Traumatic tricuspid valve insufficiency

Experience in thirteen patients

From 1964 through June 1993, thirteen patients with traumatic tricuspid insufficiency were treated surgically; all were male, and the ages ranged from 17 to 64 years (median 39 years). The condition was associated with blunt chest trauma in all patients: motor vehicle accidents in twelve and an explosion of a tank of compressed air in one. The median duration between trauma and operation was 17 years (range 1 month to 37 years). Preoperatively, six patients were in sinus rhythm and seven were in atrial fibrillation. At operation, the right ventricular function appeared moderately to severely depressed in twelve patients. In twelve patients, the anterior leaflet was flail because of chordal rupture ($n = 9$), rupture of anterior papillary muscle ($n = 3$), or tear in the anterior leaflet ($n = 1$). In one patient, the septal leaflet was missing and in another it was retracted and adherent to the ventricular septum. In five patients the tricuspid valve was repaired and in eight it was replaced. In seven patients in the latter group, the chordae, papillary muscles, and/or tricuspid valve leaflet(s) were found to be in a contracted and atrophic state, precluding repair. No early or late deaths occurred. At follow-up extending to 26 years (median 12 years), 12 patients are in New York Heart Association class I and one patient is in class II. Nine patients were in sinus rhythm and four were in atrial fibrillation. Although our experience indicates that good functional results can still be achieved many years after the onset of traumatic tricuspid valve insufficiency, earlier diagnosis and surgical treatment should increase the feasibility of tricuspid valve repair, prevent progressive deterioration of right ventricular function, and increase the possibility of maintaining late sinus rhythm in a greater number of patients.
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Traumatic tricuspid valve insufficiency, although being a relatively uncommon condition, has been reported with increasing frequency during the past 35 years.[1-20] Traditionally, the tricuspid valve has usually been replaced in this condition. During the past decade, however, there have been reports of successful repair of the tricuspid valve, allowing a more physiologic approach to traumatic tricuspid valve insufficiency.[13, 15, 18-20] In this article we report our experience with surgical treatment of traumatic tricuspid valve insufficiency.

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Patients and methods

Between 1964 and 1993, thirteen patients underwent operation for traumatic tricuspid valve insufficiency. The patient data are summarized in Table I. All patients were male. The ages ranged from 17 to 64 years (median 39 years, mean = 36.6 years). In all thirteen patients the tricuspid insufficiency was attributed to blunt chest trauma: motor vehicle accident ($n = 12$) and explosion of a tank of compressed air ($n = 1$, patient 2). The diagnosis of a traumatic cause was based on a history of severe chest trauma combined with one or more operative findings compatible with tricuspid valve trauma including an avulsed papillary muscle, ruptured chordae, lacerated valve leaflet(s), and dehiscence of valve leaflet(s) from the anulus. Many patients also had rupture of the pericardium or other traumatic cardiac lesions including aortic cusp rupture, atrial or ventricular septal defect, left ventricular–right atrial fistula, and ventricular aneurysm with normal coronary arteries. Some patients had a new cardiac murmur and many had associated hemopericardium and/or adhesive or constrictive pericarditis. Twelve patients were operated on at the Mayo Clinic, three of whom have been included in earlier reports,[21-23] and one patient was operated on elsewhere by the senior author (G.K.D.).[24] All

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Table I. *Patient data*

Patient	Year of operation	Age (yr)	Interval, trauma to operation (yr)	Preop. rhythm	Prior operation	Operative findings	Associated injuries	Procedure
1	1964	42	24	AFib		Chordal rupture ant. leaflet, PFO	Closed head injury	TV replacement, closure PFO
2	1967	44	18	AFib	Closure PFO (Mayo Clinic)	Ant. papillary muscle rupture, PFO	None	TV replacement, closure PFO
3	1967	39	½	SR		Chordal rupture ant. leaflet, constrictive pericarditis, hemopericardium	Multiple rib fractures	TV replacement, pericardiectomy
4	1968	17	½	SR		Ant. papillary muscle rupture, traumatic VSD	Vertebral fracture (L-4)	TV replacement, closure VSD
5	1978	42	20	SR		Chordal rupture and tissue defect of ant. leaflet, absent left pericardium	Wrist fracture	TV repair
6	1981	29	6	SR		Chordal rupture ant. leaflet, PFO	Wrist fracture, multiple rib fractures	TV repair, closure PFO
7	1981	21	1	SR		Chordal rupture ant. leaflet, prolapse and partial avulsion of right aortic cusp, right sinus of Valsalva aneurysm, constrictive pericarditis	Femur and mandibular fractures, closed head injury	TV repair, resuspension right aortic cusp, closure right sinus of Valsalva aneurysm, pericardiectomy
8	1988	49	29	SR		Chordal rupture ant. leaflet	Closed head injury	TV repair, right reduction atrioplasty
9	1989	40	25	AFib	Closure traumatic ASD and VSD (elsewhere)	Tear ant. leaflet, missing septal leaflet, left ventricular–right atrial fistula, PFO, ASD	Ruptured spleen	TV replacement, closure left ventricular–right atrial fistula, closure PFO, closure ASD
10	1989	25	17	AFib	Closure traumatic VSD (elsewhere)	VSD, left ventricular aneurysm, ruptured septal leaflet, PFO	Closed head injury	TV replacement, revision of ventricular septal patch, resection left ventricular aneurysm, closure PFO, right reduction atrioplasty
11	1991	64	37	AFib		Chordal rupture ant. leaflet	Closed head injury	TV repair, right reduction atrioplasty
12	1993	32	10	AFib		Ant. papillary muscle and chordal rupture	Femur fracture, closed head injury	TV replacement, right reduction atrioplasty
13	1993	32	16	AFib		Chordal rupture ant. leaflet, fibrosis ant. papillary muscle, PFO, pericardial rupture	Femur fracture, closed head injury	TV replacement, right reduction atrioplasty, closure PFO

AFib, Atrial fibrillation; *ASD*, atrial septal defect; *SR*, sinus rhythm; *PFO*, patent foramen ovale; *TV*, tricuspid valve; *VSD*, ventricular septal defect.

twelve patients who were involved in motor vehicle accidents had additional injuries (Table I). Patient 5 had associated congenital absence of the left pericardium with displacement of the heart into the left pleural space.[23]

Symptoms included fatigue, dyspnea, orthopnea, palpitations, and vague chest pain. Physical findings included peripheral edema, hepatic congestion, ascites, a prominent V wave in the external jugular vein, and a loud systolic murmur that increased with inspiration. Preoperatively, six patients were in normal sinus rhythm and seven patients were in atrial fibrillation. The intervals between trauma and operation for the entire group ranged from 1 month to 37 years (median 17 years, mean 16 years). The intervals between trauma and operation for the patients in sinus rhythm ranged from 1 month to 29 years (median 3.5 years, mean 9.4 years), and for the patients in atrial fibrillation the intervals ranged from 10 to 37 years (median

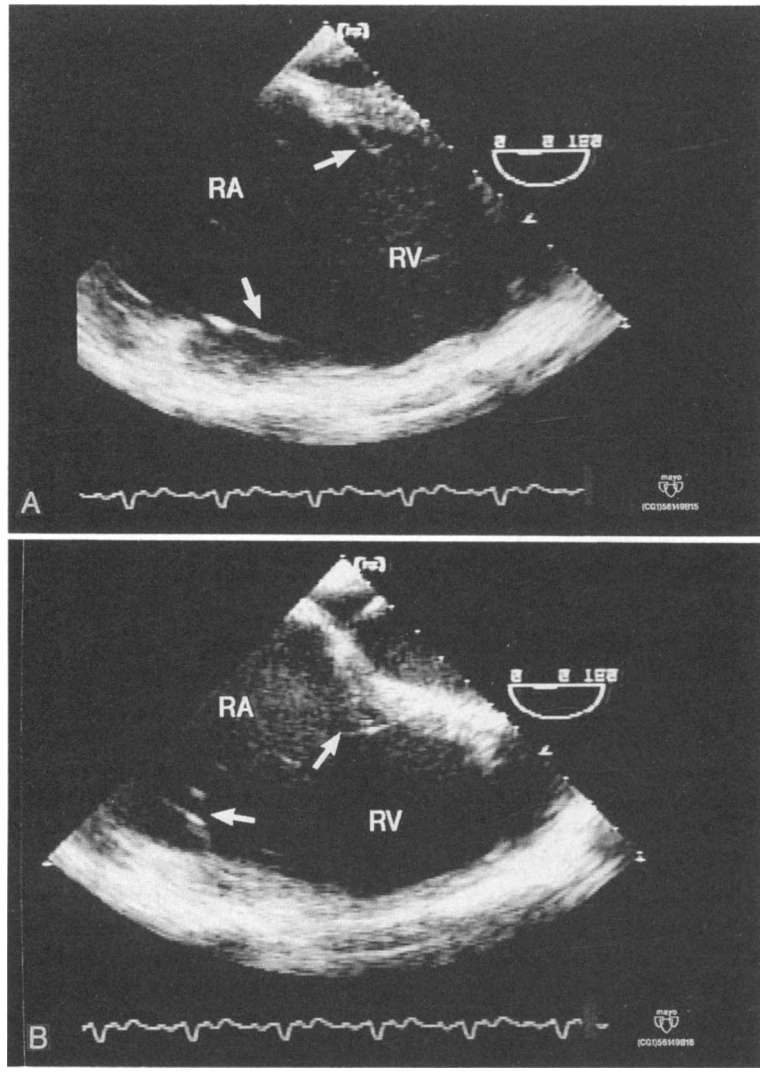

Fig. 1. Multiplane transesophageal examination from patient 12 with traumatic tricuspid insufficiency. The plane of ultrasound is horizontal to the body, as indicated in the small icon on the upper right. **A,** In diastole, the anterior *(lower arrow)* and septal *(upper arrow)* tricuspid leaflets are opened. There is marked enlargement of the tricuspid anulus and of the right ventricle *(RV)* and right atrium *(RA)*. **B,** In the systolic frame, the septal leaflet is properly supported *(upper arrow)* and the anterior leaflet *(lower arrow)* is flail, moving into the right atrium.

18 years, mean 21.0 years) (Mann-Whitney test for the means, $p = 0.12$). The electrocardiograms showed complete or incomplete conduction delays of the right bundle branch type in all patients. The diagnosis of severe tricuspid regurgitation was made angiographically in the first four patients (in the 1960s) and echocardiographically (Fig. 1) in the subsequent nine patients. Two patients had constrictive pericarditis; in the remaining eleven patients, the right ventricle was moderately to markedly dilated. Right ventricular function was moderately to severely depressed in twelve patients. Six patients had a patent foramen ovale with a predominantly right-to-left shunt; one of these had an additional atrial septal defect. In three patients (two seen in the 1950s, one at the Mayo Clinic and one elsewhere, and one recently seen elsewhere), the initial diagnosis was Ebstein's anomaly.

Three patients had undergone prior operations shortly after initial trauma, two elsewhere and one at our institution (see Table I). Patient 9 had undergone attempted repair of traumatic atrial and ventricular septal defects; when seen at our institution he had a residual atrial septal defect, a left ventricular–right atrial fistula, and severe tricuspid insufficiency. Patient 10 had closure of a traumatic ventricular septal defect 17 years before being evaluated at our institution for left ventricular aneurysm and severe tricuspid insufficiency. Patient 2 was first seen at the Mayo Clinic in 1953, when a diagnosis of Ebstein's malformation with a patent foramen ovale and right-to-left shunt was

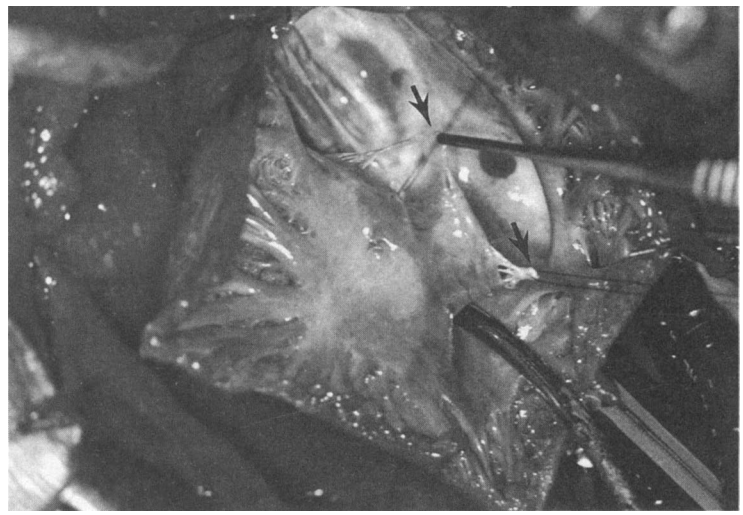

Fig. 2. Operative view of tricuspid valve from the same patient as in Fig. 1. Note the flail anterior leaflet and the ruptured papillary muscle *(upper arrow)* and ruptured chordae *(lower arrow)*.

made.[22] In retrospect, this diagnosis was made 4 years after closed chest trauma. In the same year, the atrial well technique was used to close the patent foramen ovale. In 1967, because of increasing cardiac disability resulting from severe tricuspid insufficiency, reoperation was performed for tricuspid valve replacement and closure of a partially patent foramen ovale. Findings at operation confirmed that the tricuspid regurgitation was due to trauma rather than to Ebstein's malformation.

Results

At operation, a median sternotomy was performed in all patients and cardiopulmonary bypass was instituted. The tricuspid valve was exposed through a right atriotomy. In twelve patients, the anterior leaflet of the tricuspid valve was completely or partially flail as a result of chordal rupture ($n = 9$), rupture of the anterior papillary muscle ($n = 3$), or tear in the anterior leaflet ($n = 1$). The latter patient (patient 9) also had a missing septal leaflet. In another patient (patient 10), the septal leaflet was retracted and adherent to the ventricular septum. A patent foramen ovale was confirmed in all six patients in whom it was diagnosed before the operation. Additional operative findings are listed in Table I. In five patients the tricuspid valve was repaired by one or more of the following procedures: annuloplasty ($n = 5$; pursestring technique in four and Carpentier-Edwards ring [Baxter Healthcare Corp., Edwards Div., Irvine, Calif.] in one), plication of a flail anterior leaflet ($n = 4$), and plication of elongated chordae ($n = 3$). In eight patients, tricuspid valve replacement was performed instead of valve repair primarily because the involved leaflet(s) and/or subvalvular apparatus were contracted (Fig. 2): Carpentier-Edwards bioprosthesis ($n = 3$); Starr-Edwards valve ($n = 3$); St. Jude Medical valve (St. Jude Medical, Inc., St. Paul, Minn.) ($n = 1$); and Kay-Shiley valve (Shiley, Inc., Irvine, Calif.) ($n = 1$).

Additional procedures included closure of patent foramen ovale (one with additional atrial septal defect, $n = 6$), right reduction atrioplasty ($n = 5$), resection of constricting parietal and visceral pericardium ($n = 2$), closure of traumatic ventricular septal defect ($n = 1$), replacement of a redundant ventricular septal patch that was bulging too far into the right ventricular cavity ($n = 1$), resection of traumatic left ventricular aneurysm ($n = 1$), resuspension of partially avulsed right aortic cusp ($n = 1$), closure of right sinus of Valsalva aneurysm ($n = 1$), and closure of left ventricular–right atrial fistula ($n = 1$).

In all patients, the early postoperative course was uncomplicated. At follow-up extending to 26 years (median 12 years, mean 12.6 years), twelve patients are in New York Heart Association class I and one patient is in class II. The latter patient (patient 11) had the longest interval between trauma and operation (37 years), and he continues to have atrial fibrillation. Patient 3 had rereplacement of a stenotic Kay-Shiley valve 22 years after tricuspid valve replacement; he is currently in class I. Patient 4 required reclosure of a residual ventricular septal defect. Among the six patients who were in sinus rhythm before the operation, sinus rhythm persisted in four and chronic atrial fibrillation developed in two. Among the seven patients who were in atrial fibrillation before the operation, sinus rhythm developed shortly after the operation in five and atrial fibrillation persisted in two.

Discussion

Cardiac trauma encompasses a large spectrum of presentations. Its epidemiology has recently been reviewed by Mattox and colleagues.[25] Cardiac injury resulting from nonpenetrating chest trauma, especially when caused by vehicular accidents, is often found in combination with other organ system injuries, which tend to obscure the cardiac damage. A review by Parmley, Manion, and Mattingly[1] in 1958 reported the infrequency with which the diagnosis was made at that time, mainly because of failure to appreciate the relationship between blunt chest trauma and cardiac lesions. Because of increased awareness of this relationship and the wide application of two-dimensional echocardiography with Doppler and color flow imaging during the past two decades, traumatic cardiac lesions have been reported with increasing frequency.[2-20] Despite this trend, however, it is apparent from our and other reports that the interval between the event of the injury and the diagnosis of the cardiac lesion may still be many years.

Although traumatic tricuspid insufficiency is a relatively uncommon lesion, its frequency is probably underestimated.[17] The pathophysiology of the injury presumably involves a severe elevation of right ventricular intracavitary pressure from sudden compression of the heart. As in our series, the most frequently reported injury is chordal rupture, followed by rupture of the anterior papillary muscle and leaflet tear, primarily of the anterior leaflet.[16]

Rupture of the papillary muscle typically becomes symptomatic rapidly, whereas ruptured chordae or torn leaflets may have a more insidious onset of symptoms. Exertional dyspnea and fatigue are the most common early symptoms. If untreated, progressive dilatation of the tricuspid anulus occurs and right heart failure eventually develops, usually associated with atrial fibrillation.[16] Some patients become cyanotic because of stretching of the right atrium with resultant enlargement of a patent foramen ovale and right-to-left shunting.[12, 21, 22] The finding of cyanosis in combination with cardiomegaly, dilatation of the right side of the heart, and right bundle branch block may suggest Ebstein's malformation, which was the initial diagnosis in three of our patients.[21, 22] This misdiagnosis underscores the value of including questions about chest trauma in the cardiac review of symptoms.

In the last nine patients in our series, two-dimensional echocardiography allowed visualization of the enlarged right atrium and right ventricle and the tricuspid valve abnormality including ruptured chordae and flail tricuspid leaflets. More recently, color flow imaging has improved documentation of tricuspid regurgitation and has provided semiquantitation of its severity.[26, 27] The recent introduction of transesophageal imaging has provided increased resolution, which allows more precise definition of specific tricuspid valve lesions.

Even though isolated tricuspid regurgitation may initially be clinically benign, moderate-to-severe right heart failure eventually developed in twelve of our thirteen patients. Traditionally, this has been the indication for surgery, which usually consisted of tricuspid valve replacement. When operative intervention is unduly delayed, however, irreversible right ventricular myocardial dysfunction may develop. The only patient is our series who did not have right ventricular dysfunction before the operation had the shortest interval between trauma and operation (1 month). Long-term results will likely be better if operation is performed before right ventricular function deteriorates, rather than after the onset of progressive right heart failure. This improved outlook may be particularly true if the ruptured tricuspid valve can be successfully repaired rather than replaced. Although the literature contains little information concerning the late results of tricuspid valve repair, we believe results should be better with repair than with valve replacement because the geometry and function of the right ventricle are better preserved and complications inherent in prosthetic heart valves are avoided. This would be analogous to the improved late results of mitral valve repair compared with valve replacement.[28] However, the small number of patients in this series does not allow statistical support of this contention.

Although tricuspid valve repair had been performed sporadically as early as the late 1950s and 1960s,[1, 2] during the past decade more patients have benefited from repair as a result of improved surgical techniques, new technical developments, widespread use of intraoperative echocardiography to document the status of the repair, and general agreement that valve repair, when feasible, should be superior to valve replacement.[13, 15, 18-20] The operative technique will be dictated primarily by the specific injury encountered at the time of operation. If the injury is limited to the chordae tendineae, papillary muscle, or a leaflet, repair can usually be effected. The recent use of artificial chordae may facilitate salvage of some valves.[29] If operation is delayed, in our experience and that of others, the papillary muscles, the chordae tendineae, and the involved leaflet(s) are frequently found in a contracted and atrophic state, precluding valve repair. Therefore, we believe a shorter duration between trauma and operation may be advantageous in terms of feasibility of tricuspid valve repair. Additionally, although the small number of patients does not lend statistical support, we believe early repair should limit right atrial dilatation and increase the likelihood of maintaining or restoring sinus rhythm.

Addendum

Subsequent to the submission of this manuscript we have operated on three more patients with traumatic tricuspid insufficiency. Two were in motor vehicle accidents (both women) and one was kicked by a cow (a man). Tricuspid valve repair was performed in two patients and valve replacement in one; all three are well on follow-up.

We appreciate the opportunity of including data from patients operated on by Drs. F. H. Ellis, Jr, J. W. Kirklin, D. C. McGoon, J. M. Piehler, and R. B. Wallace.

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