Clinical and Hemodynamic Studies of Tricuspid Stenosis

By Paul N. Yu, M.D., Dwight E. Harken, M.D., Frank W. Lovejoy, Jr., M.D., Robert E. Nye, Jr., M.D., and Earle B. Mahoney, M.D.

Five patients with tricuspid stenosis accompanying mitral stenosis were studied. The characteristic features include a diastolic rumbling murmur in the tricuspid area, accentuated by inspiration, prominent "a" wave in the jugular pulse, prominent P wave in the electrocardiogram, absence of physical or electrocardiographic signs of right ventricular hypertrophy, and disproportionate right atrial enlargement by fluoroscopy. The diagnosis may be confirmed by the presence of a significant diastolic pressure gradient across the tricuspid valve. Mitral valvuloplasty alone produces no improvement. The concept of "functional tricuspid stenosis" is introduced and a one-stage operative technic for both mitral and tricuspid valvuloplasty is described.

Tricuspid stenosis rarely exists as a solitary valvular disease. In various autopsy studies it has occurred in 10 to 15 per cent of patients with chronic rheumatic valvular disease.\(^1\)\(^2\) It is most frequently associated with combined aortic and mitral valvular lesions.\(^1\)\(^2\) Mitral stenosis without aortic valve disease is complicated by tricuspid stenosis in about 6 per cent of the instances.\(^3\) The principal symptoms and signs of tricuspid stenosis may be obscured by those of concomitant mitral stenosis, and the diagnosis of the combined lesion is easily missed.

Since scattered case reports have shown that patients with combined mitral and tricuspid stenosis may not improve following mitral valve surgery alone,\(^3\)\(^4\)\(^5\) and since tricuspid stenosis can be surgically corrected, the detection of significant tricuspid stenosis is of paramount importance. This lesion may be strongly suspected from the presence of certain symptoms, signs and changes in the electrocardiograms and roentgenograms. A correct diagnosis may be established by cardiac catheterization and confirmed by exploration of the tricuspid valve.

In the past five years, hemodynamic studies of tricuspid stenosis have been reported in only 10 cases\(^3\)\(^-\)\(^9\) and this condition has not been generally recognized.

During the past three years, we have studied, preoperatively, about 100 patients with mitral stenosis by means of cardiac catheterization. Five of these had significant tricuspid stenosis. The clinical diagnosis was made before catheterization in 4 cases and confirmed by operation. The diagnosis in one case was made only in retrospect, during a review of catheterization data, and was subsequently confirmed by autopsy.

The purpose of this paper is (1) to report the results of the clinical and hemodynamic studies of these 5 cases, (2) to emphasize the diagnostic criteria of this condition, (3) to stress the importance and feasibility of tricuspid valvuloplasty in patients with combined mitral and tricuspid stenosis and (4) to introduce a one stage operative technic for both mitral and tricuspid valvuloplasty.

Case Reports

Case 1. M. N. This 31 year old housewife had chorea at age 10. She first developed exertional dyspnea, fatigue, ankle edema and high blood pressure at age 27, during pregnancy. Following...
delivery, the edema subsided and the patient felt quite well for about a year. At age 28, she had acute rheumatic fever and was bedridden for about two months. Subsequently, she had progressive exertional dyspnea, lassitude, and intermittent ankle edema. After an episode of acute pulmonary edema the following year, she was digitalized and required intermittent mercurials to keep her edema-free. Several months later, she had another episode of pulmonary edema followed by “pneumonia” and severe hemoptysis, which required three weeks of hospitalization.

On Dec. 2, 1952 physical examination revealed a pallid, cyanotic woman, who was not orthopneic or dyspneic at rest. The cervical veins were slightly distended without intrinsic venous pulsation. The lungs were clear. The apical heart rate was 108 per minute and the rhythm was regular. The first mitral sound and second pulmonic sounds were accentuated. In the mitral area were heard a grade 2 systolic murmur and a rumbling presystolic murmur. Blowing systolic and early diastolic murmurs were heard along the left sternal border but were also readily heard at the right sternal border. Along either border of the sternum an additional murmur was heard in middiastole. The blood pressure was 140/95 and the liver was just palpable.

A 12 lead electrocardiogram (fig. 1) showed right axis deviation and high peaked P waves indicating right atrial hypertrophy, but no evidence of either right or left ventricular hypertrophy.

Fluoroscopy and roentgenograms revealed generalized cardiomegaly with clear lung fields. The left ventricle overlapped the vertebral column by 2 cm. in the left anterior oblique view. The right ventricle was considerably enlarged, there was also some enlargement of both atria, the right more so than the left.

On Dec. 16, 1952, cardiac catheterization was performed. Blood-gas analysis failed to disclose left-to-right shunting but indicated mild arterial oxygen unsaturation at rest. Following exercise, the arterial blood became normally saturated. The cardiac output and A-V oxygen difference were within normal limits. There was a definite diastolic pressure gradient across the tricuspid valve which was not appreciated at this time (fig. 2). During a review of the intracardiac tracings, a diagnosis of both mitral and tricuspid stenosis was suspected.

Mitral valve surgery was advised but the patient refused. She became steadily worse and was admitted to another hospital where she died on Jan. 22,
The pertinent findings on postmortem examination were as follows: There was a bilateral hydrothorax. The heart weighed 400 Gms. and was hourglass in shape, due to enlargement of both atria and ventricles and constriction around the atrioventricular ring. The aortic valve was mildly stenotic. The mitral valve was heavily calcified and fixed, and would not admit the fingertip. The tricuspid valve was deformed and narrowed, and the chordae tendineae were shortened and thickened. There were no endocardial vegetations. Both ventricles were only moderately hypertrophied. The left atrium, pulmonary veins, pulmonary arteries and right ventricle were moderately dilated. The right atrium and venae cavae were greatly dilated. The myocardium and coronary arteries appeared normal. The lungs and liver were passively congested.

Case 2. M. P. A 32 year old housewife, had scarlet fever at age five and pleurisy at age 18, when a heart murmur was heard. At age 20, a diagnosis of mitral stenosis was made. When she was 25 years old, she had an episode of hypotension and a diagnosis of pulmonary embolism was entertained. Her activities were unrestricted until Oct. 1953, when she began to notice exertional dyspnea, swelling of her ankles and pulsation of the neck vessels. In Nov. 1953, she was hospitalized and treated with digoxin and a low salt diet. Her exertional dyspnea became less marked and ankle edema completely subsided. She was then maintained on digitalis therapy.

On Jan. 4, 1954 she was well-nourished, although she looked chronically ill. There was no cyanosis, clubbing of the digits or edema of the legs. The jugular veins were moderately distended. The heart was enlarged to both sides, the rate was 70 per minute, and the rhythm was slightly irregular. The first mitral sound was somewhat accentuated. A grade 3 rumbling diastolic murmur was heard in the mitral area as well as in the lower sternal region. In the tricuspid area the diastolic murmur was intensified by deep inspiration. A grade 2 systolic murmur was heard in the pulmonic area. The second pulmonic sound was louder than the second aortic sound. The blood pressure was 102/70.

A 12 lead electrocardiogram showed atrial flutter with varying A-V block. In lead V1 the S wave was decreased in amplitude and the R/S approached 1.

Fluoroscopy and roentgenograms of the chest (fig. 3) disclosed a bilaterally enlarged cardiac silhouette. The pulmonary artery and its branches were enlarged. There was definite enlargement of the right ventricle and only slight enlargement of the right atrium. The left atrium appeared normal.

Cardiac catheterization was performed on January 6, 1954. The pertinent data are presented in table 1. On March 2, 1954, mitral and tricuspid valvuloplasty was performed in one stage by a new method.11

During the 19 months that have elapsed since surgery, the patient has been free of cardiac symptoms upon ordinary exertion. She takes no medication other than oral prophylactic penicillin. The diastolic murmurs in the mitral and tricuspid areas have diminished in intensity. The heart size is unchanged except for some increase in the size of the left atrial shadow. The electrocardiogram now reveals established atrial fibrillation. Data from postoperative catheterization are given in table 1 and figures 4 and 5.

Fig. 3. Roentgenograms of the chest of 2 patients with tricuspid stenosis, case 2 on the left and case 5 on the right.
### Table 1.—Blood Flow, Pressures, Resistances in Five Patients with Tricuspid Stenosis

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Blood Flow</th>
<th>Blood Pressure (mm. Hg)</th>
<th>Resistance (dynes/sec/cm.²)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cardiac Index (L. M.²/min.)</td>
<td>Stroke Index (ml./M.²/min.)</td>
<td>Pulmonary &quot;Capillary&quot; (m)</td>
</tr>
<tr>
<td>1. M. N., 31, F</td>
<td>R 2.69 28</td>
<td>32</td>
<td>68/37</td>
</tr>
<tr>
<td>BSA = 1.56</td>
<td>E 3.26 25</td>
<td>—</td>
<td>100/58</td>
</tr>
<tr>
<td>2. M. P., 32, F</td>
<td>R 2.10 35</td>
<td>12</td>
<td>27/15</td>
</tr>
<tr>
<td>BSA = Pre-op., 1.64</td>
<td>E — —</td>
<td>—</td>
<td>30/15</td>
</tr>
<tr>
<td>Post-op., 1.64</td>
<td>R 2.75 42</td>
<td>22</td>
<td>39/19</td>
</tr>
<tr>
<td>E — —</td>
<td>—</td>
<td>—</td>
<td>51/24</td>
</tr>
<tr>
<td>3. L. F., 42, F</td>
<td>R — —</td>
<td>15</td>
<td>33/19</td>
</tr>
<tr>
<td>BSA = Preop., 1.53</td>
<td>E — —</td>
<td>—</td>
<td>42/25</td>
</tr>
<tr>
<td>Postop., 1.53</td>
<td>R 2.15 31</td>
<td>13</td>
<td>30/12</td>
</tr>
<tr>
<td>E — —</td>
<td>—</td>
<td>—</td>
<td>38/16</td>
</tr>
<tr>
<td>4. C. B., 46, F</td>
<td>R 3.40 30</td>
<td>26</td>
<td>46/17</td>
</tr>
<tr>
<td>BSA = 1.58</td>
<td>E — —</td>
<td>—</td>
<td>51/21</td>
</tr>
<tr>
<td>5. I. R., 34, F</td>
<td>R 2.42 29</td>
<td>13</td>
<td>24/18</td>
</tr>
<tr>
<td>BSA = 1.54</td>
<td>E 2.97 28</td>
<td>—</td>
<td>28/17</td>
</tr>
</tbody>
</table>

R = at rest.
E = after exercise.
M = mean.
S = systolic.
D or D₂ = end-diastolic.
D₁ = early diastolic.
BSA = body surface area in M.²

**Fig. 4.** Simultaneous right atrial and right ventricular pressure tracings obtained through a double-lumen catheter in a patient (case 2) with tricuspid stenosis and atrial flutter. Note the absence of giant “a” wave in the right atrial pressure and the elevated right ventricular end-diastolic pressure.
CLINICAL AND HEMODYNAMIC STUDIES OF TRICUSPID STENOSIS

CASE 3. L. F. A 42 year old housewife had acute rheumatic fever at age 12 and was found to have a heart murmur at age 22. Five years prior to admission, she developed increasing exertional dyspnea and fatigue to the point of inability to walk more than a block. She also noticed pulsation of the neck vessels and frequent palpitation of the heart.

Physical examination showed dyspnea on slight exertion and stasis cyanosis of the face and ear lobes. The jugular veins were distended and a distinct "a" wave was demonstrable. There were classical signs of mitral stenosis. In addition, a rumbling diastolic murmur, exaggerated by deep inspiration, was audible at the tricuspid area. There was a distinct opening snap at the apex and along the left sternal border. The liver was palpable 2 cm. below the costal margin.

An electrocardiogram showed a tall P wave in leads 2, 3 and V₁ and was suggestive of right ventricular hypertrophy (fig. 1). Radiologic examination showed moderate cardiac enlargement with prominent pulmonary vessels, enlarged right ventricle, slightly enlarged left atrium and moderately enlarged right atrium.

Cardiac catheterization showed a diastolic pressure gradient across the tricuspid valve and slightly elevated pulmonary artery and pulmonary "capillary" pressures (table I). A diagnosis of mitral and tricuspid stenosis was made. Operation was planned in two stages. Mitral valvuloplasty was performed on May 13, 1954. The mitral valve orifice was very small and was estimated to be less than 1 sq. cm. in size. Adequate fracture of the commissures was carried out to about the size of 2 fingerbreadths.

After the operation the patient was not improved, although physical examination showed considerable decrease in the intensity of both the first mitral sound and the rumbling diastolic murmur at the apex. However, the opening snap and the rumbling diastolic murmur along the left sternal border persisted. On July 7, 1954, she developed atrial flutter.

Fig. 5. Right atrial and right ventricular pressure tracings obtained through a double-lumen catheter in a patient (case 2) with tricuspid stenosis before and three months after mitral and tricuspid valvuloplasty. In the preoperative tracings, the right ventricular end-diastolic pressure fell toward normal after exercise. In the postoperative tracings there was no longer any diastolic pressure gradient across the tricuspid valve at rest or after exercise.
with 2:1 A-V block and a ventricular rate of 120 per minute. Digoxin therapy was started and the atrial flutter changed to atrial fibrillation.

Tricuspid valvuloplasty was performed on July 9, 1954. The tricuspid valve orifice was found to be small and the operator could not insert his index finger beyond the first interphalangeal crease. With adequate fracture, the tricuspid valve was considerably enlarged.

Following the second operation, the patient improved steadily and the atrial fibrillation reverted to sinus rhythm. In three months all the physical signs of tricuspid stenosis disappeared and the physical signs of mitral stenosis were minimal. Postoperative cardiac catheterization was performed (table 1). In Oct. 1955, the patient had no cardiac symptoms upon moving the lawn, climbing stairs or walking on level ground.

Case 4. C. B. This 46 year old married housewife complained of severe fatigue of several years' duration, and swelling of the abdomen of three months' duration. When she was nine years old, she had a febrile illness associated with swollen joints. She was well until the age of 37, when she had sudden onset of acute dyspnea and orthopnea necessitating hospitalization and digitalization. At age 41 intermittent ankle edema appeared. She also noted pulsation of the neck vessels and frequent palpitation of heart. For a year before we examined her, symptoms of fatigue and exertional dyspnea became progressively more severe, resulting in almost complete disability with confinement to bed most of the time. Treatment consisted of digoxin, Diamox and salt restriction.

On physical examination, the patient appeared malnourished and chronically ill. The pertinent findings included erythema of the palms, some stasis cyanosis of the face and distended cervical veins with slight pulsation. The left border of the cardiac dullness was in the anterior axillary line. There was a slight right ventricular precordial heave. The heart sounds were of good quality. At the apex, the first mitral sound was moderately accentuated and was followed by a grade 2 blowing systolic murmur. The second apical sound was followed by a rumbling middiastolic murmur of grade 2-3 intensity, which increased during expiration. Over the tricuspid area were a grade 2 systolic murmur and a grade 2 rumbling diastolic murmur, which became louder during full inspiration. Along the left sternal border, from the second to the fourth intercostal spaces, was a grade 1-2, blowing, decrescendo, early diastolic murmur. The second pulmonic sound was slightly accentuated and split. The lungs were clear, the liver was felt about 4 fingerbreadths below the costal margin, and no peripheral edema was demonstrable.

![Phonocardiogram](image)

**Fig. 6.** Phonocardiogram of a patient (case 4) with tricuspid stenosis. The microphone was placed over the tricuspid area. Note the accentuation of the diastolic murmur during inspiration.
A phonocardiogram confirmed the tricuspid diastolic murmur, which was intensified during inspiration (fig. 6). A 12 lead electrocardiogram showed atrial fibrillation, right axis deviation, and abnormal T waves and ST segments in the precordial leads. There was no hypertrophy of either ventricle. Fluoroscopy and roentgenograms of the chest revealed enlargement of both atria, particularly the right, and of the right ventricle, and questionably the left ventricle. The pertinent data of the cardiac catheterization are tabulated in table 1. Cineangiogram showed retention of radiopaque substance in a large right atrium.

On March 1, 1955, a thoracotomy was performed and revealed a fairly adherent pericarditis. The mitral valve was fibrotic, opened quite widely and admitted 2 fingers; there was a definite regurgitant jet. When the right atrium was entered, the tricuspid valve leaflets were found to be thin and the orifice was estimated at 1.5 sq. cm. The fused posterior superior commissure of the valve was fractured, whereupon the valve opened widely. A slight regurgitant jet felt before the fracture was not altered by the procedure. Before the fracture of the tricuspid valve the right atrial mean pressure was 12 mm. Hg; immediately following the fracture it decreased to 6 mm. Hg (fig. 7). The patient’s post-operative course was rather stormy and associated with bilateral hydrothorax necessitating right thoracentesis. There was some increase in her pulmonary congestion or infection. Following digitalis therapy, diuretics and salt restriction, she gradually improved and was discharged after 29 days of hospitalization.

Six months after surgery, she stated that she was less dyspneic on exertion and able to do more housework than previously.

Case 5. I. R. This 34 year old housewife had scarlet fever at age 4. At age 21 a pre-employment examination disclosed cardiomegaly as well as a heart murmur. Her activities were restricted because of one flight dyspnea, fatigue, and mild orthopnea. She had always been subject to frequent respiratory infections, usually associated with wheezing and nonproductive cough. She also noticed moderate pulsation of the neck veins. In October, 1954, digitalis was prescribed, following a respiratory infection associated with severe cough and tachycardia.

In February, 1955, physical examination revealed normal development and nutrition without cyanosis or orthopnea. The cervical veins were slightly distended and there was a distinct "a"
wave. Examination of the heart showed the point of maximum impulse to be in the sixth intercostal space along the left midclavicular line. The first mitral sound was slightly accentuated and the second pulmonic sound was louder than the second aortic sound and was split. A blowing decrescendo, early diastolic murmur of grade 2 intensity, accentuated by inspiration, was heard over the base of the heart but maximal along the left sternal border. In addition, a soft systolic murmur was heard over the base. At the tricuspid area there was a grade 3 rumbling middiastolic murmur, which was intensified by inspiration and decreased during expiration. At the apex, there was a middiastolic rumbling murmur of grade 3 intensity with presystolic accentuation. The lungs were clear to percussion and auscultation. The liver was palpable about 2 fingerbreadths below the costal margin. There was no peripheral edema.

A 12 lead electrocardiogram (fig. 1) showed a rate of 90 and a first degree A-V block. The P waves were very broad, tall and notched in leads 1, 2 and aVF. The electrocardiographic position of the heart was vertical. A high peaked P wave was also present in lead V1. There was no evidence of either right or left ventricular hypertrophy.

Fluoroscopy and roentgenograms of the chest (fig. 3) showed an increased cardiac silhouette with the right heart border projected well to the right of the spine and round in contour. The pulmonary artery segment was normal and the left ventricular border was rounded and prominent. In the right anterior oblique position, both atria appeared enlarged and the pulmonary artery segment was prominent. In the left anterior oblique view, the right ventricular enlargement was quite distinct. On March 22, 1955, cardiac catheterization revealed a diastolic pressure gradient across the tricuspid valve, which was accentuated following exercise (table 1 and fig. 8).

In June, 1955, thomeotomy disclosed moderately severe mitral stenosis for which an adequate valvuloplasty was performed. The tricuspid valve, which would admit no more than the index finger up to the second joint, was considerably enlarged by finger fracture. Mitral valvuloplasty resulted in a fall in left atrial, but no change in right atrial, pressure. Following tricuspid valvuloplasty the right atrial pressure fell. The pulmonary artery pressure changed little throughout. Three months later, the patient claimed marked improvement in exercise tolerance. She was able to do all the housework without symptoms. The cervical veins were no longer distended although a distinct "a" wave was still visible. The size of the heart became smaller and the rumbling diastolic murmur over the tricuspid area was less distinct.
TABLE 2.—Pertinent Symptoms and Important Findings in Five Patients with Tricuspid Stenosis

<table>
<thead>
<tr>
<th>Pertinent Symptoms</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of acute rheumatic fever</td>
<td>3</td>
</tr>
<tr>
<td>History of scarlet fever</td>
<td>2</td>
</tr>
<tr>
<td>Severe exertional dyspnea</td>
<td>5</td>
</tr>
<tr>
<td>Increased fatigue</td>
<td>5</td>
</tr>
<tr>
<td>Marked functional disability</td>
<td>5</td>
</tr>
<tr>
<td>Awareness of pulsations in the neck veins</td>
<td>4</td>
</tr>
<tr>
<td>Swelling of the legs or abdomen</td>
<td>3</td>
</tr>
<tr>
<td>Palpitation of the heart</td>
<td>3</td>
</tr>
</tbody>
</table>

Important Physical Findings

<table>
<thead>
<tr>
<th>Finding</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distended cervical veins</td>
<td>5</td>
</tr>
<tr>
<td>Rumbling diastolic murmur in the tricuspid area (accentuated by inspiration in 4)</td>
<td>5</td>
</tr>
<tr>
<td>Hepatic enlargement</td>
<td>4</td>
</tr>
<tr>
<td>Pulmonic second sound slightly accentuated and split</td>
<td>4</td>
</tr>
<tr>
<td>Stasis cyanosis of the face</td>
<td>3</td>
</tr>
</tbody>
</table>

**Comments**

**A. Clinical Studies**

All these 5 patients were women. Their pertinent symptoms and signs are listed in table 2. Three patients had had acute rheumatic fever and the other 2 had had scarlet fever. All were severely disabled by exertional dyspnea and fatigue, 4 were aware of pulsation in the neck; palpitation and peripheral edema or ascites were present in 3 cases. In agreement with the findings described by McCord and his associates and by Chesterman and Whitaker, the most characteristic physical signs were distended cervical veins and a rumbling diastolic murmur over the tricuspid area. The tricuspid diastolic murmur was intensified during inspiration in 4 patients, in whom the diastolic murmur of mitral stenosis was usually diminished with deep inspiration. The second pulmonic sound was slightly accentuated and split in 4 cases. Even though the patients were greatly disabled, the physical signs of marked pulmonary hypertension were conspicuously absent. Hepatic enlargement was present in 4 patients and 3 had stasis cyanosis of the face.

Electrocardiograms showed sinus rhythm in 3, atrial flutter in 1 and atrial fibrillation in the fifth patient. The P wave was very prominent in all 3 patients with sinus rhythm, particularly in lead V1. The electrocardiographic position of the heart was vertical in all. Suggestive evidence of right ventricular hypertrophy was noted in only 2 patients.

Roentgenograms and fluoroscopy of the chest showed moderate to marked enlargement of the cardiac size in all patients. The enlargement involved right atrium, pulmonary artery, right ventricle and left atrium. In a patient reported by Trace and co-workers, there was no enlargement of the left atrium. A cineangiocardiogram performed in one patient demonstrated prolonged retention of radiopaque substance in a large right atrium.

**B. Hemodynamic Studies**

The hemodynamics in patients with predominant tricuspid stenosis have been admirably described by Ferrer and co-workers. The presence of a significant diastolic pressure gradient across the tricuspid valve is emphasized by most workers; this is accentuated by exercise and is present regardless of the cardiac rhythm. As pointed out by McCord and his associates, the gradient was more marked during the early diastolic period than during the end-diastolic period.

Small diastolic pressure gradients across the tricuspid valve do not necessarily indicate anatomic stenosis. In another patient, not described in this report, the clinical diagnosis of tricuspid insufficiency was substantiated by finding a "ventricular" contour in the right atrial pressure pattern during exercise. There were physical signs of associated tricuspid stenosis and a consistent diastolic pressure gradient of 2 mm Hg across the tricuspid valve at rest. At operation it was thought that the valve was purely insufficient.

The elevation of diastolic pressures in the right ventricle in 3 of our patients, (M. P., L. F., C. B.), before operation is difficult to explain. It is not likely that it was due to right ventricular failure, as a consequence of the associated mitral valve disease, since the right ventricular systolic pressures were only mildly elevated. Furthermore, when one of these 3 patients, (I. R.) was exercised, the right ventricular diastolic pressure fell to normal (fig. 8).

In patients with sinus rhythm a giant "a"
wave, produced by vigorous contractions of the right atrium, was seen in the pressure tracings obtained from the right atrium and superior vena cava (fig. 9). The peaks of atrial systole were very high, around 18 mm. Hg in our 3 cases. At the conclusion of atrial systole, the atrial pressure fell rapidly to the lowest point in the cycle, as other workers have noted. We consider this probably an exaggeration of the "x" descent following vigorous systole in a distended right atrium. It did not occur in our 2 patients with atrial fibrillation or flutter. With the opening of the tricuspid valve the right atrial and vena caval pressures did not fall markedly. Ferrer and her associates suggest that, during early diastole, the volume change in the right atrium is small and large right ventricular filling is not accomplished.

The pulmonary artery and pulmonary "capillary" pressures were normal or slightly elevated in 4 patients at rest and rose slightly with exercise. These were distinctly lower than in patients comparably disabled by predominant mitral stenosis. In patient M. N., both the pulmonary artery and pulmonary "capillary" pressures were much higher than in the other 4 patients. At autopsy, the degree of mitral stenosis was considerably more marked than that of tricuspid stenosis. In our experience as well as in that of McCord and associates, the degree of pulmonary hypertension usually varies inversely with the severity of tricuspid stenosis in a patient with combined mitral and tricuspid stenosis.

The resting cardiac index and stroke index were subnormal in all but one patient. With exercise, cardiac index increased slightly but the stroke index actually decreased, although the total pulmonary resistance was either normal or only slightly increased.

C. Surgical Aspects

Combined mitral and tricuspid valvuloplasty was performed in 3 patients with excellent results. Patient 4, whose mitral valve was predominantly insufficient, showed moderate improvement following tricuspid valvuloplasty alone. The operation was performed in one stage in 3 patients and in two stages in 1 patient.

The advent of surgical intervention in any condition requires reappraisal of that pathologic state. It is not uncommon to find the accepted morphology at variance with new kinetic pathologic concepts. Since the advent of satisfactory surgical correction, the limited opportunity for observing tricuspid stenosis permits only preliminary impressions. It would appear that often the fusion bridge is in the anterior commissure. The longest axis of leaflet contact is here, between the anteromedial and anterolateral cusps. Two general patterns of tricuspid stenosis are commonly palpated at the operating table. The first is a residual posterior transverse slit-like orifice where the medial and lateral cusps fuse. The slit-like orifice is made up of the commissure between the lateral and posterior cusps. Tricuspid stenosis is virtually always associated with mitral valve disease, which produces hypertrophy and dilatation of the right ventricle. With such hypertrophy and dilatation of the right ventricle this transverse fissure is stretched taut to produce much more significant stenosis hemodynamically than one might anticipate in the flaccid heart at post-

---

**Fig. 9.** Blood pressure tracings obtained from superior vena cava in 3 patients with tricuspid stenosis and sinus rhythm. Note the presence of giant "a" wave in each tracing.
postmortem. The conventional postmortem opening of the heart, in the direction of blood flow, divides the annulus and leaflets of the tricuspid valve in such a way that this type of stenosis is difficult to recognize. This type of lesion may be called “functional tricuspid stenosis.” Correction of mitral stenosis alone might so reduce pulmonary hypertension and right ventricular size that the tricuspid annulus could contract and the “functional tricuspid stenosis” might well disappear.

A second form of tricuspid stenosis is more significant and straightforward. It consists of a diaphragm with more or less circular central defect. The stenotic orifice may be as small as 2 sq. cm. This stenotic diaphragm is held as a rigid tambour by its circumferential attachment to the dilated annulus. Enlargement of the annulus is secondary to right ventricular hypertrophy and dilatation, which in turn is secondary to the mitral valve disease. This type of tricuspid stenosis will produce significant clinical and hemodynamic changes and the lesion should be adequately corrected.

A simple technic has been developed for fracturing both mitral and tricuspid valves in one stage through a left thoracic incision.11 The technic may be summarized briefly as follows: The mitral valvuloplasty is completed after opening the pericardium in the usual way posterior to the phrenic nerve, then the pericardium is loosely approximated and incised again obliquely upward over the pulmonary artery from the phrenic nerve in the direction of patient’s right shoulder. The incision is carried over the anterior aspect of the pulmonary conus. Marsupializing sutures are used for traction to rotate and elevate the pericardium gently, from right to left, until the enlarged right atrial appendage appears. With the appendage in the operative field, the usual circumferential purse-string sutures are placed in it and the finger fracture valvuloplasty of the tricuspid stenosis is carried out. The criteria for adequate tricuspid valvuloplasty have not been established, but an orifice of 4 or more sq. cm. would seem to be adequate.

At operation, a decline in the left atrial pressure followed mitral valvuloplasty and a fall in the right atrial pressure followed tricuspid valvuloplasty.

Postoperative cardiac catheterization in 2 patients revealed disappearance of the diastolic pressure gradient across the tricuspid valve, both at rest and after exercise. The pulmonary artery and pulmonary “capillary” pressures rose slightly in one patient and cardiac output increased. The pressures in the pulmonary circuit fell slightly in the other.

Summary

1. Tricuspid stenosis occasionally accompanies mitral stenosis.

2. Distinct findings on physical examination, electrocardiogram and x-ray examination suggest the diagnosis, (1) a diastolic rumbling murmur in the tricuspid area, accentuated by inspiration; (2) prominent “a” wave in the jugular pulse; (3) “P pulmonale” waves in the electrocardiogram; (4) absence of signs of marked pulmonary hypertension or of right ventricular hypertrophy, although the patient is markedly disabled and (5) disproportionate right atrial enlargement by fluoroscopy.

3. The diagnosis is confirmed by a significant diastolic pressure gradient across the tricuspid valve at rest and during exercise.

4. In a patient with combined mitral and tricuspid stenosis, the expected improvement may not occur unless both mitral and tricuspid valves are fractured.

5. The concept of true tricuspid stenosis and “functional tricuspid stenosis” was introduced and a 1-stage operative technic for both mitral and tricuspid valvuloplasty is described.

6. With an increased awareness of its existence, more cases will be correctly diagnosed and benefited by proper surgical treatment.

Appendix

Since the presentation of this paper an excellent paper entitled “The Diagnosis of Tricuspid Stenosis” by Drs. Ronald Gibson and Paul Wood was published in the British Heart Journal 17: 552 (Oct.), 1955. The clinical and hemodynamic features of their 14 cases were very similar to those reported in this paper.

Acknowledgment

We are grateful to Dr. R. Taylor, Auburn, New York, and Dr. J. W. Walker, Geneva, New York, for permission to include the description of the terminal illness and autopsy findings of Case 1(M.N.). We are indebted to Dr. Andrew Dale for permission
to include the third case (L. F.). He performed both mitral and tricuspid valvuloplasty on this patient.

SUMMARIO IN INTERLINGUA

1. Stenosis tricuspid accompania a vices stenosis mitral.

2. Le diagnose se suggere per le sequente distincte constataiones physic, electrocardiographic, e roentgenographic: (1) Un rolante murmure diastolico in le area tricuspid, accentuate per inspiration. (2) Un prominente unda “a” in le pulso jugular. (3) Undas “P pulmonal” in le electrocardiogramma. (4) Absentia de signos de marcate hypertension pulmonar o de hypertrophia dextero-ventricular, in respecto del facto que le patiente es marcate-mente invalidate. E (5) un disproportionate allargamento dextero-atrial, visible per fluoroscopia.

3. Le diagnose se confirma per le constataiones de un significative gradiente de pression diastolico a transverso le valvula tricuspid in stato de reposo e durante exercitio.

4. In un patiente con combine stenosis mitral e tricuspid, le expectate melioration non pote occurrer si non le valvulas tanto mitral como etiam tricuspid es fractusrate.

5. Le concepto de ver stenosis tricuspid e de “functional stenosis tricuspid” eseva introduite. Es descrivite un technica a operation unic pro valvuloplastia mitral e etiam tricuspid.

6. Con le diffusion del information que iste condition existe, plus numerose casos de illo va esser diagnosticate correctemente e va profitar del appropriate tractamento chirurgic.

REFERENCES


Clinical and Hemodynamic Studies of Tricuspid Stenosis
PAUL N. YU, DWIGHT E. HARKEN, FRANK W. LOVEJOY, JR., ROBERT E.
NYE, JR. and EARLE B. MAHONEY

Circulation. 1956;13:680-691
doi: 10.1161/01.CIR.13.5.680

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/13/5/680

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/