Valve Replacement for Tricuspid Stenosis or Insufficiency Associated with Mitral Valvular Disease

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ACQUIRED tricuspid valvular disease of surgical significance is being recognized and treated successfully with increasing frequency in our clinic. Within the last 18 months five patients with tricuspid insufficiency and mitral stenosis had their lesions corrected with the insertion of a ball valve in the tricuspid area and valvuloplasty or replacement of the mitral valve with no mortality and significant clinical improvement in all. The diagnosis and management of severe organic tricuspid valvular disease in the face of advanced mitral valve lesions has presented certain problems that are discussed herein.

Etiology

The etiologic factor in each instance appeared to be rheumatic fever although a positive history was recorded in only two of the five patients.

In chronic rheumatic heart disease 10 to 20 per cent of the hearts examined at necropsy showed evidence of organic tricuspid valvular involvement.¹ Unlike the mitral valve, which ultimately resembles a funnel with a button-hole, stenotic orifice, the tricuspid valve, after chronic rheumatic involvement with fusion of commissures, resembles a diaphragm with a central or eccentric orifice. Such a tricuspid valve functions with varying degrees of stenosis and insufficiency.² A study of necropsy material suggests that insufficiency predominates more frequently than stenosis, while physiologic studies would suggest just the opposite.³

Recognition

Tricuspid incompetence is a relatively common concomitant of advanced rheumatic mitral valvular disease, and at times may dominate the clinical picture. It may result from organic involvement of the valve by the rheumatic process, or from inability of the leaflets to appose completely due to an overdilated right ventricle. When severe, tricuspid involvement is manifest by increased venous pressure, sometimes with markedly pulsatile characteristics, invariably giving rise to hepatomegaly and hepatic dysfunction, ascites, and marked peripheral edema.

Organic tricuspid insufficiency needs to be differentiated from functional tricuspid insufficiency, tricuspid stenosis, and mitral insufficiency. Correction of the factors in the left side of the heart producing right-sided cardiomegaly can be expected to restore normal annulus diameter and tricuspid competency with resultant disappearances of the signs of insufficiency when there is no significant organic disease in the tricuspid valve. Functional tricuspid insufficiency is seldom seen without significantly increased pulmonary vascular...
resistance. Organic tricuspid insufficiency is seldom seen without evidence of tricuspid stenosis.¹ Both stenosis and insufficiency of the tricuspid valve produce right atrial enlargement, increased atrial pressure waves, and systemic venous congestion and edema. The pansystolic murmur which waxes with inspiration is characteristic of tricuspid insufficiency and most helpful in the distinction. The absence of significant regurgitation of radiopaque material into the left atrium at the time of left ventriculogram in a patient with a pansystolic precordial murmur and increased venous pressure further distinguishes tricuspid insufficiency from mitral insufficiency.

The most useful method for final determination of the functional efficiency and the anatomic state of the tricuspid valve of the patient with severe mitral disease is by direct palpation via the finger within the right atrium in the beating heart before initiation of bypass, and by direct vision inspection if any doubt exists. The suspicion of significant multivalvular disease accompanying severe mitral stenosis has been an indication for open mitral commissurotomy³ on our service. The right-sided approach to the mitral valve lends itself admirably to the detection or confirmation and treatment of coexisting tricuspid disease, which occasionally is unsuspected. Our past experience has clearly indicated that closed mitral commissurotomy in patients with significant organic tricuspid involvement may occasionally produce temporary relief of symptoms, but inevitably later this clinical improvement becomes limited or even reversed by the untreated tricuspid lesion. Moreover, our observations suggest that the mortality (early and late) for surgical treatment of mitral valve disease is lessened by coincident surgical correction of both valves when the tricuspid involvement is severe and organic.

Clinical Evaluation and Surgical Management

Preoperative evaluation of each patient has included routine blood and urine tests, renal function studies, and cardiac fluoroscopy. In addition, a kymogram, electrocardiogram, vec-

torcardiogram, and retrograde left ventriculogram and aortogram are performed. Right heart catheterization has proved helpful and provides valuable hemodynamic data for postoperative comparison, but has not been considered essential for diagnosis of tricuspid lesions if there is clear indication of operative need on the basis of mitral valve disease inasmuch as finger palpation or visual inspection at operation is so easily accomplished.

The DeWall-Lillehei oxygenator⁴ with a Zuhdi heat exchanger was used for cardiopulmonary bypass. Moderate hypothermia and the hemofiltration technic are utilized. For adults, the pump was usually primed⁵ with 16 ml/Kg. of body weight of 10 per cent low molecular weight dextran in normal saline⁶ and 200 to 400 ml of 25 per cent mannitol. One milligram of Dibenzyline/Kg. of body weight is given intravenously before perfusion for protection against the deleterious effects of grossly elevated plasma catecholamines.⁶

A right posterolateral thoracotomy through the bed of the fifth rib was used to approach both the mitral and tricuspid valves. The tricuspid valve was routinely palpated through the right atriotomy incision utilized for caval cannulation. The left atrium was then opened just anterior to the right pulmonary veins. The mitral valve may be cautiously visualized, if any doubt exists as to its functional integrity, with the heart beating by keeping a pool of blood over the valve to prevent air embolism and at the same time allowing visualization of its functional efficiency. Once the status of the mitral valve has been ascertained, the heart action is immediately electrically arrested with a 60-cycle alternating-current fibrillator using 2 to 4 volts.⁷ When the mitral valve was stenotic, but with pliable leaflets, it was split by using a Tubbs dilator with the blades directed against the leaflets at right angles to the commissures, following which commissurotomy is completed with a scalpel or the scissors to separate the leaflets out to the annulus. If the mitral valve was insufficient or stenotic with nonpliable leaflets, prosthetic replace-

ment was carried out. When the mitral valve procedure has been completed, the left atrium is allowed to fill with blood and the atriotomy is closed. When the tricuspid valve also has significant impairment, it is exposed via a right atriotomy. If significant insufficiency or stenosis exists, it has been treated by prosthetic valve replacement. Considerable study and clinical experience has convinced us of the futility of valvuloplasty for tricuspid lesions. Prosthetic valve placement is begun by dividing the fused commissures out to the annulus and then reefing or rolling up the scarred leaflets with a continuous over-and-over suture of 2-0 Mersilene interrupted in at least two places (fig. 1) to prevent purse-stringing of the annulus.

The papillary muscle-chordae tendineae-tricuspid annulus relationship has been preserved intact in the belief that this strengthens the force of ventricular contraction and increases the efficiency of right ventricle systolic ejection. A mitral-type ball-valve prosthesis is then seated and anchored into the tricuspid annulus by means of 12 to 16 interrupted 2-0 Mersilene mattress sutures, each reinforced with a short segment of Teflon spaghetti..
tubing placed in such a manner that the Teflon tubing buttresses the mattress suture on the atrial side of the annulus (fig. 1). The free ends of the suture are brought up through the Teflon skirt of the ball valve and are tied when the valve is placed in the annulus, a technic identical to that used in mitral valve replacement\(^9\),\(^10\) (fig. 2). The valve holder with the right atrium is then allowed to fill with blood, and the atriotomy is closed. The combined valve and suture-holding device has been very helpful.\(^11\)

**Case Reports (Table 1)**

**Patient 1**

E. B., a 36-year-old white woman was admitted to this hospital because of progressive shortness of breath on exertion, paroxysmal nocturnal dyspnea, and chronic cough. She had St. Vitus dance at the age of 9 years and rheumatic fever at 12. A heart murmur became audible at 18. Two years later dyspnea was noted and digitalis was prescribed. Frequent episodes of heart failure ensued. At age 31 a closed mitral commissurotomy was planned. At operation mitral insufficiency rather than stenosis was found, and no definitive procedure was performed. At a second operation, 3 months later, with the heart-lung machine, a polyvinyl spindle was placed on the posterior cusp of the mitral valve to correct this insufficiency. When seen 4 years later, the patient manifested severe right and left heart failure. Left heart catheterization revealed pure mitral stenosis. The atra were fibrillating. A grade-IV/VI coarse systolic murmur was heard in the left lateral position. The liver was enlarged. With a clinical diagnosis of mitral stenosis and tricuspid insufficiency, open cardiac surgery was performed again on February 4, 1963. The right atrium was enormously enlarged, the left slightly. The thrill of tricuspid insufficiency could be felt through the right atrial wall. Under total cardiopulmonary bypass, the foramen ovale region of the right atrium was opened. The mitral valve was found to be narrowed to a diameter of 1½ cm. The Tubbs dilator was introduced and expanded to 4 cm. No mitral insufficiency was created. The atrial septal incision was closed after air was displaced from the left atrium. An effort to plicate the tricuspid valve proved unsuccessful. Therefore, a 3M Starr-Edwards ball-valve mitral prosthesis was inserted into the tricuspid annulus. Anticoagulation was instituted early in the postoperative period. The patient was discharged on the twenty-second postoperative day after an uneventful convalescence. Eighteen months after surgery, there had been complete relief of her symptoms.

**Patient 2**

F. Z., a 46-year-old woman, was admitted to this hospital for the first time on January 27, 1964, with the complaint of exertional dyspnea, easy fatigability, and intermittent ankle edema requiring diuretics. The heart was fibrillating. A loud blowing systolic murmur radiating to the left axilla and a soft rumbling diastolic murmur were heard best at the apex. The liver was enlarged. Both legs were edematous from the ankle to the knees. A retrograde left ventriculogram and aortogram demonstrated pure mitral stenosis and a normal aortic valve. A preoperative diagnosis of severe mitral stenosis and severe tricuspid insufficiency was made. On February 7, 1964, the heart was exposed through a right posterolateral incision. Through the greatly enlarged right atrium the thrill of tricuspid insufficiency could be palpated. The tricuspid valve was diffusely scarred with cicatricial retraction of all three leaflets. The mitral valve also was severely stenotic with an orifice of 3 to 4 mm. Under total cardiopulmonary bypass, the right and left atria were opened. The entire interior of the left atrium was lined by a soft clot approximately 2 cm.

### Table 1

<table>
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<th>Patient</th>
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<td>Tricuspid insufficiency</td>
<td>Mitral valvuloplasty</td>
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<td>left atrial clot removal</td>
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<td>Mitral valvuloplasty</td>
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<td>Tricuspid insufficiency</td>
<td>Mitral ball-valve prosthesis</td>
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<td>&amp; tricuspid stenosis</td>
<td>with left atrial clot removal</td>
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<td>22</td>
<td>Tricuspid insufficiency</td>
<td>Mitral valvuloplasty</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&amp; tricuspid stenosis</td>
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*No hospital or late mortality.*

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thick. This clot was carefully removed and the mitral valve opened to 3.2 cm. with a Tubbs dilator plus scalpel incision at the commissures. The heart was re-started, and there was no mitral insufficiency. Then the heart was fibrillated again, and a no.-4M Starr-Edwards ball-valve mitral prosthesis was anchored in the tricuspid area in the usual manner, preserving all chordae and papillary muscles. Heart size was strikingly diminished in the immediate postoperative interval. Anticoagulation was started on the third postoperative day. The patient was discharged after an uncomplicated convalescence.

**Patient 3**

E. D., a 53-year-old woman, was admitted to this hospital with the complaints of progressive exertional dyspnea, easy fatigability, and intermittent pedal edema requiring digitalis and diuretics. At age 17 she had suffered an episode of scarlet fever at which time a murmur was detected. At 37 years of age she developed atrial fibrillation and was digitalized. Five years later, in 1952, a closed mitral commissurotomy was performed with fair clinical improvement. Three years later progressive exertional dyspnea developed together with episodes of heart failure. On this admission the patient was emaciated and dyspneic. The liver was enlarged. The electrocardiogram showed atrial fibrillation and right ventricular hypertrophy. Cardiac catheterization showed mitral stenosis with only minimal insufficiency and severe tricuspid insufficiency together with severe pulmonary hypertension (88/40 mm. Hg) and right ventricular failure. On March 21, 1964, the heart was exposed through a right lateral thoracotomy. The right atrium was greatly enlarged, the inferior vena cava and left atrium less so. At open cardiotomy, the mitral valve was stenotic with a 1.5-cm. orifice. The

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**Figure 3**

Selected serial films from a left ventriculogram study (anteroposterior, above) (lateral, below) in patient 4 preoperatively. (There is only a trace of mitral insufficiency.) Note the classic appearance of the “mitral stenosis sign” consisting of a filling defect in all films both anteroposterior and lateral, throughout both systole and diastole. The presence of this sign is a very reliable indication of a stenotic valve with rigid scar or calcium and is a definite indication for open operation. In patients with mitral stenosis and pliable leaflets, this sign is usually not seen.
tricuspid orifice was markedly enlarged with the valve practically prelapping into the right atrium with what appeared to be ruptured chordae tendineae. The valve tissue per se appeared normal. With the patient on total cardiopulmonary bypass, the heart was electrically fibrillated. Through a left atriotomy the mitral valve was inspected and mitral commissurotomy was performed to 2.5 cm. diameter with a Tubbs dilator. One annuloplasty stitch was placed in the mural leaflet near the anterior commissure to control the minimal insufficiency. The tricuspid valve was inspected through the right atrium. A no.-4 Starr-Edwards ball-valve mitral prosthesis was anchored at the annulus in the usual manner. No thrill was felt in the left atrium. The patient tolerated the procedure well, but required respiratory assistance by means of a tracheostomy and respirator. When seen 6 months later, she was found to have a great increase in exercise tolerance.

**Patient 4**

J. M., a 30-year-old woman, was admitted complaining of gradually increasing exertional dyspnea, easy fatigability, and frequent coughing requiring diuretics and digitalis. There was no history of rheumatic fever. The patient was emaciated and without dyspnea only at rest. A grade-III/VI systolic murmur was heard at the apex. A snapping mitral first heart sound was heard. There were marked hepatomegaly and ankle edema. The urinalysis was positive for albumin. The electrocardiogram indicated atrial fibrillation, right axis deviation, and possible right ventricular hypertrophy. A retrograde aortogram and ventriculogram demonstrated the classical mitral stenosis sign (fig. 3), mild aortic insufficiency and no mitral insufficiency. The cardiomegaly was due primarily to right ventricular enlargement. On April 6, 1964, the heart was exposed through a right posterolateral incision. A distinct thrill was palpable in the right atrium. After cardiopulmonary bypass was established, the tricuspid valve was seen to be stenotic and insufficient due to diffuse cicatrical retraction. The orifice was shrunken to only 1½ fingerbreadths in diameter. The mitral valve showed severe stenosis with an orifice diameter of only 3 mm. (fig. 4A). Owing to retraction of the mitral leaflets with a severe subvalvular component resulting from fusion and obliteration of the chordae tendineae, the mitral leaflets were attached directly to the papillary muscles. The entire left atrium was lined with a large amount of soft clot. This was removed (fig. 4B) and the atrial appendage sewed off from the inside of the heart. The mitral valve was opened with a Tubbs dilator, but it was found that it could function at best very poorly because of the fusion of the chordae tendineae. Therefore, the anterior and part of the posterior leaflets were excised and the remaining posterior leaflet was reefed and a no.-4 Starr-Edwards ball-valve mitral prosthesis was anchored to the annulus in the usual manner. The tricuspid valve was incised at its commissures and reefed up. A 4M mitral ball-valve was seated down and anchored to the annulus. Large papillary muscles running to the anterior leaflets looked as if they might interfere with the valve function and were excised. The heart took over well, although it remained in atrial fibrillation. Anticoagulation was instituted on the fourth postoperative day. The patient was discharged in good condition (fig. 5).

**Patient 5**

M. K., a 21-year-old woman, was admitted to
Figure 5

Postoperative roentgenogram in patient 4, 1 month after mitral and tricuspid replacement (both 4M) with removal of left atrial clots.

The hospital complaining of excessive tiredness, shortness of breath, and palpitation on exertion. At 10, she had had an illness suggestive of rheumatic fever. The heart was enlarged. A blowing holosystolic murmur, grade III/VI, was heard over the entire precordium with maximal intensity at the tricuspid area. A grade-IV/VI mid-diastolic murmur was audible at the apex. An opening snap could be heard. The liver was not enlarged. The electrocardiogram showed P mitrale and P pulmonale. The vectorcardiogram indicated right ventricular hypertrophy. The right ventricle and left atrium were enlarged. Left heart catheterization revealed severe mitral stenosis without insufficiency and a trace of aortic insufficiency without stenosis. On May 14, 1964, the patient was operated upon with a diagnosis of severe tricuspid stenosis and insufficiency and severe mitral stenosis. The heart was exposed through a right posterolateral thoracotomy. There was only a trace of aortic insufficiency. Both atria were markedly distended. There were cicatricial retraction and severe scarring of all three leaflets of the tricuspid valve. Mitral stenosis was extremely severe, with a central opening of 4 to 5 mm. diameter. After establishing total cardiopulmonary bypass, the mitral valve was observed through a left atriotomy with the heart beating. The heart was then electrically fibrillated. The mitral valve was opened to a 3.8-cm. diameter with a Tubbs dilator. The opening was completed by incising both anterior and posterior commissures with the scissors. There was only slight mitral insufficiency at the posterior commissure after valvuloplasty. The three commissures of the tricuspid valve were incised out to the annulus. The leaflets then were reefed with an over-and-over suture of 2-0 Mersilene carried around the circumference and in-
TRICUSPID VALVE REPLACEMENT

terrupted at two points. A no.-4 ball-valve mitral prosthesis of the Starr-Edwards type was then seated in the tricuspid area in the usual manner. Both atria were closed after evacuating all air, and the heart took over well after two direct current shocks at 50 watt seconds. The postoperative period was uneventful. The patient was discharged on the twentieth postoperative day on a regimen of digitoxin, and Coumadin, the latter having been started on the fourth postoperative day.

Discussion

Relatively little mention has been given in the literature to the surgical treatment of organic tricuspid stenosis and insufficiency. The fact that five cases of this condition occurred during an 18-month period on one service indicates that the lesion is by no means rare. It is our belief that cases of significant organic tricuspid insufficiency associated with mitral or other valvular disease of rheumatic origin are being overlooked or are being left untreated on the assumption that they are always functional and will regress with surgical treatment of only the mitral valve lesions.

It is presumed that all of our cases resulted from rheumatic fever although only two of five patients gave a history of the disease. The reported higher incidence among women of tricuspid disease has been borne out in our series. All had tricuspid insufficiency and mitral valve disease. In addition, two had associated tricuspid stenosis, four had relatively pure mitral stenosis, and one patient had mitral insufficiency. Two patients had very mild aortic insufficiency. Four had atrial fibrillation and four had diastolic murmurs. Clinically tricuspid disease was suggested by the following: systolic murmurs without evidence of aortic stenosis or significant mitral insufficiency, pronounced hepatomegaly, peripheral edema, and ascites.

In our experience the cicatricial scarring and retraction of the leaflets in the tricuspid valve appear to preclude a satisfactory valvuloplasty in most all cases of tricuspid insufficiency. In the one instance in this series when it was attempted, it proved futile. Therefore, we have utilized a ball-valve mitral prosthesis for tricuspid replacement in all five of these patients. In three the tricuspid valves and chordae were left completely intact. In another, part of the remaining valve tissue was resected. In one patient the tricuspid valve with multiple ruptured chordae was completely excised. Open mitral commissurotomy with the Tubbs dilator and scalpel produced satisfactory valvuloplasties in four patients. One patient with mitral stenosis and cicatrical destruction required a ball-valve prosthesis replacement (figs. 4 and 5). There have been no operative, postoperative, or late deaths in the follow-up period, which now ranges from 17 to 31 months. All patients have had their functional capacity significantly improved. One patient (case 1) has returned for postoperative catheterization studies and clinical improvement was confirmed by hemodynamic measurements.10

Summary and Conclusions

Severe organic tricuspid insufficiency or stenosis often accompanies mitral insufficiency or stenosis in chronic rheumatic heart disease.

The presence of severe mitral valve diseases makes the clinical diagnosis of organic tricuspid insufficiency or stenosis somewhat more difficult, as the mitral disease may cause symptoms that predominate in the clinical picture and may lead to functional tricuspid insufficiency.

The diagnosis of organic tricuspid stenosis or insufficiency, or both, has been made on the basis of clinical, X-ray, and, if necessary, by right-sided heart catheterization findings preoperatively. At operation the diagnosis can be confirmed easily and with certainty by palpation or direct vision inspection at the time of open cardiotomy for correction of the mitral valve lesions.

Five patients having severe mitral valve disease with significant organic tricuspid stenosis or insufficiency were treated successfully without hospital or late mortality. Four had open mitral commissurotomy with a ball-valve prosthesis in the tricuspid valve area. The fifth patient had a ball-valve prosthesis placed in both tricuspid and mitral valve areas.
areas along with removal of extensive left atrial clots. In all valve replacements (with one exception) the chordae tendineae-papillary muscle-annulus continuity was preserved to improve postoperative ventricular function. The valve tissue left in situ was rolled up to the annulus with a running suture before prosthetic placement.

The functional capacity of these five patients has been significantly improved and there have been no late complications in any patients during the follow-up period which now ranges from 17 to 31 months.

Total ball-valve replacement of the organically inadequate tricuspid valve associated with advanced rheumatic mitral valve disease restores the right side of the heart to a more physiologic state, and this may be essential for survival in certain far-advanced cases and most certainly improves the long-term surgical palliation achieved in others.

References
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C. WALTON LILLEHEI, PAUL G. GANNON, MORRIS J. LEVY, RICHARD L. VARCO and YANG WANG

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