-old man was a front-seat passenger in an automobile struck by a trailer truck. When admitted to the hospital he was unconscious, with scalp and face lacerations and a fracture of the right clavicle. Blood pressure was 150/76. Babinski reflexes were positive bilaterally, and
spastic paraplegia and paresis of the right arm were present. A chest roentgenogram (fig. 13 Top) revealed the clavicular fracture but no significant abnormality of the cardiac silhouette. The patient remained in coma and his temperature was elevated. Treatment was supportive and included the administration of antibiotics. Chest roentgenogram taken a week after injury was apparently unchanged from the first. Hemograms showed that the hemoglobin had fallen from 13.5 Gm. per 100 ml. 10 days after the accident to 8.5 Gm. 2 weeks later, despite transfusions of whole blood totaling 1,500 ml. given during the interval. The site of bleeding was not found. Three weeks after injury bilateral temporal decompression was carried out because of persisting coma, but hematoma was not demonstrated. A chest roentgenogram taken 1 month after injury revealed marked enlargement of the cardiac silhouette (fig. 13, Bottom) believed to be due to pericardial effusion or hemopericardium. An electrocardiogram (fig. 14) demonstrated S-T and T-wave abnormalities compatible with pericarditis or myocardial injury, and a P-R interval of 0.21 to 0.22 second. Signs of cardiac tamponade or congestive failure were absent. The area of precordial dulness was increased and heart sounds were distant, but there was no pericardial friction rub. Pericardiocentesis was performed and 600 ml. of serosanguineous fluid were removed. The remaining hemoseropericardium regressed without further treatment. Studies for tuberculosis were negative.

**Medicolegal Considerations**

Traumatic heart disease has become an important medicolegal problem as emphasized by the many articles in the literature.72, 73 Aside from strictly medical considerations concerning nonpenetrating traumatic cardiac injury, the legal implications of the injury in our present-day society require evaluation. Excellent criteria72, 73 have been established whereby the relation of trauma to production of cardiac disability may be gauged.

In the over-all management of a patient with either supposed or actual traumatic cardiac injury, the psychologic implications are of primary importance.4 Permanent or serious disability as a result of traumatic cardiac lesions has been demonstrated to be rare and an optimistic approach to the patient’s injury is justified until there is definite evidence to the contrary. Every measure to prevent neurosis should be taken, for many individuals are susceptible to iatrogenic disease whether or not they are conscious of the possibility of a disability claim. Litigation too frequently stems from undue concern on the part of the physician or a thoughtless comment in respect to unproved or minor traumatic injury to the heart.

The factor of pre-existent cardiac disease is one that cannot be evaluated accurately in many cases of traumatic injury, and yet it is often the most important.5, 74 There are reports demonstrating that pre-existent disease was responsible for cardiac disability erroneously attributed to an incidental traumatic injury.75 It has also been shown that a diseased heart is more susceptible to traumatic injury than a normal heart.16, 76 Thus trauma may produce cardiac disability that would not have appeared for some time in the natural course of the disease. The problem of aggravation of pre-existent disease by trauma is most difficult in the medicolegal evaluation of traumatized patients.

No answer to this medicolegal dilemma can be given. However, an appreciation of the natural course of the cardiac lesions that may be produced by trauma and a knowledge of the criteria established for substantiating a relation between injury and cardiac disability will enable the physician to evaluate the majority of cases properly. It must be recognized, however, that the role of trauma in the etiology of the cardiac disability cannot be established with certainty in some cases of pre-existent heart disease.
Summary and Conclusions

1. Cardiac injury caused by nonpenetrating trauma is common, but often the lesion is minimal and the morbidity slight. The mortality rate from injuries of this type is low, and when death occurs, it is most often the result of cardiac rupture.

2. The character of forces responsible for the production of these injuries may be grouped in seven categories: direct, indirect, compressive, decelerative, blast, concussive, and combined forces.

3. Our evaluation of 546 autopsy cases and additional clinical patients with nonpenetrating traumatic cardiac injury has shown that myocardial rupture is the most common finding at autopsy, whereas myocardial contusion and pericardial lesions are the most common injuries encountered clinically.

4. Almost invariably ventricular rupture is immediately fatal, but survival after atrial rupture has been shown to be possible, and since it is amenable to surgical treatment, prompt diagnosis is essential. An individual may also survive rupture of one of the cardiac septa, particularly the interventricular septum. This lesion may now be considered surgically correctible.

5. Although myocardial contusions or traumatic pericardial lesions are often well tolerated, the sequelae may be serious. Therefore, careful clinical evaluation of every traumatized individual for possible cardiac injury is imperative so that the more serious complications may be recognized and treated promptly.

6. Thrombosis of a major coronary artery as a result of nonpenetrating trauma was not observed in this series and is considered rare.

7. The effect of nonpenetrating trauma on a previously diseased heart is often serious. The extent to which trauma aggravates a pre-existent cardiac disease is often hard to assess.

Acknowledgment

The authors wish to acknowledge the assistance of Mrs. Helen Knight Steward in the most helpful editing of this series of articles on traumatic heart disease.

Summario in Interlingua

1. Lesiones cardiac per trauma non-penetrante es commun, sed frequentemente illo es minime e le morbidade es leve. Le mortalitate es basse, e le mortes que occurre es le plus frequentemente le resultato de ruptura cardiac.

2. L fortias responsabile pro tal lesiones pote esser grupparate in septe categorias: Directe, indirecte, compressive, decelerative, explosional, concussive, e combine.

3. Nostre evaluation de 546 necropsias e datos clinic additional ab patientes con non-penetrante lesions cardiac ha monstrate que ruptura myocardial es le constatation le plus commun al necropsia, durante que contusion myocardial e lesions pedicardial es le constatationes le plus commun in le examine clinic.

4. Ruptura ventricular es quasi invariabilemente sequite per morte immediate, sed il ha essite demonstrate que superviventia post ruptura atrial es possibile. Proque ruptura atrial es attaceabile per mesuras chirurgic, un prompte diagnose es indispensabile. Il es etiam possibile superviver a ruptura de un del septos cardiac, specialmente del septo interventricular. Il es permissibile al tempore presente considerar iste ultime typo de lesion como chirurgicamente corrigibile.

5. Ben que contusiones myocardial o traumatic lesiones pericardial se tolera frequentemente satis ben, le sequellas pote esser grave. Ergo, un meticulose evaluation clinic de omne victima de trauma pro le presentia possibile de lesions cardiac es indicate a fin que le plus grave complications pote esser recognoscite e tractate promtemente.

6. Thrombose de un major arteria coronari como resultato de trauma non-penetrante esseva absente in le presente serie e pote esser considerate como rar.

7. Le effecto de trauma non-penetrante in cordes anteriormente morbide es frequentemente molto serie. Le grado a que trauma resulta in un aggravation de pre-existent morbo cardiac es frequentemente difficile a estimar.
REFERENCES


40.  PEACOCK, T. B.: Tr. Path. Soc. London 31: 72, 1880, cited by Kellert.18


48.  PIERCE, C.: To be published.


52.  LILLEHEI, C. W.: Personal communication cited by Cary et al.16


68.  LEVY, M. L.: Non-penetrating wound of the heart: Injury to the pericardium and left...
“Pulseless Disease”—Description by Robert Adams, 1827
An Historical Note

JAMES H. CURRENS, M.D.

In reading some of the literature on Cheyne-Stokes breathing in the Dublin Hospital Reports, I have come upon an account by Robert Adams of an early description of “pulseless disease.” This happens to be in the same report that contains the classical description of Robert Adams of the Adams-Stokes syndrome in 1827. It is probable from the case report that the patient suffered a dissecting aneurysm of the aorta to account for the pulseless state rather than being due to the “Complete Ossification of the Coronary Arteries of the Heart and of the Aortic Valves,” which was Adams’ interpretation of the case. An autopsy was done, but the examination of the aorta was not described.

“A gentleman, aetat. 68, of a pallid countenance, yet full and corpulent, while exerting himself in arranging some books on a high shelf in a library, suddenly felt severe pain in his chest, extending down his right arm, accompanied by a sensation of numbness: his sight became dim, he had vertigo, but did not fall. From that moment his breathing became oppressed, and in a little time he discovered that his pulse, which was unaccountably weak in his left arm, was altogether imperceptible in the right.

“On the following day, the 18th of October, he had still further grounds for alarm; the most careful examination could not detect the least pulsation in any artery in the body; nor was the movement of the heart sensible to the hand laid over the breast; an obscure undulating motion could alone be heard when the ear was for some moments attentively applied to the side of the thorax.

“His breathing was high and laborious, and could only be performed when the body was nearly erect, inclined a little backward or forward. At night he became worse, and enjoyed no sleep; he occasionally turned on his side with a wish to rest, but this posture increased the dyspnoea, and could be preserved only for a few moments.

“Although this gentleman was perfectly aware of the alarming nature of his symptoms, being himself a physician, he was cheerful, and his countenance was but little disturbed. His appetite was not good; but he was able to eat some chicken or fish for his dinner daily. His digestive organs performed their functions but imperfectly; he suffered great distress from flatulence; thus he remained for six weeks, with little alteration in his symptoms, except that his strength was observed declining daily and his breathing becoming more difficult; his rest during the night was still more imperfect; during the entire of this distressing period, no pulse was to be felt in any artery in the body. Although I daily made the most careful examination, it was in vain.”—Robert Adams. Cases of Disease of the Heart, Accompanied with Pathological Observations. The Dublin Hospital Reports and Communications in Medicine and Surgery 4: 443, 1827.

PARMLEY, MANION, MATTINGLY

Nonpenetrating Traumatic Injury of the Heart
LOREN F. PARMLEY, WILLIAM C. MANION and THOMAS W. MATTINGLY

Circulation. 1958;18:371-396
doi: 10.1161/01.CIR.18.3.371
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1958 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/18/3/371

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/