Nonpenetrating Traumatic Injury of the Aorta


Rupture or laceration of the aorta is a more common result of nonpenetrating traumatic injury than is generally appreciated. Approximately 15 per cent of individuals with traumatic rupture survive temporarily. If the lesion is promptly diagnosed appropriate surgical treatment may be life-saving. Diagnosis may be difficult and at times the rupture may remain clinically silent for variable periods. The natural course from aortic rupture to false aneurysm formation with secondary rupture of the aneurysm may be brief or extend over many years. Surgical treatment of a false aneurysm that has remained stable for a prolonged period has been successful, but in some instances conservative management may be the treatment of choice.

Of all the cardiovascular lesions that result from nonpenetrating traumatic injury none demonstrates the need for prompt recognition and surgical therapy as emphatically as rupture of the aorta that is not immediately fatal. That anyone survives complete transection of this major artery is almost unbelievable; nonetheless, an appreciable number have survived.

The medical literature contains many case reports of aortic rupture caused by trauma. According to Sailer, one of the first cases of aortic aneurysm, presumably the result of trauma, was reported in 1557 by Vesalius. Two of the larger series were reported by Kuhn, who collected 75 cases published between 1895 and 1925, and Strassmann, who reviewed the literature in 1947 in connection with his report on 72 cases of his own. Patients who have survived aortic rupture, including those in whom post-traumatic aortic aneurysms developed, have been less frequently reported. The English literature lists at least 57 cases of aortic rupture caused by nonpenetrating trauma which the patients have survived for varying lengths of time. The lesion in all these cases was confirmed by postmortem or surgical examination. Other cases of aortic rupture with survival have been reported, but the diagnoses were made clinically. The 5 cases of traumatic aortic aneurysm recently reported by Steinberg are well documented despite lack of surgical or postmortem confirmation in 4. Although an individual with an apparent traumatic aneurysm of the abdominal aorta has been reported to have survived 27 years after injury, the relationship to trauma in this case may be questioned. However, the 27-year survival of an individual with a traumatic aneurysm of the thoracic aorta was also reported by Steinberg, and 4 others in his series had survived for periods of from 2 to 21 years. One patient with a traumatic aneurysm of the thoracic aorta survived 8 years before surgical repair, but most patients have lived for periods of from 15 minutes to 3 weeks before surgical intervention or death.

It is the purpose of this paper to present the salient clinical and pathologic features observed in our series of 296 cases of aortic injury caused by nonpenetrating trauma and to discuss the diagnosis and management of aortic rupture in those who survive the immediate effects of the injury. Of the 1,174 necropsy cases of traumatic injury of the heart and aorta we have studied from the files of the Armed Forces Institute of Pathology, 275 were cases of aortic rupture, 104 combined with cardiac injury (table 1). Included in the series are 2 living patients in whom traumatic aortic aneurysm has been corrected surgically. In addition to the 275 cases of aortic rupture, 21 cases of laceration...
involving only 1 or 2 layers of the aorta were studied, for a total of 296. In all but 1 of the 21 there was associated cardiac injury. Thirty-eight of the patients with aortic rupture survived the injury for a time.

Injuries other than cardiovascular were frequently sufficient to cause death. In an analysis of the cause of death in this series it was found that approximately 80 per cent of patients with combined cardiac and aortic injury and at least half of those with aortic rupture only would have died from their other injuries had there been no cardiovascular lesion.

The majority of patients were young men, only 4 of the 275 being women. This distribution does not reflect a true sex incidence, but only the sex distribution of the material at the Armed Forces Institute of Pathology, which is drawn from a predominantly male population. The average age in this series was 27 years, 18 and 85 years being the extremes.

The forces that produce aortic rupture are the same as for other nonpenetrating cardiovascular injuries; namely, direct and indirect forces, deceleration, compression, and blast. Often a combination of 2 or more of these forces is responsible.

The mechanism whereby aortic injury or rupture is produced varies with the type of force. A direct force that results in fracture or displacement of one of the dorsolumbar vertebrae may cause rupture of the aorta by shearing action.

Indirect force apparently acts through production of increased intravascular pressure. It has been demonstrated experimentally that such a pressure may exceed 1,000 mm. Hg without causing rupture. An apparently normal human aorta has been ruptured by an aortic pressure of 2,070 mm. Hg. Zehnder tested the tensile strength of strips of aorta and calculated that pressure sufficient to cause rupture is equivalent to an intravascular pressure of 2,500 mm. Hg. Our series includes an instance of rupture of the arch of the aorta by indirect force resulting from a cave-in accident that suddenly covered the body with earth to the level of the midthorax. It is postulated that the volume of blood suddenly trapped in the aorta or forced into the aorta at the time of systole was sufficient to produce the extremely high pressure required to cause rupture.

The most common cause of traumatic aortic rupture is the force generated by rapid deceleration of the body in either the vertical or horizontal plane. Hass has pointed out that deceleration sets up differential forces between the various organs and tissues of the body, depending on their structure, location and attachments, and the direction of deceleration. Regions subjected to the greatest strain are at the points of junction of these differentially decelerated tissues. In the aorta the greatest strains are put upon the isthmus, where the relatively mobile thoracic aorta joins the fixed arch of the aorta, and on the ascending aorta, which joins the arch and the easily displaceable heart. Substantiation for this concept was provided by our series, for the most frequent sites of rupture were at these 2 locations (table 1), the isthmus being the more vulnerable. Attention has also recently been drawn to a “whiplash” mechanism as a factor in producing aortic rupture.

Usually a combination of forces is required to produce rupture. Zehnder's calculations have raised reasonable doubt that the usual deceleration force encountered in

<table>
<thead>
<tr>
<th>Site of rupture</th>
<th>Isolated aortic rupture</th>
<th>Combined with cardiac injury</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ascending aorta</td>
<td>17</td>
<td>47</td>
<td>64</td>
</tr>
<tr>
<td>Arch</td>
<td>16</td>
<td>6</td>
<td>22</td>
</tr>
<tr>
<td>Isthmus</td>
<td>95</td>
<td>29</td>
<td>124</td>
</tr>
<tr>
<td>Thoracic aorta</td>
<td>27</td>
<td>8</td>
<td>35</td>
</tr>
<tr>
<td>Abdominal aorta</td>
<td>11</td>
<td>2</td>
<td>13</td>
</tr>
<tr>
<td>Multiple sites</td>
<td>5</td>
<td>12</td>
<td>17</td>
</tr>
<tr>
<td><strong>Totals</strong></td>
<td><strong>171</strong></td>
<td><strong>104</strong></td>
<td><strong>275</strong></td>
</tr>
</tbody>
</table>
automobile accidents could in itself produce aortic rupture. Table 2 lists the kinds of accidents in which individuals with aortic rupture were involved. The great majority were automobile accidents, where many factors may operate to produce aortic rupture.

**PATHOLOGY**

This study is based for the most part on autopsy material, the lesions of the aorta either being lethal or associated with other injuries that caused death. Most of the aortic lesions were ruptures, varying in length from a millimeter to complete transection, the majority being either complete or so extensive as to be classed as transections, since only a centimeter or so of the circumference of the aortic wall remained intact (fig. 1). No particular site in the circumference of the aorta seemed to be predisposed to rupture. In the majority it appeared as if rupture were spontaneous and simultaneous throughout the vessel wall. Ruptures involving only part of the aortic circumference were often posterior. Although partial ruptures of this type have been cited as the precursor of aneurysm formation, this development was equally frequent in those of our series who had complete transections. Occasionally, ragged, spiral, or longitudinal ruptures occurred, but in the majority the rupture was transverse and

![Image](http://circ.ahajournals.org/issue/onlineonly/611721/571821/571821-fig1.jpg)

*Fig. 1. (AFIP Acc. 571821). A 24-year-old white man killed in an automobile accident. Top, A. The gross specimen of the heart and great vessels reveals a Y-shaped laceration almost completely transecting the aorta 1 cm. above the aortic valve (arrow) and a 3-cm. transverse rupture of the right atrium. Bottom, B. The external appearance of the chest revealed minimal evidence of chest trauma.*
smooth. In a few instances dissecting aneurysm resulted from intimal and medial lacerations with subsequent rupture through the adventitia after dissections of the arterial wall for varying distances. Surprisingly, very few (21) lacerations of the aorta were found in which the trauma involved only 1 or 2 layers of the aortic wall. In these instances they were usually small horizontal or longitudinal tears involving the intima and occasionally extending into the media. Thus in our series the extent of involvement of the aortic wall varied from a simple subintimal hemorrhage to complete laceration of the aorta. The lesions may be classified as follows: (1) intimal hemorrhage; (2) intimal hemorrhage with laceration; (3) medial laceration; (4) complete laceration of the aorta; (5) false aneurysm formation; (6) periaortic hemorrhage.

An evaluation of the gross and histopathologic features of trauma to the aorta requires
an appreciation not only of the normal microscopic structure of the aorta, but also of the possible pathologic alterations that might have preceded the wound. It would be fallacious to assume that all aortas were free from pathologic change at the time of injury. Indeed, our experience with a large series of autopsies in a similar age group in which trauma was not the cause of death indicates that pathologic changes in the aorta, particularly atherosclerosis, are common.

Atherosclerosis was found in our series, but in only 1 instance was it considered to have added significantly to the traumatic lesion. However, medial cystic necrosis of minimal to moderate degree in 3 instances probably made the aorta more vulnerable to unusual stress. Syphilitic involvement of the aorta was noted twice but was not considered a factor in production of the rupture. In neither of these cases was there aneurysmal dilatation of the aorta. Congenital cardiovascular defects were not demonstrated in our series.

**Lesions of the Intima**

The lesions involving the intimal layer in our series may be grouped as (1) intimal hemorrhage and (2) intimal hemorrhage associated with laceration.

**Intimal Hemorrhage**

Areas of intimal hemorrhage were described occasionally in association with fatal lesions. They varied in size and often were noted only on microscopic examination (fig. 2A). It is probable that lesions of this type occur frequently but are not recognized.

**Intimal Laceration with Hemorrhage**

This lesion differs in that the endothelial surface of the aorta is broken and the collagenous and elastic fibers of the “subendothelial layer” of the intima are more severely disrupted and separated by hemorrhage (figs. 2B and 2C). The limiting effect of the internal elastic lamina on the extent of the laceration of the wall is illustrated in both figures. It is lesions of this type that could serve as the nidus of thrombus formation, either bland or septic, with possible peripheral embolization. The break in the endothelial lining might also lead to further dissection.

**Lesions of the Media**

The tunica media forms the bulk of the aortic wall. The elastic fibers of the media with their great tensile strength and their concentric lamellar arrangement provide much of the resiliency of the arterial wall. The smooth muscle fibers provide the contractile properties and serve to maintain the “vascular tone,” but probably contribute relatively little to the over-all strength of the arterial wall. The elastic lamina may be frayed, split, or even destroyed in various diseases, such as advanced atherosclerosis or syphilitic aortitis, and such changes may render it more susceptible to traumatic laceration. The importance of the amorphous intercellular substance of the intima and media has recently been recognized. It is increased in amount in atherosclerosis, and accumulations of this material, often with focal pooling and vacuolization, characterize the lesions of idiopathic cystic medial necrosis of the aorta. Thus, it would appear that an abnormal accumulation of the amorphous intercellular substance must be considered in the evaluation of the effect of trauma on the aorta.

**Traumatic Medial Lacerations**

Lacerations extending into, but not through, the tunica media (fig. 2D) were observed occasionally in this series. Such lesions were described at autopsy, usually in association with other aortic lesions, but sometimes alone. The possibility that a medial laceration might lead to further dissection, rupture, or formation of an aneurysm should not be overlooked.

**Lesions of the Adventitia**

The tunica adventitia provides a portion of the tensile strength necessary for the maintenance of the normal caliber of the aorta, and through the medium of the vasa vasorum contributes to the sustenance of the arterial wall. It is vulnerable to systemic and local disease.
Thus, pathologic processes of the adjacent structures, particularly benign or malignant lesions affecting lymph nodes and involving the adventitia secondarily, should be considered in evaluating any traumatic rupture of the aorta.

The traumatic lesions that involved the adventitia of the aorta were of 3 types: (1) traumatic false aneurysm formation; (2) complete aortic laceration; (3) periaortic hemorrhage.

Traumatic Aneurysms

Included under this type are all cases in which aneurysmal bulging of the aortic wall followed complete laceration of the intima and media, and also those in which false aneurysm formed after rupture of all 3 layers. In some instances a distinction between the 2 could not be made with certainty, even on microscopic examination of the wall of the aneurysm.

In cases in which circumferential laceration of the intima and media was complete, a fusiform aneurysmal bulging of the wall of the aorta developed, while in those in which only a portion of the wall was lacerated the bulging appeared as a localized diverticulum or pouch-like formation. The structure varied with the age or duration of the aneurysm. When a repair operation or postmortem examination was performed soon after injury, the pouch of the aneurysm contained a thrombus consisting of fibrin with many enmeshed red blood cells, and usually there was hemorrhage into adjacent tissue. If the patient survived for a few days, the thrombus was soft and fibroblastic proliferation and early vascularization were observed in the wall of the aorta. It is at this stage, when the thrombus is of recent origin, that peripheral embolization is likely. With longer survival, the thrombus may become organized and the wall of the pouch lined with endothelial cells.

Complete Laceration of the Aorta

Complete rupture of the aorta, including the adventitia and the attached connective tissue, was the most common lesion found. Usually death was immediate. However, false aneurysm formation or occlusion of the site of rupture by thrombus (fig. 3) may permit temporary survival.

Periaortic Hemorrhage

Periaortic hemorrhage often accompanied complete rupture or other traumatic lesions of the aorta, but occasionally occurred independently.

Aortic Rupture with Survival

In our series of 275 cases of aortic rupture caused by nonpenetrating trauma the initial survival rate was 13.8 per cent (table 3). Of the 38 patients who survived for various periods, 2 on whom surgical resection of a post-traumatic aneurysm was performed are
still living. As mentioned previously, serious trauma usually produces combined aortic and cardiac injury and other body injury so extensive that death would be inevitable irrespective of the cardiovascular insult. The difference in survival of patients with and without associated cardiac injury is striking. Of the 171 patients with isolated aortic rupture, 34 or 19.8 per cent survived the initial injury, whereas only 4 or 3.8 per cent of the 104 with combined rupture and cardiac injury survived even temporarily. It is of clinical importance that the site of rupture in the first group usually was the isthmus, a region surgically accessible, while in the second group it was more often the ascending aorta that presented a more formidable surgical problem.

The extent of aortic rupture did not appear to have a significant bearing on survival. Nine of the 38 patients who survived the immediate effects of injury had complete transections of the aorta; 8 of the 9 lived longer than 5 days. Only 3 of the 38 died within 1 hour; thus, most lived long enough so that prompt diagnosis and surgical treatment might have offered a good chance of recovery.

Survival is dependent on the formation of a hematoma that is contained by the mediastinal structures and adjacent tissues. The hematoma so formed may continue to dissect the tissues progressively and rapidly, or dissection may be temporarily halted, only to resume hours or days later. In either event, fatal hemorrhage is the final result. A false aneurysm may develop and survival may be prolonged for months or years. In some instances the false aneurysm grows progressively larger over the years, while in a few it remains stable. No matter what the course, the threat of eventual rupture is always present.

**Clinical Aspects**

The clinical manifestations of traumatic aortic rupture are by no means uniform. The majority of patients die immediately or within a brief period. However, the 10 to 15 per cent that do survive the initial injury usually present one of several general clinical patterns. Those in critical condition may have obvious and severe body trauma, often orthopedic, or may show little clinical evidence of serious injury. Symptoms suggesting a cardiovascular lesion may not appear until hours or days after injury, often with the development of premonitory signs or symptoms of impending hemorrhage, followed by sudden death. Others, usually those who have survived the period after injury without evi-
dence to suggest aortic injury, may exhibit the signs and symptoms of aortic aneurysm with impending rupture weeks or months later; or, in the absence of significant symptoms, aortic aneurysm formation may be revealed by a routine chest roentgenogram. Rarely, aneurysm with dissection may follow traumatic aortic injury of this type. An unusual complication is subacute bacterial endoartitis, which has been reported by Stryker.

Failure to diagnose the aortic rupture while the patient is still alive is the chief obstacle to proper treatment. In 12 of the 38 cases in this series, the correct diagnosis was considered before death, in 9 because an aortic aneurysm was demonstrable by roentgenogram. This diagnosis is seldom considered in the early postinjury period because of the mistaken concept that a patient could rarely, if ever, survive such a lesion. The realization that this concept is false will remove the major impediment to diagnosis.

Unfortunately, there are no clear-cut early diagnostic features of aortic rupture. The patient's vascular injury is usually overlooked and the more obvious injuries are problems for the orthopedic or neurologic surgeon. Eighteen, or 47 per cent of the 38 patients, had a history of unconsciousness for varying periods of time. Twenty-seven had fractures other than of the ribs, usually of an extremity, and many died from unsuspected aortic rupture during or soon after orthopedic treatment. Fractures of the dorso-lumbar vertebrae were found in 34 of the entire series of patients, and in 29 the rupture was definitely related to the site of fracture. Possible aortic rupture should be considered in every patient incurring a dorso-lumbar vertebral fracture in a serious accident.

External evidence of chest injury was helpful in arousing suspicion of possible cardiovascular damage, but was often absent even when cardiovascular damage was extensive (fig. 1B). In a review of all cases of aortic rupture from this standpoint it was found that 101, or 36 per cent of the 275, had minimal or no external evidence of chest injury, despite the high incidence of rib fracture.

Although many patients were in shock initially, blood pressure was within normal range in 10 and became normal in an additional 19 soon after measures to combat shock were instituted. Rarely were there any specific symptoms or signs of cardiovascular injury. Chest or abdominal pain, dyspnea, tachycardia, hemoptysis, and cyanosis were the more common clinical manifestations. These signs and symptoms were most often attributed to other injuries, frequently to associated pulmonary lesions.

Unexplained hemorrhage and hemothorax with roentgenographic evidence of widening of the mediastinum are the early manifestations of diagnostic importance. The early roentgen signs of rupture of the aorta have recently been described. The most important is widening of the mediastinum (fig. 4). Usually there is associated hemothorax that may be present early or may be delayed. Fractures of the thoracic cage are common, despite a lack of external evidence of injury. Clinical and laboratory evidence of unexplained
hemorrhage has in several instances led to exploratory laparotomy on the assumption that continuing blood loss was due to hepatic or splenic laceration. The possibility of aortic rupture must always be considered in such circumstances.

After the first 24 hours have passed and the patient’s condition becomes stable, the probabilities for diagnosis are greatly improved. The signs and symptoms of aortic rupture then resolve into those produced by a mediastinal mass, the hematoma or false aneurysm, and the tendency of this aneurysm to leak blood. This often produces a delayed or recurrent hemothorax, which is the one most important diagnostic feature. Failure to recognize its importance is disastrous, since fatal rupture soon follows. Frequently associated with this finding, although it may occur independently, is the recurrence or aggravation of chest pain caused by further dissection of mediastinal structures and pleura by the enlarging hematoma. The appearance of dysphagia, evidence of tracheal or bronchial obstruction, vena caval obstruction, or the development of hoarseness have been encountered in patients of this series. Impending rupture of the aneurysm into a bronchus may be heralded by hemoptysis with or without associated hemothorax. Roentgenographic examination of the chest at this time may demonstrate that a previously nonspecific widening of the mediastinum now appears as a mass. In one patient of this series, enlargement of the aneurysm was delayed sufficiently long after the accident to suggest a rapidly growing lymphoma or abscess. In other instances, the false aneurysm may remain relatively stable for a prolonged period before evidence of secondary rupture appears. In some cases rupture occurs without prior warning and is catastrophic in extent.

Diagnosis of aortic rupture is easiest in those instances in which a false aneurysm develops and then remains stable and asymptomatic. Routine x-ray examination of the chest is almost always the means whereby diagnosis is made. This was the circumstance in 9 of the patients of this series. The etiology of the aneurysm is suggested when the past history is evaluated in the light of the roentgen finding.

Three cases of the series emphasize the clinical and pathologic features most commonly encountered.

Case 1. AFIP Acc. 128610: This 20-year-old white man sustained a comminuted fracture of the clavicle and an acromioclavicular separation on the right when the truck in which he was riding overturned. There was no other obvious injury and the orthopedic injuries were repaired. Roentgen examination of the chest 1 month later revealed considerable widening of the upper mediastinum, primarily to the right. Repeated roentgen studies demonstrated progressive increase of this widening. Forty-eight days after the injury, evidence of superior vena caval and respiratory obstruction developed with increasingly severe chest pain. Within 24 hours the signs of superior vena caval obstruction were pronounced and the patient was orthopneic with an audible stridor. Within the next 24 hours asphyxia was imminent. Bronchoscopy revealed marked compression of the lower trachea. An emergency thoracotomy was performed, and the aneurysm of the aortic arch that was found ruptured spontaneously during operation. Death occurred 50 days after injury. At necropsy a defect large enough to admit the index finger was noted in the right posterior aortic wall just proximal to the innominate artery. The margins of the defect were smooth and covered with endothelium and communicated with a “false” aneurysmal sac approximately 7 cm. in diameter lying to the right of the superior mediastinum. The sac was filled with partially organized thrombus and was surrounded by considerable fibrosis and edema. The underlying superior vena cava and lower end of the trachea were compressed by the aneurysm. In the right lateral wall of the aneurysmal sac a large tear communicated with the right pleural cavity, which was filled with blood.

Comment. This patient is typical of those in whom orthopedic injuries dominate the clinical picture and vascular injury is not suggested. As in this patient, progressive widening of the mediastinum may be seen in the roentgenograms days after the injury. The rapid development of obstructive symptoms signals the rupture of the aneurysmal sac.

Case 2. AFIP Acc. 639151: This 23-year-old white man was pinned under a wrecked automobile.
On admission to the hospital, he complained of pain in the right side of the chest and the left thigh. Blood pressure was 105/65, pulse 90 per min., and respiration 20 per min. A few rales were heard in the right lung. There were abrasions of the chest, fractures of the sixth and seventh ribs on the left, and the third, fourth, and fifth on the right, and a compound comminuted fracture of the left femur. The patient was given a whole-blood transfusion and the compound fracture was treated surgically. Postoperatively and until 6 days later the patient did well. Then his temperature rose to 101°F., and tachycardia and dyspnea appeared. Because of dulness and absence of breath sounds on the right, thoraecentesis was performed with recovery of 500 ml. of blood. The patient became cyanotic, but oxygen therapy brought relief. Hoarseness developed. During the course of the next 24 hours he was given 1,500 ml. of whole blood and by the end of this period the temperature returned to normal and his breathing was normal. Blood pressure was normal and there were no physical signs of hemothorax. However, on the eighth day after injury he suddenly died. Review of chest x-rays taken during hospitalization revealed no definite abnormalities except hemothorax. At necropsy a complete horizontal transverse rupture of the aorta, 8 mm. below the origin of the left subclavian artery, was surrounded by a large hematoma forming a false aneurysm 6 by 5 by 4 cm. that was contained by the pleura (fig. 5). The recurrent laryngeal nerve was incorporated in this hematoma. A rent 1.5 cm. long in the parietal pleura that contained the aneurysm accounted for the massive hemorrhage, which produced left hemothorax of 4,200 ml.

Comment. This is an example of the patient who has no obvious signs or symptoms of aortic rupture when seen initially. The presence of other injury, usually orthopedic, is the obvious and immediate problem. Hours or days later the sudden development of hemothorax is the sign that there is impending rupture of a heretofore silent false aneurysm. Delayed hemothorax appearing suddenly after injury and for no apparent reason should always suggest the presence of a traumatic false aneurysm of the aorta.

Case 3. AFIP Acc. 709699: This 24-year-old white man was the driver of a car involved in a head-on collision. On admission to the hospital, 2 hours later, he was alert and stated that he had not been unconscious. The blood pressure was 55/40 but rose to 150/70 after administration of 1,000 ml. of dextran. Whole-blood transfusion of 500 ml. was then given. Physical examination revealed abrasions of the face, anterior chest, and lacerations of the extremities. Breath sounds were diminished on the right and the
patient complained intermittently of anterior chest pain. X-ray revealed a comminuted fracture of the femur, right clavicle, and right fourth rib. The chest x-ray also showed widening of the superior mediastinum (fig. 4). Lacerated wounds of the extremities were sutured. During the next 5 hours the blood pressure remained stable although a sinus tachycardia persisted. The electrocardiogram revealed nonspecific T-wave changes. Following sedation with morphine and Demerol he became comatose with sonorous respiration. His condition remained essentially unchanged until he died suddenly 26 hours after injury. At necropsy the significant findings consisted of extensive hemorrhage within the soft tissues of the anterior and posterior mediastinum with hemorrhage surrounding the arch and thoracic aorta. A transverse irregular rupture, 2 cm. proximal to the origin of the innominate artery, encircled all but 4 mm. of the ascending aorta. Bilateral hemothorax of approximately 1,500 ml. was present. The third, fourth, and fifth ribs on the right anterior were fractured, with laceration of the pleura, and there were fractures of the second and third anterior ribs on the left. The lungs were edematous.

Comment. This case is illustrative of the patient with serious injuries, often of the chest, whose condition deteriorates rapidly or progressively. The only signs of aortic injury may be questionable widening of the medi-
NONPENETRATING INJURY OF THE AORTA

1097

astinum roentgenologically and shock or other evidence of hemorrhage.

MANAGEMENT

Once the diagnosis of rupture of the aorta is made, surgical treatment must be considered. The surgical technics employed have been well established.23, 40, 41 Review of the available literature reveals 8 reported cases23, 26-28, 30, 31, 35 of traumatic aortic aneurysm successfully treated by resection. Two additional cases treated by resection and homograft replacement are included in our series. The progression of aortic rupture from a massive hematoma to the formation of a well-defined false aneurysm is well illustrated in these 2 cases.

Case 4. WRAH Reg. No. 3774003: This 27-year-old white soldier incurred a steering wheel injury to the chest and a fracture of the right patella in an automobile accident on December 6, 1953. He was hospitalized immediately and placed in an oxygen tent because of dyspnea. Multiple rib fractures were found on the left side. Transfusions were required because of blood loss due to the development of a left hemothorax, which was considered to be an extrapleural hemorrhage (fig. 6A-D). This was treated by thoracentesis. Following operation for the patellar fracture performed on December 17, hoarseness developed but cleared after 3 weeks. The patient was returned to duty but because of repeated symptoms referable to his knee was rehospitalized in November 1954. On direct questioning the patient admitted to slight exertional dyspnea, a mild nonproductive cough, and a pulling sensation in the chest, which he had ignored. A routine chest roentgenogram revealed a left superior mediastinal mass considered to be an aneurysm of the aorta, presumably traumatic (fig. 6E). Chest x-rays taken before the automobile accident were reviewed and found normal. Physical examination revealed an increased pulsation of the suprasternal notch and widening of the cardiac base on percussion but no other cardiovascular abnormalities. Blood pressures were 130/90 in the right arm, 134/94 in the left arm, and 144/110 in the left leg. Laboratory studies including the serologic tests for syphilis were normal. The electrocardiogram was normal. A venous angiocardiographic study revealed an aneurysm approximately 5 cm. in diameter involving the aorta just below the subclavian artery (fig. 6F). On May 31, 1955, the patient was operated on by one of us (E.J.). The aneurysm arose just distal to the left sub-

clavian artery and was approximately 8 cm. in length and 7 cm. in width. Under endotracheal anesthesia and hypothermia the aneurysm was resected and the defect was repaired with a 6-cm. homologous aortic graft (fig. 7). The patient did well postoperatively and was later discharged from military service in good condition.

Case 5. WRAH Reg. No. 4017011: This 20-year-old white man fell asleep at the wheel of his automobile in October 1955 and the car ran into a tree. He remained unconscious and in shock for 3 hours. Later he complained of headache, pain in the left side of the chest and back, and pain and numbness below the left knee. Epistaxis and vomiting of bloody material occurred. Chest roentgenograms revealed a left
pneumothorax and a small right pneumothorax with atelectasis and fractures of the fourth left and sixth right ribs. X-ray evidence of consolidation of the left lower lobe appeared later and the patient continued to run a low-grade fever. It was believed that an organizing fibrothorax (fig. 8) had developed on the left, and on November 9, 1955, a left thoraecotomy was performed and decortication was accomplished. An aneurysm of the isthmus region was noted, but at that time surgical correction was not considered feasible. The patient was transferred to Walter Reed Army Hospital. Physical examination there revealed blood pressure of 128/90 in the upper extremities and 130/90 in the lower extremity. Expansion of the left hemithorax was reduced and breath sounds in the lower lung field posteriorly were decreased. At a site just medial to the left scapular border a systolic bruit was heard. A similar bruit was heard anteriorly only with the patient sitting and leaning forward in full expiration. Results of laboratory studies were normal. On January 24, 1956, under general oxygen-ether endotracheal anesthesia with hypothermia, the aneurysm was identified just distal to the left subclavian artery. It was 7 cm. in length and 5 to 6 cm. in width. Resection of the aneurysm was completed and a 7-cm. fresh aortic homograft was used to bridge the resulting defect (fig. 9). The postoperative course was uneventful, and the patient was discharged from the hospital in excellent condition.

Ten other patients in this series were operated upon for various reasons. Some were known to have an aortic aneurysm, others underwent thoracotomy because of unexplained and persistent thoracoabdominal

Fig. 8. (WRAH Reg. 4017011) (Case 5). Posteroanterior chest roentgenogram prior to surgery demonstrating the aortic aneurysm of the isthmus region and associated fibrothorax.

Fig. 9. (WRAH Reg. 4017011) (Case 5). Left, A. Photographs obtained at time of thoracotomy demonstrating false aneurysm of the aorta just below the left subclavian artery. Right, B. The homograft in place following resection of the aneurysm.
NONPENETRATING INJURY OF THE AORTA

hemorrhage or an enlarging mediastinal mass of unknown nature. In only 3 instances was operation performed soon after the injury. In the other 7 cases treated surgically the patients had survived from 10 weeks to 4 years, and definite aneurysm formation demonstrable by x-ray made the diagnosis certain. In 1 case that has been reported in detail, the postoperative course was complicated by an esophageal pleural fistula and the aortic homograft ruptured at its proximal anastomosis 3 days postoperatively. One patient, whose case is also reported elsewhere, underwent exploratory thoracotomy only to have the aneurysm declared inoperable. Four other patients died during or after thoracotomy from complications of a prolonged surgical procedure. The seventh patient (case 1) underwent late exploratory thoracotomy with rupture of the aneurysm.

If the patient is in extremis when first seen, even a reasonable certainty of diagnosis would rarely justify exploratory thoracotomy. However, in a traumatized patient who is considered to be in relatively good condition and who demonstrates a widened mediastinum on roentgen study, delayed or recurrent hemothorax, or other evidence of unexplained hemorrhage, the diagnosis of aortic rupture and the need for thoracotomy must be seriously considered.

Those individuals in whom a traumatic aortic aneurysm may be demonstrated to be present late in the period after injury and who are otherwise in good condition should, with present day surgical technics, be considered candidates for surgical correction of the aneurysm. However, it has recently been emphasized that the natural course of traumatic aortic aneurysms may be prolonged and before surgical treatment is decided upon its risks must be carefully evaluated.

SUMMARY AND CONCLUSIONS

A review of 296 cases of nonpenetrating traumatic injury resulting in aortic laceration in 21 and rupture in 275, indicates that this lesion should be suspected more frequently in the traumatized individual.

Study of the causation and the pathology of this aortic injury demonstrates that the commonest site of rupture in this series is at the aortic isthmus, just distal to the left subclavian artery; 45 per cent or 124 occurred at this site.

Almost one fifth of all individuals who sustain traumatic aortic rupture without associated cardiac lesions may be expected to survive at least temporarily. The over-all survival rate in our series was 13.8 per cent (38 of 275 cases) but this figure included those with associated cardiac injury.

A review of the clinical features of the survivors emphasizes that external evidence of trauma may be minimal and initial evidence of definite cardiovascular injury may be lacking. Roentgen signs of a widened mediastinum, evidence of persistent thoraco-abdominal hemorrhage, and the appearance of delayed hemothorax are shown to be early diagnostic criteria of aortic rupture.

Prompt diagnosis of traumatic aortic rupture is essential if surgical treatment is to be performed before fatal sequela develop. Surgical treatment of a traumatic aortic aneurysm that is producing no symptoms and has been demonstrated to have remained unchanged over a period of years should be approached only after careful consideration. Successful surgical resection of a traumatic aortic aneurysm in 2 patients is reported.

ADDENDUM

Since the preparation of this paper 3 additional patients with thoracic aorta aneurysms due to nonpenetrating trauma have been evaluated at Walter Reed Army Hospital. Retrograde aortography was demonstrated to be the most effective roentgenographic means of delineating the extent of these lesions. Two of these patients underwent surgical treatment. In the one the aneurysm could not be resected because it involved the arch of the aorta and its major vessels; the patient died at the time of operation. In the other, whose false aneurysm was a result of almost complete transection of the aorta at the isthmus region, resection with homograft replacement was successful. The operation was performed by means of partial bypass from left atrium to femoral artery with sigmamotor pump.
SUMMARIAL INTERLINGUA

Un revista de 296 casos de non-penetrante traumatisation que resultava in laceration aortie in 21 e in ruptura aortie in 275 casos indica que iste lesion debereaa esser suspicite plus frequentemente in individuos traumatisate.

Le studio del causas e del pathology de iste typo de vulneration del aorta demonstra que in le serie presente le plus commun sito del ruptura es le isthmo aortie, justo distal al arteria sinistro-subclavian. Isto valeva in 124 o 45 pro cento del casos.

On pote expectar que quasi un quinto de omne individuos que sufre un traumatic ruptura aortie sin associate lesions cardias va superviver al minus temporariamente. In nostre serie le superviventia total amontava a 13.8 pro cento (i.e. 38 ex 275 casos), sed iste cifra includева le casos con associate lesions cardiae.

Un revista delas aspectclin del superviventia indica que le evidentia externe de trauma pote esser minimal e que al inizio omne evidentia de un lesion cardiovascular definite pote esser absente. Es monstrate que signos roentgenographic de allargamento mediastinal, manifestationes de persistente hemorrhagia thoraco-abdominal, e le apparition de un retardate hemothorace es precoce criterios diagnostic de ruptura aortie.

5. Le prompte diagnose de un traumatic ruptura del aorta es essential si on vole que le intervention chirurgic pote esser effectuate ante le disveloppamento de sequellas mortal. Le tractamento chirurgic de un traumatic aneurysmo aortie que produce nulle symptomatos e que es demonstramente inalteratey deposit plurum annos es un interprisa que require le plus circumspecte consideracion de omne factores involvite. Es reportate 2 casos de successo in resection chirurgic de traumatic aneurysmos aortie.

REFERENCES

NONPENETRATING INJURY OF THE AORTA


31. DeBakey, M. E.: Personal communication to Storey.39


A new method has been developed to determine the unesterified fatty acids in blood plasma. This technic has been used in these experiments. During a fast arteriovenous differences were observed. These indicated a net transport of fatty acid from fat tissue to heart, voluntary muscle and viscera. Amino acids given during a fast had a similar smaller effect. The suggestion is made that adipose tissue releases more or less unesterified fatty acids into the blood to preserve "caloric homeostasis." A mechanism that is sensitive to the availability of nonfat calories seems to exist, which controls the release of unesterified fatty acids.

Oppenheimer
Nonpenetrating Traumatic Injury of the Aorta
LOREN F. PARMLEY, LT. COLONEL, THOMAS W. MATTINGLY, BRIG. GEN., WILLIAM C. MANION and EDWARD J. JAHNKE, JR., MAJ.

Circulation. 1958;17:1086-1101
doi: 10.1161/01.CIR.17.6.1086
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/17/6/1086

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/