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SURGICAL TREATMENT OF TRAUMATIC TRICUSPID INSUFFICIENCY: REPORT OF A CASE AND REVIEW OF OPERATED CASES

BY

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ABSTRACT

The incidence of blunt injury to the heart is becoming more frequent with the increasing number of automobile accidents. However, traumatic tricuspid insufficiency is less frequently encountered. This report concerns the hemodynamic data and operative treatment of a patient with traumatic rupture of the chordae tendineae to the anterior leaflet of the tricuspid valve by the Starr-Edwards disc valve 7 years and 5 months after the injury. A review of the surgically treated cases suggested that the number of valve repair could be increased when treated at an earlier stage.

CASE REPORT

A 52-year old man was in good health until February, 1962, when he was involved in an automobile accident. He was brought to the nearest hospital in a comatose condition and 5 days later he regained consciousness. Initial examination revealed a fracture of several ribs. After 3 months he was discharged and remained well except for a slight back pain for 2 years and 6 months at which time he developed progressive swelling of the abdomen and legs, followed by shortness of breath and easy fatigability. He had been admitted several times with a diagnosis of constrictive pericarditis but no relief of the symptom was obtained. He was referred and admitted to the Tokyo Medical and Dental University Hospital on June 16, 1969, 7 years and 4 months after the severe automobile accident.

On clinical examination, he appeared chronically ill and was moderately distressed. The jugular venous pressure was greatly elevated and displayed large collapsing systolic waves. The venous pressure was recorded

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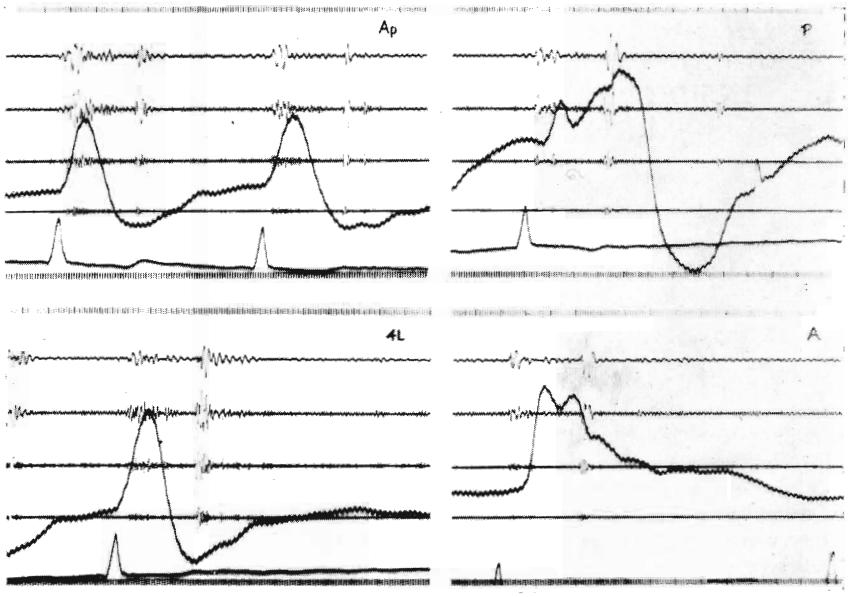
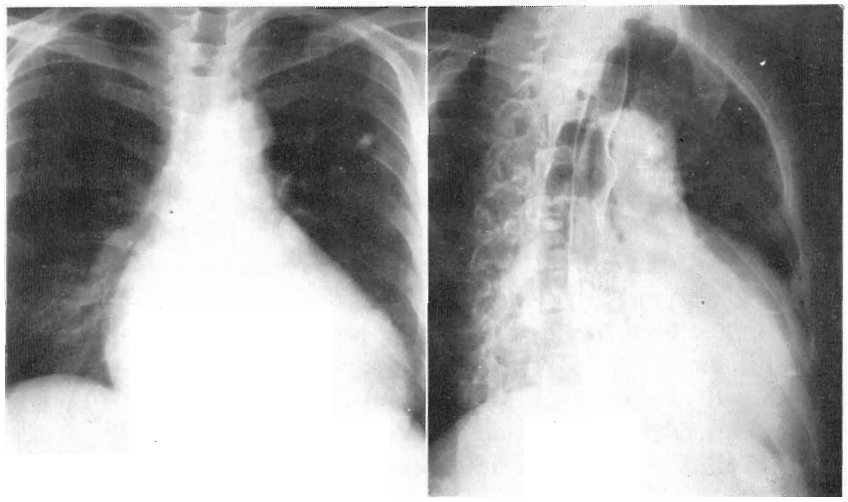


Fig. 1. Phonocardiogram.



Postoro-anterior view Right anterior oblique view

Fig. 2. Preoperative roentgenogram of the chest. Note the right atrial and ventricular enlargement.

as 155 mm of H_2O. Edema was moderate and was seen on the face and lower extremities. The blood pressure was 114/72 mm Hg with a pulse rate of 52 per minute which was irregular. The precordium was slightly elevated and there was a slight right ventricular lift in the 4th intercostal

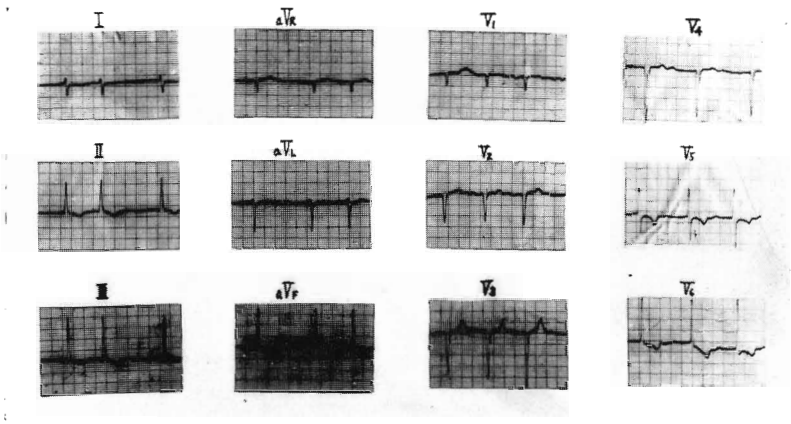


Fig. 3. Electrocardiogram. Atrial fibrillation with right axis deviation. The chest leads show left ventricular overloading pattern.

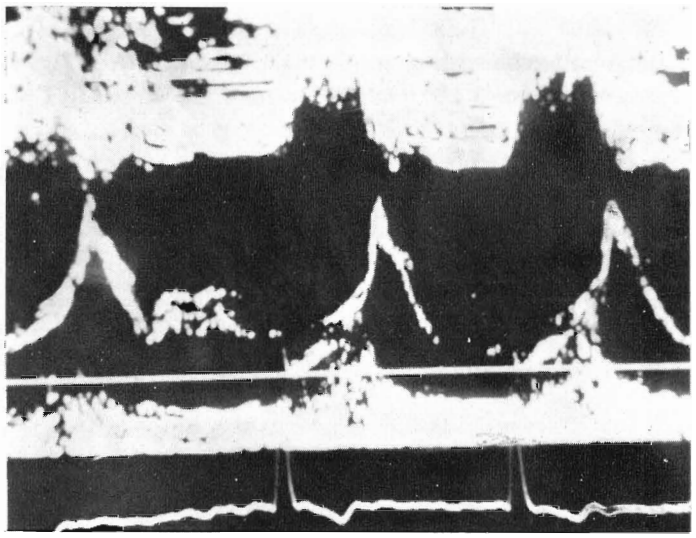


Fig. 4. Ultrasound echocardiogram of the tricuspid valve shows the diastolic descent slope of 240.9 mm/sec.

space along the left sternal border. The apex beat was 1.5 finger-breadths lateral to the midclavicular line. A grade 2/6 pansystolic murmur which did not increase in intensity during inspiration was heard at the left sternal border (Fig 1). The liver was enlarged to 4 finger-breadths below the costal margin in the midclavicular line and pulsation of the liver was palpable. There was a positive hepatojugular reflux. The routine hematologic studies, liver function tests, serum electrolytes were reported as showing no abnormality. The chest films (Fig. 2) were suggestive of right atrial enlarge-

Table 1. Cardiac catheterization

	Pressure (mmHg)
Superior vena cava	30/13
Inferior vena cava	30/13
Right atrium	30/5
Right ventricle	30/6
Cardiac output	5.0 L/min
Cardiac index	3.1 L//M²/min
Stroke volume	116.2 ml/beat
Stroke index	72.1 ml/M²/beat
$\dot{Q}R/\dot{Q}F$ (%)	
Korner-Shillingford's 1/slope method	126%
Direct method	102%

ment in the posteroanterior and lateral projections. Pulmonary vascular markings were not prominent and pericardial calcification was not observed. The electrocardiogram (Fig. 3) showed atrial fibrillation with a right axis deviation and chest leads showed a left ventricular overloading pattern. Ultrasound echocardiogram of the tricuspid valve (Fig. 4) revealed a diastolic descent slope of 240.9 mm/sec which was suggestive of tricuspid insufficiency.

Cardiac catheterization studies (Table 1) were performed to evaluate the clinical suspicion of severe tricuspid incompetence. The intracardiac and intravascular pressures were typical of severe tricuspid insufficiency. The right atrial pressure was 30/5 and almost identical with the right ventricular pressure of 30/6 mm Hg. Regurgitant volume was estimated using the indicator dilution curves. By direct method $\dot{Q}R/\dot{Q}F$ was 104% and 126% by Korner-Shillingford's 1/slope method. Catheter, prevented by regurgitant flow, could not be inserted into the pulmonary artery.

Operation was performed on July 9, 1969. With the patient in the supine position, the heart was approached through the median sternotomy. The fibrous adhesion was observed between the heart and parietal pericardium which was easily freed, and adhesion was the strongest at the right atrium where a sac was formed and contained 5 ml of clear liquid (Fig. 5). The right atrium and ventricle were grade 5/6 enlarged and systolic thrill was not palpated over the right atrium, probably due to the ventricularization of the right atrium. The patient was placed on total cardiopulmonary bypass using disposable sheet oxygenator primed with Ringer-lactate solution. The tricuspid valve orifice was dilated to 4 finger-breadths. The chordae tendineae of the anterior leaflet of the tricuspid valve was found to be ruptured (Fig. 6). The repair of the valve was first tried but the possible hazard of postoperative care due to the residual incompetence prompted us to insert the 4-M Starr-Edwards disc valve (Model 6500) into

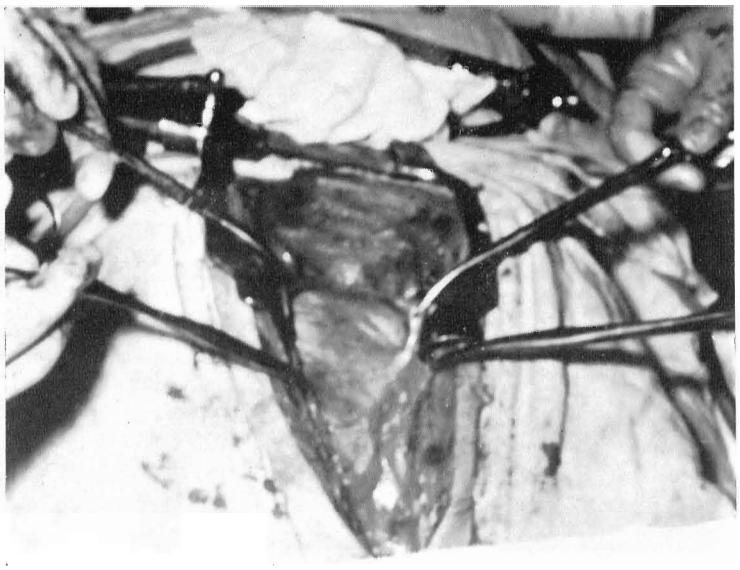


Fig. 5. The right atrium is severely adhered to the pericardium and a cyst is found containing 5 ml of clear liquid.

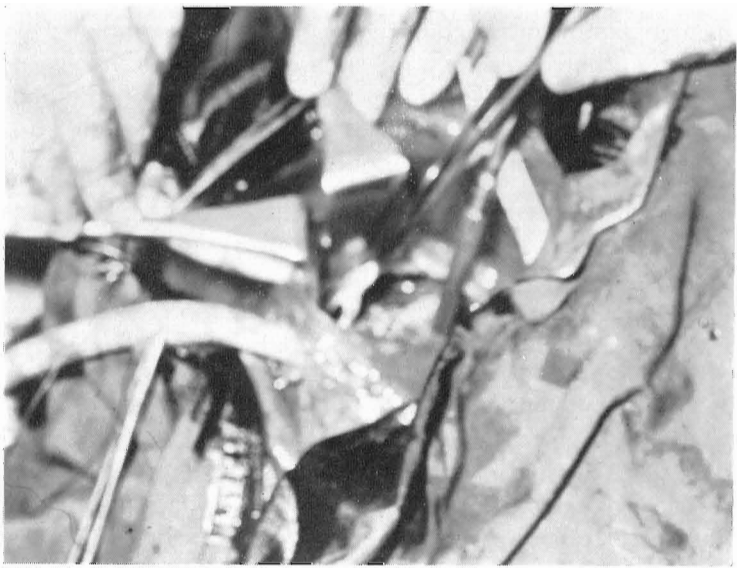
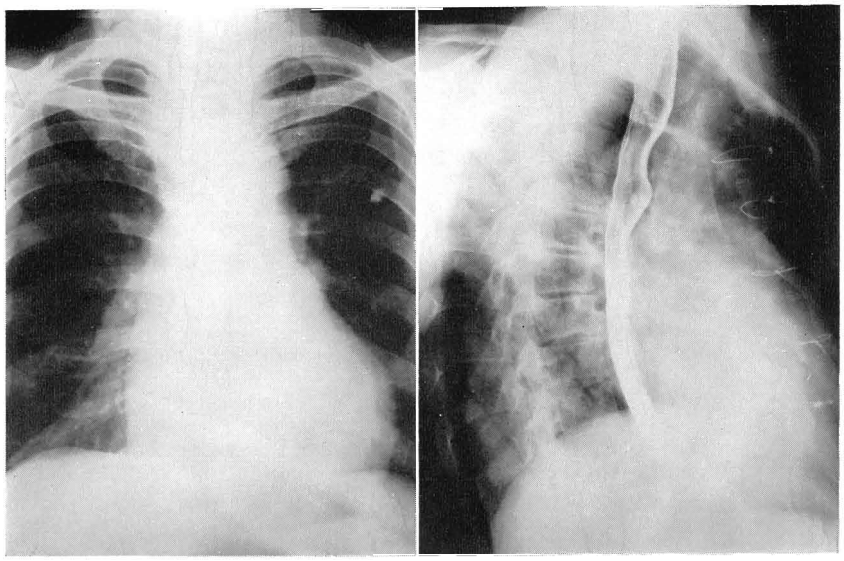


Fig. 6. The tricuspid valve with rupture of chordae tendineae to the anterior leaflet.



Postoro-anterior view Right anterior oblique view

Fig. 7. Postoperative roentgenogram of the chest. Note the diminution in the size of the right heart.

the tricuspid orifice. Except for the transient respiratory distress, the patient took a benign postoperative course. The neck vein pulsation disappeared and the central venous pressure was 80 mm of H_2O on the 7th postoperative day. The liver swelling also disappeared. Postoperative chest x-rays revealed diminution in the size of the right heart (Fig. 7). The patient was anticoagulated and has had no complication to date.

Discussion

Traumatic rupture of the papillary muscle occurs in less than 5% of autopsied blunt heart injury cases, and when it occurs it is usually combined with cardiac rupture or other cardiac injuries[1]. When it was seen clinically, valvular lesion was usually isolated and tricuspid valvular lesion was less frequently seen than the aortic and mitral valve lesions. Fifteen cases of isolated tricuspid insufficiency and one with traumatic ventricular septal defect have been reported[2-10]. Of the 16 tricuspid lesions found in the literature, 13 cases were due to automobile accidents, 2 followed by direct blows to the chest and one as the result of a blast injury. With the increasing number of automobile accidents, the incidence of rupture of papillary muscles, chordae tendineae or valve leaflets has become more frequent. If the combination of violent compression of the heart associated with some obstruction to the great vessel outflow occurs during diastole or

early systole, when the heart is distended, the tricuspid valves are also closed and their supporting chordae tendineae and papillary muscles are under tension, the rupture of one or more of these structures is likely to occur.

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. On the contrary, the rupture of the papillary muscle situated on the low pressure side of the heart is of less serious consequence than the rupture on the left with its relatively high pressure. Time lapse from injury to operation ranged from 3 days to 24 years (Table 2). All these cases had rather mild symptoms except in one case with traumatic ventricular septal defect[8]. Symptoms caused by tricuspid insufficiency have generally been mild and have appeared gradually according to the severity of the hemodynamic changes, consisting of easy fatigability, shortness of breath on exertion due to low cardiac output, and liver swelling and edema due to right heart failure. In our case, symptoms of the right heart failure appeared 2 years and 7 months after the injury.

The indication for open heart surgery will be a matter for decision in individual cases. One positive indication for surgery has been the presence of chronic right-sided heart failure and residual debilitating symptomatology not relieved by adequate medical management. Patients who had a surgical treatment for tricuspid incompetence (Table 2) improved greatly except in Brandenburg's case[5] who had an operation 24 years after the injury. The patient still had fatigue and dyspnea on moderate exertion, and there had been no decrease in the heart size. The findings at the time of operation were rupture of the papillary muscle, chordae tendineae of the anterior leaflet and valvular laceration. Several of the cases had multiple damages and one was described as virtually complete valve destruction. Three cases out of 10 were successfully repaired and others had a valve replacement of various types. The use of an artificial valve for replacement of the valve has become the method of choice. However, thromboembolic complications have been reported to occur in approximately 10 to 30% of the cases[12,13]. Mechanical failures or outflow tract obstruction were noted as a cause of postoperative death[14,15]. It would seem that papillary muscle and valvular reconstruction would be the preferred method of management[11]. Repair of the valve was accomplished in only three cases and they were operated upon within 5 months from the time of the injury. The other 7 cases required valve replacement. In our case, repair of the valve was first tried, but possible hazard of postoperative care due to severe residual incompetence prompted us to insert the artificial valve into the tricuspid orifice. If the patient were treated for tricuspid insufficiency at an earlier date, it might have been possible to repair the valve without inserting the artificial

Table 2. Reported cases of operated traumatic tricuspid insufficiency

Case No.	Author	Year	Age	Sex	Cath. data RA V waves pressure	RV mmHg	Pathology	Time from injury to surgery	Operation
1.	Cooley et al.	1959	28	M	11		Rupt. ant. pap. muscle	4 mo	Repair
2.	Osborn et al.	1964	33	M	19.5	20/5	Rupt. ant. pap. muscle Laceration 2 valve cusps Patent foramen ovale	3 mo	Repair
3.	Bjork	1965	44	F	18	24/8	Rupt. chordae and ant. and septal cusp	1 yr 7 mo	Starr-Edwards 4M
4.	Brandenburg et al.	1966	42	M	28	28/12	Rupt. chordae and ant. Patent foramen ovale	24 yrs	Starr-Edwards 4M
5.	Shabetai et al.	1966	45	M	15	15/3	Complete valve destruction	10 yrs	Starr-Edwards 4M
6.	Jahnke et al.	1967	30	M	19	20/9	Rupt. ant. post. papillary muscle	3 days	Hufnagel lenticular valve
7.	Inoue et al.	1968	26	M	34	58/0	Rupt. chord. ant. cusp	5 mo	Repair
8.	Shabetai et al.	1969	39	M	15	38/14	Rupt. chord. ant. cusp	5 mo	Kay-Shiley disc valve No. 5
9.	Aoki et al.	1969	24	M			Rupt. ant. pap. muscle Patent foramen ovale	4 yrs 7 mo	Starr-Edwards 4M
10.	Yamada et al.	1969	52	M	30	30/6	Rupt. chord. ant. cusp	7 yrs 5 mo	Starr-Edwards disc valve 4M

prosthesis. It has been confirmed by postoperative evaluation that the valve replacement is superior in restoring the normal hemodynamic pressure relationships in the right heart. Jahnke et al.[7] mentioned that when patients are operated at an earlier date it might be followed by a more pronounced improvement. And they have 3 patients, who received the injury 3 to 5 years ago, not operated on yet and doing well. Furthermore, they mentioned that experience to date would seem to indicate that all patients will eventually require surgery. When papillary muscle or chordae tendineae ruptures and become incompetent for many years, dilatation of the tricuspid orifice, elongation, shortening of the chordae tendineae of the valve were likely to occur. Shabetai et al.[6] found that the chordae tendineae were shortened along the wall and the rupture could not be identified and only the remnants of the tricuspid valve tissue remained in a case treated 10 years after the injury. In Brandenburg's case operated after 24 years[5], the valve had become very fragile and atrophic. Even a light touch with the forceps caused tear of the valve.

In reviewing all of these cases, it now becomes clear that an operation should be done as soon as a diagnosis of tricuspid insufficiency has been established. By operating the patient at an earlier date, the reconstruction of the valve or papillary muscle is possible and insertion of the artificial prosthesis could be avoided.

References

1) Palmley, L. P. et al.: Nonpenetrating traumatic injury of the heart. Circulation, 18: 371–396, 1958.
2) Cooley, D. A. et al.: Ventricular aneurysm following myocardial infraction. Ann. Surg., 150: 595–611, 1959.
3) Osborn, J. R. et al.: Traumatic tricuspid insufficiency. Circulation, 30: 217–222, 1964.
4) Björk, V. O.: Traumatic rupture of the tricuspid valves. Thoraxchir. Vaskul. Chir., 12: 368–372, 1965.
5) Brandenburg, R. O. et al.: Traumatic rupture of the chordae tendineae of the tricuspid valve: Successful repair 24 years later. Am. J. Cardiol., 18: 911–915, 1966.
6) Shabetai, R. et al.: Successful replacement of the tricuspid valve ten years after traumatic incompetence. Am. J. Cardiol., 18: 916–920, 1966.
7) Jahnke, E. J., Jr. et al.: Tricuspid insufficiency: The result of nonpenetrating trauma. Arch. Surg., 95: 880–886, 1967.
8) Inoue, T. et al.: A successful operated case of nonpenetrating cardiac injury. (in Japanese). J. Japan. Assoc. Thorac. Surg., 16: 1182–1187, 1968.
9) Shabetai, R. et al.: Traumatic hemopericardium with tricuspid incompetence. J. Thorac. Cardiovasc. Surg., 57: 294–297, 1969.
10) Aoki, E. et al.: A successful operated case of traumatic tricuspid insufficiency with cyanosis. (in Japanese). Paper presented at the Japan. Circulation Society, 54th Kanto-Koshinetsu Section Meeting.
11) Kay, J. H., and Egerton, W. S.: The repair of mitral insufficiency associated with ruptured chordae tendineae. Ann. Surg., 157: 351–360, 1963.

12) Cooley, D. A. et al.: Mitral valve replacement with a discoid prosthesis. Ann. Thorac. Surg., 3: 487–502, 1967.
13) Starr, A. et al.: Mitral replacement: Review of six years' experience. J. Thorac. Cardiovasc. Surg., 54: 333–358, 1967.
14) Newman, M. M. et al.: Mechanical failure of Starr-Edwards aortic prosthesis due to ball fracture. J. Thorac. Cardiovasc. Surg., 53: 398–400, 1967.
15) Roberts, W. C., and Morrow, A. G.: Cause of early operative death following cardiac valve replacement. J. Thorac. Cardiovasc. Surg., 54: 422–437, 1967.