Traumatic Tricuspid Insufficiency
Hemodynamic Data and Surgical Treatment

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Isolated rupture of a right ventricular papillary muscle from any cause is rare. In our review of the literature we found only five reported cases.\textsuperscript{1-4} Antemortem diagnosis was established in only two. This report concerns hemodynamic data and operative treatment of a patient with traumatic rupture of the anterior papillary muscle of the tricuspid valve.

Case Report

A 33-year-old Negro soldier in previous good health was severely injured in an auto accident on June 10, 1962. During a head-on collision he was thrown against an unpadded dashboard sustaining a crushing injury to the chest and multiple injuries elsewhere in the body. He was semicomatose on arrival at a nearby military hospital. Pertinent findings on admission were shock, paradoxic motion of the left anterior chest wall, prominent pulsations in the neck, evidence of intra-abdominal injury, and multiple fractures of the right leg. Chest x-rays revealed fractures of the second through seventh ribs anteriorly on the left, and of the eighth, ninth, and tenth ribs posteriorly on the right. There was a pleural effusion on the left, which yielded grossly bloody fluid when aspirated. An exploratory laparotomy was performed, and a tear in the right lobe of the liver was found and packed with Gelfoam. The left chest was then stabilized with traction, a tracheostomy was performed, and the fractured right leg placed in a cast. Rapid improvement subsequently occurred. During the second month of hospitalization progressive ambulation was attempted, but was tolerated poorly because of dyspnea, fatigue, and symptoms referable to the right leg. Because prominent cervical pulsations persisted, intracardiac injury was suspected and an intravenous angiocardiogram was obtained. A precise anatomic diagnosis could not be established, however, and the patient was therefore transferred to Walter Reed General Hospital for further evaluation and treatment.

Physical examination at Walter Reed General Hospital revealed a thin, anxious Negro man who was experiencing mild orthopnea. Pulse 100 per minute; blood pressure 118/88 mm. Hg. Marked pulsation in the neck was evident. The pulsation was synchronous with ventricular systole and so marked that casual inspection suggested severe aortic insufficiency. On more careful inspection the pulsations were more lateral in the neck than seen in aortic lesions and could be differentiated from the normal pulsation in the carotid arteries by palpation. The left border of cardiac dullness was percussion 1 cm. lateral to the midclavicular line. The apex impulse was normal. There was a slight right ventricular lift along the left sternal border. There were no thrills, and pulmonary valve closure could not be felt. A quadruple rhythm was present that was proved by phonocardiography to consist of the first and second heart sounds and an atrial and ventricular gallop. The first heart sound was diminished, and there was a grade I-II/VI systolic murmur heard maximally at the lower left sternal border. The murmur increased with inspiration. On maximum inspiration the liver edge was palpable 2 cm. below the right costal margin in the midclavicular line. Mild, but distinct expansile pulsation was present. There was a positive hepatojugular reflex. Breath sounds were normal, and there were no rales. There was no peripheral edema. The venous pressure was recorded as 130 mm. of saline. The electrocardiogram was within normal limits, but when compared to a tracing taken prior to the accident there was some increase in P-wave voltage suggesting right atrial enlargement. A routine chest x-ray taken 3 months prior to the accident was normal in all respects. X-rays on admission demonstrated considerable increase in the transverse diameter of the heart (fig. 1). In the lateral view there was encroachment on the retrosternal space by an enlarged right ventricle. The venous angiogram (fig. 2) showed tricuspid regurgitation, dilatation of the superior vena cava, reflux into the hepatic veins, and simultaneous opacification of the aorta and pulmonary artery. The latter finding suggested the presence of a right-to-left shunt.

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Right heart catheterization was performed and biplane angiograms were obtained at Walter Reed General Hospital on September 19, 1962. The right atrial pressure pulse was characteristic of tricuspid insufficiency (fig. 3). The "x" descent was interrupted by a tall v wave, which reached a peak equal to 19.5 mm. Hg. Mean right atrial pressure was 13 mm. Hg; right ventricular systolic pressure 20 mm. Hg; end-diastolic pressure 5 mm. Hg; pulmonary artery pressure 20/13 mm. Hg. Indocyanine green dye curves were suggestive of a small right-to-left shunt at the atrial level. Hypaque 90, 1.2 ml. per Kg., was injected under a pressure of 10 Kg. per cm.$^2$ through a no.-7 N.I.H. catheter with its tip positioned near the apex of the right ventricle. Biplane films (fig. 4) demonstrated prompt reflux of dye into the right atrium, superior and inferior vena cavae, and the hepatic veins. Our angiographic study did not demonstrate a right-to-left shunt at the atrial level.

On the basis of all available information the patient was considered to have traumatic tricuspid insufficiency, enlargement of the right ventricle and right atrium, a right-to-left shunt, and congestive heart failure with a functional and therapeutic class of III C. The shunt was assumed to be through a patent foramen ovale, secondary to elevated right atrial pressure, although a tear in the interatrial septum could not be excluded. Because of his poor response to treatment he underwent open-heart surgery on September 26, 1962.

**Operation Report**

Under fluothane endotracheal anesthesia, the patient’s chest was entered through a median sternotomy incision. Dense adhesions were encountered between the lung, chest wall, pericardium, and heart. These were divided by sharp dissection, and a large longitudinal laceration in the pericardium was observed. Palpation of the right atrium and right ventricle through the enlarged right atrial appendage revealed a marked degree of regurgitation through a totally incompetent tricuspid valve. With each ventricular systole a ruptured anterior papillary muscle would herniate into the right atrium and strike the operator’s palpating finger. Further exploration revealed a dilated and patent foramen ovale.

Pre-bypass pressures were taken in the right atrium. The a wave, which measured 9 mm. Hg, blended immediately into a tall v wave, measuring 18 mm. Hg.

Cannulation was then carried out and extracorporeal circulation maintained with a Mayo-Gibbon pump oxygenator. The right atrium was extensively opened and all blood aspirated from the right atrium and right ventricle. Intracardiac inspection revealed the widely patent foramen ovale. The anterior cusp of the tricuspid valve was intact but was attached by its chordae tendineae to a section of the anterior papillary muscle, which had been avulsed from the right ventricle (fig. 5a). The tumbling action of the blood on this free section of papillary muscle had completely knotted the attached chordae. Further inspection revealed that sections of both the septal and posterior leaflets of the tricuspid valve...
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Figure 2
Venous angiocardiogram.

had been torn from their attachment to the tricuspid annulus.

Repair of all the observed defects was begun by suture closure of the patent foramen ovale (fig. 5b) followed by a careful attempt to untangle the chordae of the anterior leaflet. Two mattress sutures were placed through the avulsed segment of anterior papillary muscle. This structure was then reattached in its normal position in the right ventricle, and the sutures were tied outside the heart over a small piece of Teflon felt (fig. 5c). Finally, the avulsed segments of the septal and posterior leaflets of the tricuspid valve were attached to the tricuspid annulus with sutures (fig. 5d). The right side of the heart was filled with blood, the atriotomy incision closed, and cardiopulmonary bypass discontinued.

When the patient was stable, repeat pressure readings were taken in the right atrium. The pressure was now 9/4 mm. Hg with an a wave of 6 mm. Hg and a cv wave complex of 9 mm. Hg. This demonstrated mild residual tricuspid regurgitation but marked improvement over the preoperative condition. The total period of cardiopulmonary bypass was 60 minutes. The patient's immediate postoperative course was uneventful.

On December 3, 1962, right heart catheterization was repeated in the diagnostic laboratory, in order to obtain data uninfluenced by anesthesia and at a time further removed from the operative procedure. The findings which had been obtained immediately after repair were confirmed. The right atrial pressure tracing is shown in the lower portion of figure 3. Although regurgitation persisted, considerable improvement had been effected, the height of the v wave being reduced to about 50 per cent of the preoperative level.

At the time of our last evaluation of this patient, 9 months following surgery, he showed definite improvement. Visible pulsations in his neck veins persisted but they were much less prominent than preoperatively. He became somewhat short of breath on climbing more than one flight of stairs at a moderate pace, but his orthopedic problems made walking more difficult for him. From the clinical standpoint he had improved to a class II C.

Discussion
Mechanical trauma can produce a variety of lesions in the heart.4-11 Traumatic rupture of a papillary muscle occurs in less than 5

Figure 3
Atrial pressure pulses.

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per cent of autopsied cases, and when it occurs it is usually combined with cardiac rupture or other cardiac injury. Three instances of isolated traumatic rupture of the left ventricular papillary muscle have been reported. Isolated traumatic rupture of a right ventricular papillary muscle has been reported in only two cases. In 1920, Kleberger reported the case of a young soldier who had been killed by a grenade explosion. At autopsy there were a fractured sternum and rupture of the anterior papillary muscle of the tricuspid valve. The only reported clinical diagnosis of isolated traumatic rupture on the right side of the heart was made by Walker in 1956. The patient was operated upon by Cooley and the clinical and hemodynamic data were reported by Parmley et al.

Papillary muscle rupture has resulted from a variety of causes other than mechanical trauma. Myocardial infarction is the leading cause, but bacterial endocarditis, syphilis, myocardial abscess, and periarteritis nodosa have been implicated. Usually a left ventricular papillary muscle was involved. Only three cases of rupture of the right ventricular papillary muscle were reported in these categories and all three were due to endocarditis.

The clinical sequelae of a ruptured left ventricular papillary muscle are well known and have been outlined by Davison and Askey. Davison was the first to report an antemortem diagnosis. More recently Breneman and Drake have discussed the differential diagnosis. Following the onset of sudden severe mitral insufficiency, rapidly developing pulmonary edema often occurs. Intractable heart failure progressing to death in hours or days is the rule. Occasional longer survivals are recorded.

The clinical course following rupture of the right ventricular papillary muscle is less well known. The only description is that by Parmley et al. in discussing Walker’s case. A 28-year-old soldier suffered multiple rib fractures in a jeep accident. Four days after the injury a systolic murmur was heard at the apex. The patient responded to treatment and 4 months later his only symptoms were moderate exertional dyspnea and fatigue. Right heart catheterization demonstrated tricuspid insufficiency, no evidence of shunt, and normal pressures in the right ventricle and pulmonary artery. Good repair of the ruptured papillary muscle was accomplished; however, massive hemorrhage from the right atrial appendage occurred within 12 hours, and the patient died.

Our patient, likewise, was in only moderate distress 3 months after his accident. He had easy fatigability, dyspnea on exertion, and mild orthopnea, but was ambulatory.

On the basis of these two examples and from theoretic considerations it would appear

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Figure 4

Biplane angiocardiogram, posteroanterior (left) and lateral view (right).
Figure 5

Diagrams of operative repair.
that rupture of a right ventricular papillary muscle situated in the low-pressure side of the heart is of less serious consequence than rupture on the left with its relatively high pressure. The clinical course is less dramatic and the diagnosis must be sought carefully. The diagnosis should be suspected when the usual features of tricuspid insufficiency make their initial appearance following chest trauma or during the course of endocarditis. The only obvious physical finding may be marked pulsation in the neck veins, since the low pressures in the right ventricle are not associated with loud harsh murmurs or obvious chest wall thrills.

Initial treatment should be directed at the primary associated condition; i.e., severe trauma or endocarditis. Whether or not open-heart surgical intervention is indicated will be a matter for decision in individual cases. The one patient who survived such surgery is 50 per cent improved.

**Summary**

A patient with isolated traumatic rupture of the anterior papillary muscle of the tricuspid valve is described. This is the second such case in the literature.

Clinical observations, hemodynamic data, and details of surgical treatment are discussed.

The diagnosis should be suspected in any patient who initially develops clinical evidence of tricuspid insufficiency following mechanical trauma to the chest or during the course of endocarditis.

Treatment is directed at the associated primary condition, i.e., severe trauma elsewhere in the body or endocarditis. The indications for open-heart surgery will be a matter for decision in individual cases.

**References**

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