Eur J Cardio-thorac Surg (1996) 10:867–873

ORIGINAL ARTICLE

F. Maisano
R. Lorusso
L. Sandrelli
L. Torracca
G. Coletti
G. La Canna
O. Alfieri

Valve repair for traumatic tricuspid regurgitation

Received: 11 June 1995
Accepted: 20 October 1995

F. Maisano (✉) · R. Lorusso · L. Sandrelli · L. Torracca · G. Coletti · G. La Canna · O. Alfieri
II Division of Cardiac Surgery, Spedali Civili di Brescia, P. le Spedali Civili 1, I-25125 Brescia, Italy

Abstract *Objective.* The review of six cases of valve repair for traumatic tricuspid regurgitation in our institution and 74 in the literature in order to assess effective methods of treating this lesion.
Methods. Tricuspid valve regurgitation is a rare complication of blunt chest trauma. Optimal treatment for this condition is still controversial ranging from long-term medical therapy to early surgical correction. We followed the cases of six consecutive patients with post-traumatic tricuspid incompetence who were successfully treated with reparative techniques. All patients were male and their ages ranged from 18 years to 42 years. Valve regurgitation was always secondary to blunt chest trauma due to motor vehicle accident. The mechanism of valve insufficiency was invariably anterior leaflet prolapse due to chordal or papillary muscle rupture associated with annular dilatation. Surgical procedures included Carpentier ring implant (5 patients), Bex posterior annuloplasty (1 patient), implant of artificial chordae (4 patients), papillary muscle reinsertion (2 patients), commissuroplasty (1 patient) and "artificial double orifice" technique (1 patient).
Results. Tricuspid insufficiency improved in all patients after the correction. No complications were recorded and all patients were asymptomatic at the follow-up.
Conclusions. Since post-traumatic tricuspid regurgitation is effectively correctable with reparative techniques, early operation is recommended to relieve symptoms and to prevent right ventricular dysfunction. [Eur J Cardio-thorac Surg (1996) 10:867–873]

Key words Tricuspid valve insufficiency · Trauma · Valve repair

Tricuspid incompetence is a rare complication of blunt chest trauma. Around 100 cases have been reported in the literature. We have collected the reports on 74 patients who underwent surgical correction of this condition [2, 4, 6, 8, 9–11, 13–16, 18–22, 24–35, 37, 38, 40].

The real prevalence of traumatic tricuspid regurgitation is probably underestimated since chronic isolated tricuspid insufficiency is usually well tolerated and most patients experience few or no symptoms after the trauma [7]. Surgery is indicated before longstanding chronic volume overload produces right ventricular dysfunction [35]. Valve replacement and repair have been performed, depending on the specific lesions, with a slight prevalence of replacements over repairs. We report six cases of valve reconstruction for post-traumatic tricuspid regurgitation. Furthermore, the pathologic anatomy of the disease and the available surgical options are analyzed through an extensive review of the literature.

Material and methods

Patient population

Between April 1991 and October 1993, six patients were admitted to our institution with the diagnosis of post-traumatic tricuspid regurgitation. They were all male and their ages ranged between 18 and 42 years (mean 29.5 ± 9.2, median 30.5). All had a history of blunt chest trauma secondary to motor vehicle accident. Thoracic trauma had been isolated in only one case, while the others had had multiple injuries. The time lapse from the accident to the admission our hospital ranged between 2 months and 20 years. The diagnosis in all patients had been made by echocardiography in the referring center before admission to our institution. Other possible causes of isolated organic tricuspid incompetence were ruled out (e.g. endocarditis, rheumatic fever, active auto-immune or hormonal diseases).

Upon admission, two patients were in New York Heart Association (NYHA) class II, one was in class III and the remaining three patients were asymptomatic. A pansystolic murmur was audible at the left lower sternal border in all patients. The electrocardiogram (ECG) showed sinus rhythm associated with right bundle branch block in all patients (complete in 4). Transthoracic or transesophageal 2D-Doppler echocardiography was repeated to confirm the diagnosis (Table 1). In all patients, tricuspid regurgitation was severe (grade 4/4) associated with massive dilatation and deformation of the annulus. The anterior leaflet was flail/prolapsing in all patients, and one patient had associated prolapse of the posterior leaflet. The septal leaflet was never involved in the mechanism of regurgitation. Some degree of right ventricular overload was present in all patients (mean right ventricular end-diastolic dimension 52.4 ± 13.8 mm, range 30–60 mm).

In one patient (case no. 5), transesophageal echocardiography demonstrated intramural hematoma of the thoracic aorta at two sites: one about 2 cm above the sinotubular junction and extending for 1 cm towards the arch, and the other in the descending thoracic aorta, just above the diaphragm. Global left ventricular function as well as regional wall motion were impaired in this patient. The right ventricular free wall and the anterior half of the interventricular septum were akinetic and the anterior wall of the left ventricle was hypokinetic. In this patient, cardiac catheterization and aortography confirmed the echocardiographic findings and excluded coronary artery disease. Because of the potential reversibility of the contractile function in the case of myocardial contusion, the patient was discharged and treated with digoxin, diuretics and vasodilators. He was readmitted 6 weeks later when echocardiography showed a substantial recovery of myocardial contractility. At that point, simultaneous correction of the tricuspid and aortic lesions was advised.

One patient, who had been included early in the series, was completely asymptomatic on admission. Conservative medical treatment was first recommended, but 6 months later he underwent surgical correction because of the progression of right ventricular dilatation.

Surgical techniques

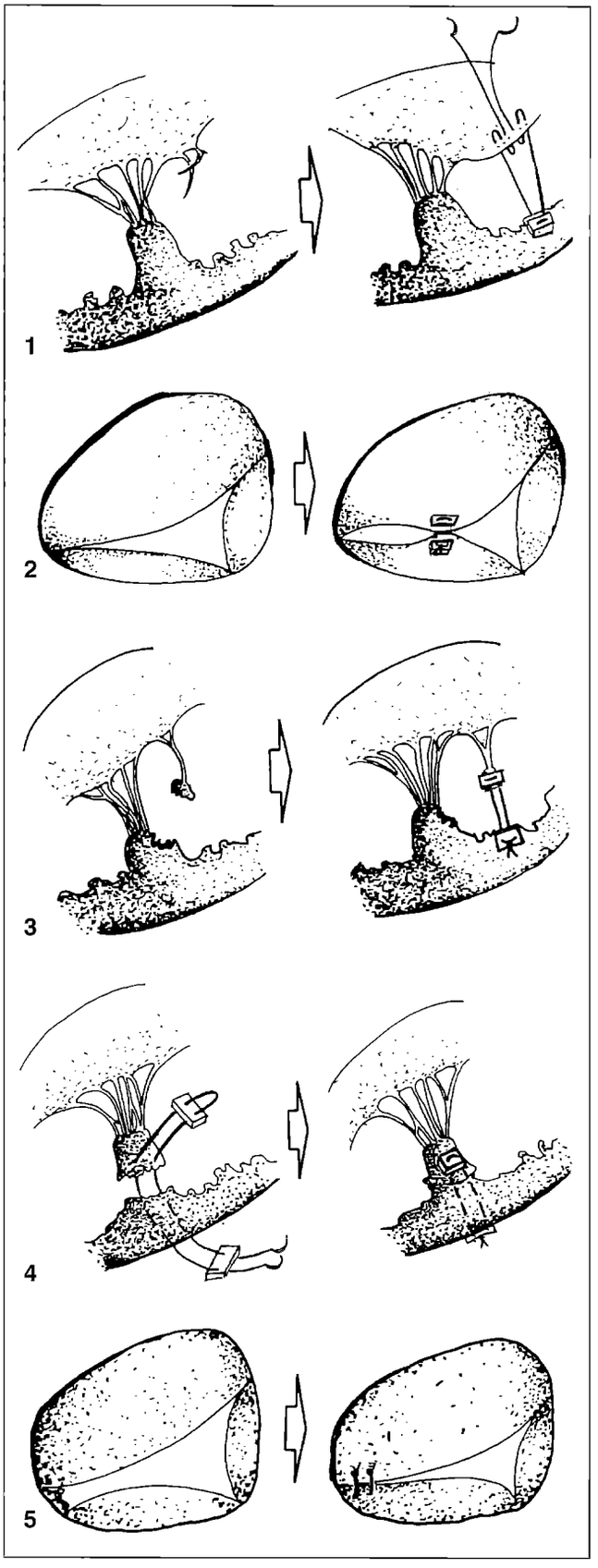
After a median sternotomy and a routine aorto-bicaval cannulation, cardiopulmonary bypass was established at mild hypothermia. The aorta was cross-clamped and cold crystalloid cardioplegic solution was injected into the aortic root. The tricuspid valve was exposed through a conventional oblique right atrial incision. The valve anatomy and mechanism of regurgitation were assessed by injecting cold saline solution into the right ventricular cavity.

In four patients with anterior flail/prolapsing leaflet due to chordal rupture (case no. 2, 3, 5, 6), artificial chordae implant was performed using pledgetted 5-0 expanded-polytetrafluoroethylene (e-PTFE) sutures. In three patients, the chordae were attached to the right ventricular free wall (Fig. 1), while in one they were secured to the anterior papillary muscle. In one patient (case no. 6), who presented with rupture of multiple chordae producing a marked prolapse of the anterior leaflet, after a single chorda implant, an artificial double orifice tricuspid valve was created to correct the prolapse of the leaflet. This was accomplished by anchoring the free edge of the anterior leaflet to the facing edge of the septal leaflet with a pledgetted mattress suture of 5-0 polypropylene (Fig. 2). In case no. 4, the regurgitation was secondary to the rupture of one of the heads of the anterior papillary muscle connected to a fan of chordae supporting the posterior half of the anterior leaflet in the region adjacent to the anteroposterior commissure. This head of the papillary muscle was reconnected to the right ventricular free wall with the interposition of a 5-0 e-PTFE suture (Fig. 3). In case no. 1, the anterior papillary muscle was completely detached at the base resulting in prolapse of the anterior and posterior leaflets. The papillary muscle was reimplanted in the right ventricular free wall with a transfixed mattress suture of 3-0 polypropylene with pledgets (Fig. 4). Thereafter, the valve was tested with saline and significant residual regurgitation was noted, primarily in the area of the anteroseptal commissure. This was partially obliterated with two interrupted 6-0 polypropylene sutures to improve the competence of the valve (Fig. 5). In 5 cases (no. 1–4, 6) a 36 mm Carpentier tricuspid ring was implanted to correct the annular dilatation and deformation, while in case no. 5 the dilatation was moderate and was corrected with a Bex posterior annuloplasty [3]. In the same patient, the ascending aorta was replaced with a Dacron tube graft.

In all cases, following the reconstruction, saline was injected into the right ventricle to test valvular competence. Additionally, during rewarming, the tricuspid valve function was evaluated by transesophageal echocardiography.

Table 1 Clinical and echocardiographic data (*AD* annular dilatation, *ALP* anterior leaflet prolapse, *PLP* posterior leaflet prolapse, *RVEDD* right ventricular end-diastolic diameter, *NYHA* New York Heart Association class, *TR* tricuspid regurgitation, + = moderate, ++ = severe)

Patient	Preoperative data								Follow-up data			
	AD	Chordal rupture	Papillary rupture	ALP	PLP	RVEDD (mm)	NYHA	TR Grade (pre-op)	Months	TR Grade (follow-up)	NYHA	RVEDD (mm)
1	++		anterior	++	++	50	III	4+	48	3+	I	45
2	++	anterior		+		N.A.	I	4+	30	1+	I	40
3	++	anterior		++		65	II	4+	30	1+	I	45
4	++		anterior	+		56	I	4+	27	3+	I	50
5	+	anterior		+		59	III	4+	24	2+	I	42
6	++	anterior		++		30	I	4+	18	0	I	20



Follow-up data

Before discharge, all patients were submitted to control transthoracic echocardiography. No medications were prescribed except for oral anticoagulation which was taken for 3 months after the repair. At follow-up, all patients were submitted to echocardiographic study.

Statistical analysis

The results are expressed as the means ± 1 standard deviation. Statistical significance was obtained by chi-square test for categorical, and by two-tailed paired *t*-test for continuous, variables; a $P \leq 0.01$ was considered statistically significant.

Results

Intraoperative direct inspection of the tricuspid valve and assessment of the mechanism of regurgitation confirmed the preoperative echocardiographic findings. Cardiopulmonary bypass time was 64.8 ± 15.4 min; ischemic time was 34.7 ± 12.7 min. Intraoperative echocardiography showed satisfactory results of the correction in all patients (regurgitation less than 2/4 and no transvalvar pressure gradient). All patients survived the operation and were discharged from our institution within 6 days after uneventful postoperative courses.

The mean follow-up time was 29.5 ± 10.1 months (range 18–48 months). The preoperative and follow-up echocardiographic findings and clinical data are shown in Table 1. At the latest follow-ups, all patients were asymptomatic in NYHA class I without medical therapy. No complications have been recorded during the study period and all patients have returned to their preoperative occupation. Echocardiography showed that all patients had reduction of the tricuspid regurgitation grade and a significant reduction of the right ventricular end-diastolic dimension (RVEDD decreased from a preoperative mean value of 52.0 ± 13.4 mm to a postoperative one of 40.4 ± 11.7 mm; $P = 0.01$). No transvalvular gradient was revealed in the one patient who had "double orifice" repair.

Fig. 1 Artificial chordae implant

Fig. 2 Artificial "double orifice valve"

Fig. 3 Interposition technique: the papillary muscle is reconnected to the right ventricular wall with the interposition of an artificial chorda

Fig. 4 Papillary muscle reimplant with a transfixed 3-0 polypropylene stitch with pledgets

Fig. 5 Partial commissuroplasty

Table 2 Anatomical findings in the cases in the literature and incidence of repair according to the type of lesion. Numbers are expressed as: total lesions/lesions repaired (percentage of repairs). The numbers reported in this table refer to the incidence of the specific lesions, the site of occurrence and the probability of valve repair for the individual lesion according to the literature. More than one lesion was present in many patients, so that the sum of the single lesions are greater than the total number of patients

	Leaflet rupture	Chordal rupture	Papillary rupture
Anterior	6/4 (66.7%)	31/14 (45.2%)	16/6 (37.5%)
Posterior	3/1 (33.3%)	4/2 (50.0%)	8/5 (62.5%)
Septal	4/2 (50.0)	4/0 (0.0%)	1/1 (100%)
Not specified	1	7	0

Discussion

Williams first reported a case of traumatic tricuspid regurgitation following non-penetrating chest trauma in 1829 [41]. The first surgical correction of post-traumatic tricuspid regurgitation was performed by Cooley and published in 1958 [29]. He reimplanted a ruptured papillary muscle in the right ventricular wall. According to the authors, the repair was good but the patient died 12 h later because of hemorrhage. Including the latter, we have found a total of 74 surgical cases of traumatic tricuspid regurgitation secondary to blunt chest trauma in the western literature. Valve repair was accomplished in 30 patients (40.5%), but in the last 10 years 24/47 patients (51.1%) were treated by repair compared with 6/27 patients (28.6%) in the earlier period ($P=0.0150$).

Pathologic anatomy

The feasibility of the repair depends mostly on the pathologic anatomy of the valve and on the mechanism of regurgitation. We analyzed the intraoperative findings of the valves in the cases reported in the literature (Table 2). The anatomical lesions in traumatic tricuspid regurgitation are quite specific. Annular dilatation was always present and it was usually severe. This is consistent with our experience: annular dilatation was severe in five patients and moderate in one (case no. 5). Presumably, the progressive enlargement of the annulus which naturally occurs in this condition was prevented by the early correction (at 3 months).

In the literature, chordal rupture appears to be the most common cause of traumatic tricuspid regurgitation. It was present in 55.4% (41/74 patients) of the reported cases. Anterior chordal rupture was the most frequent mechanism of regurgitation, occurring in 41.9% (31/74 patients) of the cases. This was also the most common finding in our series (4 patients). The other causes of traumatic tricuspid regurgitation reported in the literature are papillary muscle rupture in 27.0% of the cases (20/74 patients) and leaflet rupture in 14.8% (11/74 patients). None of the patients in our series had a leaflet rupture. In the literature, 5% of the cases had only annular dilatation and chordal shortening, which was sometimes associated with leaflet retraction in the absence of evident rupture. In this setting, the diagnosis of traumatic regurgitation is arguable and degenerative disease must be considered. During the study period, we observed a patient with this anatomical pattern (isolated annular dilatation) who was excluded from the study population.

The exact mechanism of traumatic tricuspid rupture after blunt chest trauma is not known. Probably valve rupture is secondary to compression of the heart between the sternum and the spine with sudden increase of the right ventricular pressure. The lesion is most likely to occur during the isometric systolic phase when the heart is full and the valves closed [26]. Frequently, other trauma-related cardiovascular lesions are associated, such as ventricular septal defect [16] (commonly associated with septal leaflet rupture), aortic hematoma and mitral insufficiency [31]. In our series, one patient had associated aortic lesion and two more patients had extensive laceration of the anterolateral aspect of the pericardium at operation. The latter anomaly has been described in several other reports and it is probably secondary to the trauma, but a congenital agenesis of the pericardium cannot be excluded.

Clinical characteristics

The clinical course of traumatic tricuspid incompetence is often subtle. In our series, the time lapse from trauma to surgery varied from 3 months to 20 years. This characteristic is consistent with the experience of others [6]. Symptoms are often absent, but some patients may become symptomatic early in the course of the clinical history, usually because of associated lesions (e.g. ventricular septal defects, mitral valve rupture, patent foramen ovale). In such cases, surgery may be indicated on an emergency basis. Pasic et al. reported a case requiring emergency surgical correction because of isolated acute massive tricuspid regurgitation due to the rupture of all the papillary muscles of the valve [30]. It has also been reported that patients with papillary muscle rupture present earlier in the clinical course than patients with chordal rupture. This was not the case in our experience, where the anatomical lesion did not correlate with the time of presentation or the severity of tricuspid regurgitation.

Some degree of right bundle branch block was found in all our patients, as has been reported in about 75% of the patients with traumatic tricuspid incompetence [37]. This finding, associated with a history of chest trauma should therefore warrant echocardiographic study. In any case, a history of significant chest trauma alone should be considered an indication for routine echocardiography to exclude

cardiovascular lesions [36]. Echocardiography is not only a valuable diagnostic tool but also provides enough information for the surgical decision-making. In our experience, all patients but one underwent surgery after echocardiography alone and the intraoperative findings matched the preoperative ones exactly. Cardiac catheterization and angiography may be indicated in the presence of associated conditions such as aortic lesions and coronary artery disease.

Surgical options

Depending on the anatomical lesions, several surgical options are available and the optimal technique has to be tailored to the specific patient.

Annular dilatation

The annulus in this condition is always very dilated and distorted. The annular dilatation is likely to be secondary to right ventricular dilatation and contributes to the worsening of the regurgitation. In the case reported by Salzer et al. [33] annular dilatation was the only finding at the time of operation. In such cases the diagnosis of traumatic valvular incompetence may be debatable. In all our cases, an annuloplasty was included in the surgical procedure for two reasons: to correct the dilatation/distortion and to prevent further dilatation in the event of postoperative failure of the reconstruction. We implanted a Carpentier ring in five patients, because we believe that this device satisfies these requirements with reproducible results. The size of the ring was chosen according to the septal leaflet measurement, as suggested by Carpentier. Some degree of residual central regurgitation was disclosed by the postoperative echocardiogram in all these patients. Probably this suboptimal result could be avoided by implanting rings one size smaller than calculated. A Bex annuloplasty was performed in patient no. 5, who had only moderate annular dilatation, and the mid-term result of the repair was comparable to those obtained with prosthetic rings.

Leaflet lesions

Leaflet lesions are not common in traumatic tricuspid regurgitation but, if present, they can usually be repaired. In the literature, 6/11 (54.5%) leaflet lesions were repaired by direct suture of the tear. This is the highest rate of repair compared to replacement among the various groups of lesions.

Chordal lesions

On the other hand, chordal lesions are more difficult to repair. Among 49 patients with such lesions, these were repaired in 19 patients (46.3%) representing the lowest rate of repair compared to replacement in the literature. Artificial chordal implant is an appealing technique in cases of chordal rupture and it has already been used by others for tricuspid regurgitation [5]. Katz et al. [18] used pericardial strips to replace ruptured chordae of the anterior leaflet in one patient.

We used 5-0 e-PTFE sutures with a pledget on the ventricular side. When the flail segment is short, the repair is easily accomplished with a single chordal implant, as was the case with three of our patients with chordal rupture. In the fourth patient, the anterior leaflet was completely lacking chordal attachment. The correction of the severe prolapse consisted of a single artificial chorda implant and creation of an artificial "double orifice" tricuspid valve. The alternative option for this particular lesion would have been multiple artificial chordae implant. With this original technique, which we had already applied for mitral valve repair in an extensive series of cases with good results [12], echocardiography confirmed that valvular competence was restored and the repair was non-restrictive. We therefore regard the creation of a double orifice tricuspid valve a convenient option if there is complete detachment of the chordal apparatus of the anterior leaflet.

Other techniques are available to correct flail leaflets. Others have reported quadrangular resection of the flail segment of the leaflet accompanied by a selective annuloplasty [1]. This procedure is feasible in cases of chordal rupture of the anterior or posterior leaflet, while it is not an option in the rare cases of septal chordae involvement, because the septal annulus cannot be shortened. The rationale for quadrangular resection of the anterior leaflet is based on the concept, supported by anatomical studies, that the tricuspid valve is "bicuspid" and the anterior and posterior leaflets have to be considered as two scallops of a single mural leaflet which is the counterpart of the posterior leaflet of the mitral valve [39]. This concept justifies the surgeon in applying methods available for the repair of the mitral posterior leaflet to surgical correction of lesions of the mural leaflets of the tricuspid valve. Another option, if there is isolated rupture of a chorda related to the posterior leaflet, is to implant a Carpentier ring or to perform any other method of bicuspidalization without addressing the chordal lesion directly. Chordae transposition has also been recommended in cases of chordal rupture, but this technique is more demanding and, at times, not suitable because of the flimsy nature of the tricuspid leaflets and subvalvar apparatus [14].

Papillary muscle lesions

Papillary muscle lesions are common in traumatic tricuspid regurgitation. The papillary muscles may be completely transected at the base, but sometimes only one of the heads may be torn, producing a less important prolapse of the leaflet. In our series, one patient (no. 1) had com-

plete rupture of the anterior papillary muscle. This was reimplanted to the ventricular wall with the technique originally described by Osborn [28]. After the repair, a partial anteroseptal commissuroplasty was necessary. The morphology of the commissure was that of an accessory cleft (clefts are commonly present in the anterior and posterior leaflets of the tricuspid valve). Another patient (no. 4) had a ruptured head of the anterior papillary muscle with prolapsing/flail posterior half of the anterior leaflet. The interpretation of this pathologic finding is debatable. The original site of implant on the anterior papillary muscle could not be identified with certainty, therefore this could also be classified as a rupture of an accessory anterolateral papillary muscle. Accessory papillary muscles are very common in the tricuspid valve and they usually support anomalous clefts [39]. The partially ruptured papillary muscle was reconnected to the right ventricular free wall with the interposition of a 5-0 e-PTFE suture. In the literature, papillary muscle reimplant was accomplished in 9/20 patients (45.0%); repairs are more frequent after posterior papillary muscle rupture (5/8 cases, 62.5%) than after anterior papillary muscle rupture (6/16 cases, 37.5%). Nevertheless, a word of caution is necessary: in our experience, both patients with ruptured papillary muscle have residual moderate regurgitation at follow-up, even though they remain completely asymptomatic. On the other hand, all other patients have mild or trivial regurgitation at follow-up. The series is too small to derive absolute conclusions, but in our experience the results of repair are better in the absence of papillary muscle rupture. There are at least two explanations for this finding. The good result of papillary muscle rupture repair depends very much on the correct site of reimplantation. Unfortunately, at least in our experience, the landmarks of the correct position of the papillary muscle within the right ventricular cavity are usually not clear. A minimal displacement can cause significant distortion of the valve and consequent regurgitation. This probably occurred in our two cases with papillary muscle rupture, whereas in our four cases of chordal rupture, the leaflet alignment and hemodynamic results were superior.

In conclusion, despite some evidence of residual tricuspid incompetence, all our patients are presently asymptomatic, back to their preoperative occupation and without any medication. All our patients have remained free from any morbid event since the repair of their tricuspid valve lesions. At the latest follow-ups, tricuspid regurgitation and right ventricular volumes were considerably reduced in all patients as compared to their preoperative values and had not progressed with time. Isolated mild or moderate tricuspid regurgitation can be well tolerated in the absence of left heart disease or pulmonary hypertension. The presence of a prosthetic ring prevents further annular enlargement. It is noteworthy that trivial and mild degrees of regurgitation have been detected by echocardiography in healthy volunteers [17].

The results of our study compare favorably with those reported after tricuspid valve replacement. In the tricuspid position both mechanical and biological prostheses have been proven to provide less than satisfactory results with a high incidence of complications [23]. In the literature, the number of mechanical valve implants for traumatic tricuspid incompetence is similar to the bioprostheses used, with a more recent trend in favor of tissue valves. Unfortunately, follow-up results of tricuspid valve replacement in the particular setting of traumatic tricuspid regurgitation are not available. Nevertheless, considering that the patients with post-traumatic tricuspid regurgitation are usually young and active, valve repair seems to be the best option. It avoids prosthesis-related complications and the need for long-term anticoagulation. Longer follow-up is necessary to evaluate the durability of the repair, particularly in the cases with suboptimal hemodynamic results. However, our results and other's experience show that valve reconstruction is a feasible option for the management of post-traumatic tricuspid regurgitation, even in the presence of complex lesions.

Acknowledgements We are grateful to Alvaro Santos for the illustrations, to Tej Kaul, Lesley Early and Bill Dotson Smith for their precious friendship and editorial assistance.

References

1. Allen MD, Slachman F, Eddy AC, Cohen D, Otto CM, Pearlman AS (1991) Tricuspid valve repair for tricuspid valve endocarditis: tricuspid valve "recycling". Ann Thorac Surg 51:593–598
2. Bayezid O, Mete A, Turkay C, Yanat F, Deger N, Isin E (1993) Traumatic tricuspid insufficiency following blunt chest trauma. J Cardiovasc Surg (Torino) 34:69–71
3. Bex JP, Hazan E, Neveux JY, Mathey J (1976) Annuloplastie mitrale et tricuspidienne sur reducteur lineaire souple. Nouv Presse Med 17:1141–1142
4. Bjork VO (1965) Traumatic rupture of the tricuspid valves. Thoraxchirurgie 12:368–372
5. Bortolotti U, Tursi V, Fasoli G, Milano A, Frigato N, Casarotto D (1993) Tricuspid valve endocarditis: repair with the use of artificial chordae. J Heart Valve Dis 2:567–570
6. Brandeburg RO, McGoon DC, Campeau L, Giuliani ER (1966) Traumatic rupture of the chordae tendinae of the tricuspid valve. Successful repair twenty-two years later. Am J Cardiol 18:911–915
7. Cahill NS, Beller BM, Linhard JW (1972) Isolated traumatic tricuspid regurgitation. Prolonged survival without operative intervention. Chest 61:689–672
8. Chares M, Lamm P, Leischik R, Lenz G, Steinmann EH, Polonius MJ (1993) Highly acute course of ruptured papillary muscle of the tricuspid valve in a case of blunt chest trauma. Thorac Cardiovasc Surg 41:325–327
9. Croxon MS, O'Brien KP, Lowe JB (1971) Traumatic tricuspid regurgitation. Long-term survival. Br Heart J 33:750–755

10. Dontigny L, Baillot R, Panneton J, Page P, Cossette R (1992) Surgical repair of traumatic tricuspid insufficiency: report of three cases. J Trauma 33:266–269
11. Fesani F, Barboso G, Medici D (1975) Trattamento chirurgico della insufficienza tricuspidale post-traumatica isolata. Minerva Cardioangiol 23:98–100
12. Fucci C, Sandrelli L, Pardini A, Torracca L, Ferrari M, Alfieri O (1995) Improved results with mitral valve repair using new surgical techniques. Eur J Cardiothorac Surg 9:621–627
13. Giorgi C, Lipp B, Mathieu P (1979) Les insuffisances tricuspidiennes traumatiques. A propos de trois cas. Ann Chir 33:587–590
14. Heulin A, Morin P, Baybion N, Le Feuvre C, Le Pailleur C, Vacheron A (1991) L'insuffisance tricuspidienne d'origine traumatique. Ann Med Interne (Paris) 141:285–286
15. Horeau J, Nicholas G, Moinard P, Bouhour L (1967) Insuffisance tricuspidienne traumatique a l'evolution prolongée. Arch Mal Coeur Vaiss 60:423–434
16. Jebara VA, Acar C, Dervanian P, Farge A, Sousa Uva M, Julia P, Fabiani JN, Deloche A, Blondeau P, Carpentier A (1992) Traumatic ventricular septal defects. Report of 3 cases with tricuspid valve rupture in 2 cases. J Cardiovasc Surg (Torino) 33:253–255
17. Jobic Y, Slama M, Tribouilloy C, Lan Cheong Wah L, Choquet D, Boshat J, Penther P, Lesbre JP (1993) Doppler echocardiographic evaluation of valve regurgitation in healthy volunteers. Br Heart J 69:109–113
18. Katz NM, Pallas RS (1986) Traumatic rupture of the tricuspid valve. Repair by chordal replacements and annuloplasty. J Thorac Cardiovasc Surg 91:310–313
19. Kleikamp G, Shnepper U, Korte H, Breymann T, Korfer R (1992) Tricuspid valve regurgitation following blunt chest trauma. Chest 102:1294–1296
20. Kremer R, Chalant C, Hekster C, Timmermans G, Gielen A, Lavenne P (1967) Insuffisance tricuspidienne et communication interventriculaire dues a un traumatisme ferme du thorax, correction chirurgicale. Acta Cardiol 22:381–395
21. Madoff IM, Desforges G (1972) Cardiac injuries due to nonpenetrating thoracic trauma. Ann Thorac Surg 14:504–512
22. Marvin RF, Schrank JP, Nolan SP (1973) Traumatic tricuspid insufficiency. Am J Cardiol 32:723–726
23. McGrath LB, Gonzalez Lavin L, Bailey BM, Grunkemeier GL, Fernandez J, Laub GW (1990) Tricuspid valve operations in 530 patients. Twenty-five-year assessment of early and late phase events. J Thorac Cardiovasc Surg 99:124–133
24. Moront M, Lefrak EA, Aki BF (1991) Traumatic rupture of the interventricular septum and tricuspid valve: case report. J Trauma 61:134–136
25. Naccarelli GV, Haisty WK, Kahal FR (1980) Left ventricular to right atrial defect and tricuspid insufficiency secondary to nonpenetrating cardiac trauma. J Trauma 20:887–891
26. Naja I, Pomar JL, Barriuso C, Mestrea C, Mulet J (1992) Traumatic tricuspid regurgitation. J Cardiovasc Surg (Torino) 33:256–257
27. Nataf P, Chanton E, Vernhet JC, Regan M, Chenik S, Dorent R, Jault F, Pavie A, Cabrol C, Gandjbackhch I (1993) Remplacements valvulaires tricuspides chez l'adulte, resultats a distance. Arch Mal Coeur 86:1325–1330
28. Osborn JR, Jones RC, Jahnke EJ Jr (1964) Traumatic tricuspid insufficiency. Hemodynamic data and surgical treatment. Circulation 30:217–221
29. Parmley LF, Manion WC, Mattingly TW (1958) Nonpenetrating traumatic injuries of the heart. Circulation 18:371–396
30. Pasic M, Von Segesser L, Carrel T, Jenni R, Turina M (1992) Severe tricuspid regurgitation following blunt chest trauma: indication for emergency surgery. Eur J Cardiothorac Surg 6:455–457
31. Pellegrini RV, Copeland CE, DiMarco RF, Bekoe S, Grant K, Marrangoni AG, Culig M (1986) Blunt rupture of both atrioventricular valves. Ann Thorac Surg 42:471–472
32. Primm RK, Karp RB, Schrank JP (1979) Multiple cardiovascular injuries and motor vehicle accidents. JAMA 241:2540–2541
33. Salzer J, Weintraub R, Lower R, Eldridge F (1966) Isolated tricuspid insufficiency: report of a case with valve replacement. Am J Cardiol 18:921–927
34. Sareli P, Goldman AP, Pocock WA, Colsen P, Casari A, Barlow JB (1984) Coronary artery-right ventricular fistula and organic tricuspid regurgitation due to blunt chest trauma. Am J Cardiol 54:697–699
35. Shadidnoorai S, Ameli M (1992) Post-traumatic tricuspid insufficiency: when to intervene? J Cardiovasc Surg (Torino) 32:585–588
36. Shapiro MJ, Yanofsky SD, Trapp J, Durham RM, Labovitz A, Sear JE, Barth CW, Pearson AC (1991) Cardiovascular evaluation in blunt thoracic trauma using transesophageal echocardiography (TEE). J Trauma 31:835–840
37. Sheikhzadeh A, Langbehn AF, Ghabusi P, Hakim C, Wendler G, Tarbiat S (1984) Chronic traumatic tricuspid insufficiency. Clin Cardiol 7:299–306
38. Van Son JAM, Danielson GK, Shaff HV, Miller FA (1994) Traumatic tricuspid valve insufficiency. J Thorac Cardiovasc Surg 108:593–598
39. Victor S, Nayak VM (1994) The tricuspid valve is bicuspid. J Heart Valve Dis 3:27–36
40. Vijayanagar R, Bognolo DA, Harrison E, Raju BM, Eckstein PF, Sbar S (1979) Isolated chronic tricuspid insufficiency due to closed chest trauma: report of a case with long-term follow-up. Clin Cardiol 2:364–367
41. Williams A (1829) A case of post-traumatic tricuspid insufficiency. London Med Gaz 4:78