Blunt Trauma Rupture of the Heart

H. W. TRUEBLOOD, M.D.,* R. D. WUERFLEIN, M.D.,** W. W. ANGELL, M.D.***

B RIGHT AND BECK in 1934,³ reported a series of 168 post-mortem cases of cardiac injury. One hundred and fifty-two patients in this series had rupture of one of the cardiac chambers. They pointed out that 30 of this group with chamber rupture lived longer than 1 hour after injury and predicted that with increasing development of cardiac survery, these patients could be salvaged. DesForges,⁴ in 1955, reported the first surgical salvage of a blunt trauma right atrial rupture. Since that report,^{1,2,5,7,8} six additional cases have been reported, documenting the possibility of salvaging those patients with ruptured cardiac chambers (excluding traumatic septal defects). We are adding a report of three cases to the seven previously reported.

Case Reports

Case 1. R. H. A 23-year-old man was brought to the emergency room after injury in an automobile accident on 11-23-70. He was disoriented, hysterical and could not perceive oral commands. There was no obtainable blood pressure, pulse was rapid but regular. The neck veins were distended with the patient elevated 30°. There was cyanosis over the thorax and upper extremities, neck and face. There was a red band-like abrasion across the upper chest. The abdomen was soft with normal bowel sounds. A chest x-ray demonstrated a normal cardiac and aortic silhouette (Fig. 1). There was no pneumothorax, rib fracture, or hemothorax. The admission hematocrit was 38.5%. A central venous line was inserted into the internal jugular vein, revealing a pressure of 25 cm. H₂O. With this finding and the presence of shock, a pericardial tap returned dark red blood which elevated the blood pressure for a short period of time. Blood pressure again deteriorated, however, and he underwent operation 1 hour after admission. The pericardium was opened through a sternal splitting incision. There From The Department of Surgery, Santa Clara Valley Medical Center, San Jose, California 95128

was a large amount of blood in the pericardial sac with heavy bleeding from a tear in the right atrium at its juncture with the inferior vena cava. This area was occluded and the patient was infused with blood through a Levine tube placed in the right atrial appendage which brought the blood pressure up to 100 mm. Hg. He received 8 pints of blood. The atrial tear was repaired and the chest was closed. The hematocrit after 8 units of blood was 37.9%. Postoperative course was stable and he left the hospital 7 days after admission. EKG pattern on the day of injury showed non-specific ST-T changes which were gradually resolved by the time of discharge 7 days later. The cardiac muscle enzymes were transiently elevated in the postoperative period. He was seen in follow-up clinic 1 month later with no problems.

Case 2. T. A. A 26-year-old man was riding his motorcycle on the freeway, and struck the rear of a parked car. He was brought to the emergency room on 5-16-71, with initial vital signs demonstrating a heart rate of 124, respirations of 44, and with no obtainable blood pressure. He had a compound fracture of the left lower leg, and several small scalp lacerations. He was slightly disoriented and moderately cyanotic. The chest was clear on auscultation. Cardiac examination revealed distant heart sounds, and no murmurs. Initial examination revealed a soft abdomen with normal bowel sounds. A CVP line which was inserted in the cephalic vein indicated 24 cm. of water. The chest x-ray later showed this still to be in the neck (Fig. 2). There was no cardiac enlargement on x-ray. Blood pressure rose to 60 mm. Hg with 2 liters of Ringer's lactate. Four hours after admission, blood pressure was still in the 60-80 mm. Hg range. By the time he had received 4 liters of Ringer's lactate, his bowel sounds were absent and the abdomen was somewhat distended. An abdominal tap returned bloody fluid. After immediate transfer to the operating room, he was transfused with several units of blood before he was anesthetized, increasing the pressure to 90 mm. Hg. During abdominal prep he was cyanotic over the chest, neck and face. The abdomen was opened with a midline incision revealing a lacerated liver on the undersurface at the insertion of the falciform ligament which was bleeding freely, but there was not enough blood in the abdomen to account for the degree of shock. At this point 4 units of blood had been transfused and blood pressure completely disappeared. An internal jugular vein catheter was then inserted percutaneously and fed into the right atrium with a pressure reading of 54 cm. of water. With this finding the diagnosis of cardiac tamponade was confirmed. The pericardium was opened

Submitted for publication March 20, 1972.

^{*} Assistant Professor of Surgery, Stanford University School of Medicine. Present Address: 210 South 11th Ave., Yakima, Wash.

^{**} Clinical Assistant Professor of Surgery, Stanford University School of Medicine.

^{•••} Assistant Professor of Surgery, Stanford University School of Medicine.

Vol. 177 • No. 1

by incising the diaphragm in the midline and opening into the pericardium through the abdomen. A great deal of blood was released from the pericardium, and the blood pressure immediately returned to 100 mm. Hg. Due to the fact that the blood continued to flow heavily from the open pericardium, the sternum was split. Upon opening the pericardium, a laceration of the apex of the right atrial appendage was bleeding freely. It was simply clamped and the laceration was oversewn. Following this procedure there was no further bleeding or other evidence of cardiac injury. A mattress suture was placed over Teflon bolsters to control the hemorrhage from the liver laceration. The rest of the intraabdominal examination was negative. Open reduction of the left tibia and fibula fracture was carried out with internal fixation and fasciotomies. The early postoperative course was entirely benign and he subsequently made an uneventful recovery except for slow healing of the left leg.

Case 3. J. S. A 40-year-old man was involved in an automobile accident on 6-11-71, suffering chest and facial injuries. He was admitted to an outlying hospital with a deep laceration of the right side of the face. During evaluation he went into shock. IV's were started and the blood pressure responded. Chest x-rays at that time showed a left pleural effusion and suggestion of a wide mediastinum (Fig. 3). He was immediately transferred to Santa Clara Valley Medical Center with a physician in attendance. The diagnosis was aortic rupture. Blood pressure on admission was 70/50 mm. Hg. A central venous catheter indicated 14 cm. of water. Repeat chest film demonstrated more pleural effusion. Following this he went into shock with blood pressure falling below 60 mm. Hg, and at that time the venous pressure increased to 20 cm. of water. Pericardial tap returned 30 cc. of bloody fluid and a left chest tube was inserted returning 1,300 cc. of blood. Fifty-five minutes after admission he underwent exploratory surgery through a median sternotomy. At the same time preparations were made for cardiopulmonary bypass. There was a 2 cm. tear in the outflow tract of the right ventricle and the heart was protruding into the left chest through a pericardial tear. The pericardial tear was 10 cm. long, parallel to and posterior to the phrenic nerve. The cardiac laceration was repaired with mattress sutures. It was necessary to repair the long pericardial tear to prevent further



FIGURE 2.

incarceration of the heart in the left chest. During the elevation of the heart to repair the tear, he developed arrhythmia. It was deemed necessary to place him on cardiopulmonary bypass after which the pericardial laceration was repaired. A double fracture of the upper sternum was wired. The sternotomy incision was extended into the upper abdomen for exploration. There was a mild stable hematoma of the transverse mesocolon and of the retroperitoneum. The descending thoracic aorta was examined and found free of dissection. His facial laceration was repaired; the fractured zygomatic arch was repaired after he recovered from thoracic surgery. He was noted to have transient cardiac muscle enzyme elevation during the first 4 postoperative days which returned to normal. He subsequently made an uneventful recovery.



FIG. 1. A normal cardiac silhouette.



FIG. 3. Chest X-rays show left pleural effusion and suggestion of a wide mediastinum.

	B/P Adm.	Cyo- nosis	CVP	Chest X-ray	Pericardio- centesis	Time to Surgery	Location of Tear	Type of Thora- cotomy	Additional Injuries
Desforges ⁴ 1955	70/40	Yes		Right Hemo- thorax	No Rt. chest	9 hours	Right Atrium SVC	Right Thora- cotomy	Fractured Left Femur
Bogidain¹ 1966	0/0	Yes		Multiple Rib Fxs. Enlarged Heart	Yes	Not Stated	Left Atrial Appendage	Left Thora- cotomy	RLL Hematoma
Rotman ⁷ 1970	60/40	Yes		Not Done	No	Not Stated ? 1 hr.	Right Ventricle	Not Known	Traumatic VSD 10 days postop. Repaired 2 mos. later
Borja² 1970	60/40	Yes		Medias- tinum Wide Hazy RLL	No	2 hrs.	Rt. Atrial appendage & lateral RA	Left thora- cotomy Extended across sternum	Lacerations of knees
Noon⁵ 1971	0/0	Yes	20	Multiple Rib Fxs.	Yes	Not Stated ? 1 hr.	LAA	Left thora- cotomy Extended Across	Flail left Chest
	Not Known	Not Known		Left Hemo- thorax	No Lt. Chest Tube	2-3 hrs.	LAA	Left Thora- cotomy	Depressed Skull Fracture
Siderys ⁸ 1971	0/0	Yes	35	Medias- tinum Wide	Yes	Not Stated	Rt. Atrial Appendage	Median Sternotomy	None
SCVMC R.H.	0/0	Yes	25	Normal	Yes Bloody	1 Hour	Right Atrium IVC	Median Sternotomy	Facial Lacerations
Т.А.	0/0	Yes	25-54	Normal	Trans. Abd. Bloody	4–5 hours	Rt. Atrial Appendage	Median Sternotomy	Fx. Lt. Lobe of Liver Compound Fx. Lt. Tibia, fibula
J.S.	70/30	Yes	14-20	Normal Heart Lt. Hemo- thorax	Yes Bloody	1 Hr.	Right Ventricle Out Flow	Median Sternotomy	Facial laceration & Fx. Zygoma Hematoma of Mesocolon

TABLE 1

Discussion

Table 1 presents information obtained from the seven previously reported cases and our three cases. All of the published cases concerned young adults who had been involved in automobile accidents. All of these patients entered the emergency room in profound shock and were noted to be cyanotic in the trunk, neck and face. Many had distant heart sounds. Those in whom the central venous pressure (CVP) was measured had marked elevations. The chest x-rays were normal except in those in whom the pericardium was also ruptured. In those patients there was obvious hemothorax. Only one was noted to have an enlarged cardiac silhouette on chest x-ray. It is well known that acute pericardial effusions do not stretch the pericardium, but compress the heart, producing tamponade instead. Pericardiocentesis was diagnostic in six of this series and continued bleeding from the tube thoracotomy led to the diagnosis in three cases.

All but two patients in this series had atrial ruptures,

which is what would be expected from Parnley's⁶ series of post-mortem examinations. He reported a series of 546 cases of traumatic heart injury in which presumably 298 patients had ruptures into the pericardium. Of those in whom survival time was available 13 patients with atrial ruptures survived between 30 minutes and 3 days and only one with ventricular rupture survived longer than 30 minutes. Bright and Beck,3 on the other hand, noted that 30 patients in their series survived longer than one hour, half were due to atrial rupture and half were ventricular ruptures. It is significant that the time between injury and operation was less than 5 hours in all of the reported surviving patients except the one reported by DesForges,⁴ in whom the pericardium was torn, allowing decompression of any tamponade and where the tear apparently temporarily sealed and then rebled.

The right and left atrial appendages were the site of tear in six patients. This is presumably the weakest area and certainly the thinnest portion of the heart. The etiology of the injury in these patients was undoubtedly Vol. 177 • No. 1

a compression force and rupture of the thin area. In three patients there was a tear at the junction of the atrium with the inferior or superior vena cava. In those instances the rapid movement of the heart into the left chest coupled with the compression force, presumably led to tear in the relatively fixed superior and inferior vena caval junction.

The type of thoracotomy incision is of great importance as demonstrated by a fourth case (not reported), in which the right atrial region could not be examined adequately through the left thoracotomy. The tamponade was released but the patient drained an additional two liters of blood postoperatively and may have represented a missed right atrial rupture which spontaneously sealed. If there is an associated hemothorax, then the appropriate chest may be opened. Otherwise a median sternotomy offers the superior exposure of both right and left atrium and is better tolerated by the patient. It is also easily extended into the abdomen for exploration of associated injuries.

A great deal of credit for the early identification of these injuries is due to the routine use of CVP catheters in all patients with severe trauma admitted to emergency room. The combination of shock with high central venous pressure in a patient with blunt chest injury can mean only tamponade or, less likely, a superior vena cava syndrome due to injury and bleeding from the great vessels. One must, of course, be certain of the position of the catheter tip. In our second patient the central venous pressure catheter was noted to be not quite in the superior vena cava and the high reading was therefore missed temporarily.

Cyanosis is not usually believed to be a symptom of tamponade. On the other hand, traumatic asphyxia is noted to be a cyanotic condition limited to the chest, neck and face but is usually associated with multiple petechial hemorrhages in this same distribution. Traumatic asphyxia is, of course, produced by sudden compression of the abdomen and lower chest producing a sudden rise in venous pressure of the chest, neck, face and lungs. A cardiac rupture in these instances may have protected the venous system and the tamponade simply produced a more chronic venous pressure elevation.

Summary

Blunt traumatic injuries of the heart are not uncommon. Chamber rupture into the pericardium, as evidenced by autopsy series, is one of the common injuries in severe blunt chest trauma. There is often a delay of at least 1 to 3 hours between injury and death in these instances. We have reported two cases of right atrial rupture, and one of right ventricular rupture which were surgically corrected, and have reviewed seven previously reported successful cases. With the early use of central venous catheter in the resuscitation of severely traumatized patients and suspicion aroused by finding of cyanosis in the upper one half of the body, this condition will be more readily and earlier diagnosed. Median sternotomy is the superior approach to these lesions.

References

- 1. Bogedain, W., Carpathios, J., Van Sun, D. and Moots, M. F.: Traumatic Rupture of Myocardium. JAMA, 197:154, 1966.
- Borja, A. R. and Lansing, A. M.: Traumatic Rupture of the Heart—A Case Successfully Treated. Am. Surg., 171:438.
- 3. Bright, E. F. and Beck, C. S.: Nonpenetrating Wounds of the Heart—A Clinical and Experimental Study. Am. Heart J., 10:293, 1955.
- DesForges, O., Ridder, W. P. and Lenoci, R. J.: Successful Suture Of Ruptured Myocardium After Nonpenetrating Injury, N. Engl. J. Med., 252:567, 1955.
- Noon, G. P., Boulafendis, D. and Beall, A. C., Jr.: Rupture of The Heart Secondary to Blunt Trauma. J. Trauma, 11:122, 1971.
- Parmley, L. F., Manion, W. C. and Mattingly, T. W.: Nonpenetrating Traumatic Injury of the Heart. Circulation, 18:371, 1958.
- Rotman, M., Peter, R. H., Sealy, W. C. and Morris, J. J., Jr.: Traumatic Ventricular Septal Defect Secondary to Nonpenetrating Chest Trauma. Am. J. Med., 48:127, 1970.
- 8. Siderys, H. and Strange, P. S.: Rupture of the Heart Due to Blunt Trauma. J. Thorac. Cardiovasc. Surg., 62:84, 1971.