Surgical Treatment of Tricuspid Stenosis

By Thomas J. E. O'Neill, M.D., O. Henry Janton, M.D., and Robert P. Glover, M.D.

In patients suffering with chronic valvular disease who are diagnosed as having mitral stenosis, there are found a few who have an associated tricuspid stenosis. The diagnosis of this combination of lesions is difficult. The unique opportunity to study the hemodynamics of "pure" tricuspid stenosis was presented when a patient, originally diagnosed as mitral stenosis, had in fact both lesions, and was treated by mitral commissurotomy. Subsequently tricuspid stenosis was suspected clinically and was demonstrated by serial catheterization studies. Tricuspid commissurotomy was performed four years after mitral commissurotomy.

TRICUSPID valvular disease in adults is usually rheumatic in origin. As seen in the mitral and aortic valves, severe anatomic distortion of the leaflets of the tricuspid valve produces insufficiency, stenosis, or an element of each. Such deformities, when slight, cause no physiologic derangements, but when marked interfere noticeably with cardiovascular function. From a clinical standpoint, the terms aortic and dynamic tricuspid disease seem applicable.

Whereas insufficiency of the tricuspid valve may be primary when due to distorted valve leaflets, or secondary when due to changes incident to severe right heart failure, stenosis is always an organic involvement. The size of the orifice is of considerable importance in that there probably exists a critical area above which insufficiency predominates, and under which, stenosis predominates. This area has not as yet been closely defined and requires further observation.

Rarely does this disease exist as a solitary valvular defect. Reports of autopsy studies demonstrate that its highest incidence is in association with both aortic and mitral disease; and its next highest incidence is in association with mitral valve involvement. A decisive clinical diagnosis usually cannot be made because of the masking effects of the mitral valve lesion. Therefore, the successful surgical treatment for stenotic valvular diseases now imposes a greater demand for more accurate methods of diagnosis of stenosis of the tricuspid valve, since this lesion may no longer be regarded as having merely prognostic importance.

Operative attempts have been made simultaneously on the combination of aortic and mitral stenosis with some success, but it is apparent that both of these valves, located as they are in the left heart, render operative appraisal less difficult and intervention more feasible. The tricuspid valve being on the side opposite requires a more formidable approach for simultaneous surgery, or else necessitates a two stage operation.

The diagnosis of tricuspid stenosis is not easy to make, mainly because of the absence of unambiguous cardinal features. This problem is the greater because the principle signs and symptoms are obscured by the identical findings often seen in solitary mitral stenosis. During life, there are only two methods of differentiation available: One, elimination of the symptoms secondary to mitral stenosis by means of mitral commissurotomy, thereby allowing a residue of provocative symptoms that suggest a dynamic tricuspid stenosis. The other, by certain inductions drawn from a less than impressive set of particulars, which reflect alterations in physiology dictated by something other than mitral stenosis alone. The more important symptoms and signs suggestive of tricuspid stenosis may be listed as follows:

1. History and Cardiac Findings. The patient has auscultatory evidences of mitral stenosis associated with episodic or constant peripheral edema and/or ascites, usually never having suffered the usual pulmonary symptoms secondary to pulmonary hypertension. Such
patients are not ordinarily dyspneic or orthopneic.

2. Venous Pulsations. When seen to be abnormally vigorous in the neck, these suggest tricuspid disease, regardless of whether the pulsations are caused by a regurgitation, or by atrial displacement of an otherwise flexible stenotic tricuspid valve "diaphragm." The repeated references to "liver pulsations" are possibly transmitted cardiac impulses applied to the surface of the liver itself.

3. Cyanosis. With or without jaundice, this would indicate either peripheral stasis or pulmonary stasis and could be a manifestation of mitral stenosis alone.

4. Right Atrial Enlargement. This is present in Lutembacher's syndrome and tricuspid insufficiency, as well as tricuspid stenosis. There could be absence of enlargement if the wall of the atrium is "protected" by mural thrombosis or myofibrosis.

5. Diminished Pulmonary Arterial Dimensions as Seen Radiologically. This change could occur if the tricuspid stenosis matured before the mitral stenosis.

6. Localized Diastolic Murmur at the Lower End of Sternum. There is usually great difficulty in interpreting the significance of the diastolic murmur and rarely is a diagnosis of dynamic tricuspid stenosis made on such auscultatory findings. A clear-cut demonstration, however, is helpful.

7. Catheterization. While the venous pressure is elevated and circulation times prolonged, the singular demonstration of a typical pressure tracing (fig. 1) obtained during catheterization of the right atrium is our most definitive indication of significant tricuspid stenosis. Our routine catheterizations of patients with mitral stenosis have not shown this pattern.

**CASE HISTORY**

The following case report represents an instance of chronic valvular disease, originally diagnosed as "pure" and solitary mitral stenosis which subsequently was found to have a residual "pure" tricuspid stenosis. Initially the mitral stenosis was eliminated by commissurotomy and after a four-year interval the tricuspid stenosis was treated with commissurotomy.

R. M., a 36 year old white woman was first admitted to the authors' service Oct. 10, 1949. At the age of 7 years the patient had had chorea which lasted for several months and was associated with frequent sore throats and nosebleeds. At age of 19 and again at the age of 22 years, joint pains and fever were present. The patient was bedfast from this time until the age of 30 years with cardiac decompensation (ankle edema, ascites, four-pillow orthopnea, dyspnea, liver enlargement, hemoptysis and showers of petechia). At the age of 33 years an episode of coma occurred which lasted for two days. An electrocardiogram obtained in 1941 showed "tachycardia" and "no axis deviation." Digitalis, mercurial diuretics and a low sodium diet were utilized during the eight years prior to hospital admission.

On admission the brachial blood pressure was 140/84. There was normal sinus rhythm. A diastolic murmur was present over the apex with a sharp mitral first sound. The pulmonic second sound was accentuated. On fluoroscopy, the right ventricle and pulmonary artery were enlarged but the atria and left ventricle were not enlarged. The electrocardiogram demonstrated a rate of 105 per minute, sinus rhythm and right axis deviation. The P-R interval was 0.16 second. The QRS interval was 0.08 second. The RS-T segment was depressed in leads II and III. T waves were upright in II, diphasic in lead III. Cardiac catheterization findings are shown in chart I.

Mitril commissurotomy was performed on Oct. 14, 1949. The mitral slit was found to be 20 mm. in length, and this was enlarged with the knife to a
### Cardiac Catheterizations (Patient—R. M.)

**[Pressures in mm. Hg]**

<table>
<thead>
<tr>
<th></th>
<th>Right Atrium</th>
<th>Right Ventricle</th>
<th>Pulmonary Artery</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Exercise</td>
<td>Rest</td>
</tr>
<tr>
<td><strong>Before mitral commissurotomy—10/13/49</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>5 0</td>
<td>17.5 to 14</td>
<td>14 to 11</td>
</tr>
<tr>
<td>Diastolic</td>
<td>-2 to +2</td>
<td>0 to -1</td>
<td>4.5 to -1</td>
</tr>
<tr>
<td>Average</td>
<td>5 0</td>
<td>16 0</td>
<td>12.5 2</td>
</tr>
</tbody>
</table>

| **3 months after mitral commissurotomy—1/19/50** |      |        |      |          |      |          |
| Systolic            | 9.3 to 7.6 | 14 to 10 | 19 to 8 | 20 to 17 | 12 to 9 | 21 to 17 |
| Diastolic           | -1 to -2 | 0 to 0 | 0 to -2 | -3 to -7 | 7 to 3 | 8 to 3 |
| Average             | 8.6 -1.5 | 12 0 | 13.5 -1 | 19 -5 | 10 5 | 19 5.5 |

| **3½ years after mitral commissurotomy—5/18/53** |      |        |      |          |      |          |
| Systolic            | 20 to 4 | 25     | 14 6  | 26 12.5 |
| Diastolic           | 0      | -10    |      |          |      |          |
| Average             | 12 0   | 26 12.5|      |          |      |          |

The serial pressures illustrate several features during the three and a half year period. The pressures in the right ventricle and pulmonary artery were not elevated in spite of severe mitral stenosis and did not change significantly following the mitral commissurotomy. On the other hand, the systolic pressure in the right atrium increased remarkably and to an amplitude clearly indicating obstruction at the tricuspid valve.

length of 35 to 45 mm., with good valve function resulting. It was noted that the pulmonary artery, although enlarged to three times normal size, did not appear under increased intraluminal pressure, nor did the left atrium. Twelve days after operation the patient was discharged from the hospital in good condition.

On Jan. 17, 1950, the patient was readmitted for her three-month postoperative checkup. She was subjectively improved and had improved exercise tolerance. There was ankle swelling on one occasion. There were three or four episodes of “paroxysmal tachycardia,” each lasting about five minutes. At this examination the brachial blood pressure was 130/96. Normal sinus rhythm was present. The pulmonic second sound was accentuated. There were no systolic murmurs present. The mitral first sound was sharp and there was a diastolic murmur heard at the apex. There was no evidence of either right or left heart failure; venous pressure was not increased; the liver was not enlarged; rales were not heard at the lung bases, and no pretilial edema was present. The electrocardiogram had not changed since the previous admission. A second cardiac catheterization was performed; the results are shown in table 1.

The patient was again admitted on May 18, 1953, for a third cardiac catheterization because of symptoms suggestive of tricuspid stenosis. (See table 1.) This pressure tracing indicated the probable existence of tricuspid stenosis and operation was recommended.

On July 12, 1953, the patient was admitted for the fourth time with complaints of edema of the face, breasts and legs. One year following mitral commissurotomy, the edema of the face and breasts became evident as did a decrease in exercise tolerance. There was a history of occasional dyspnea on exertion.

On examination the face and neck were seen to be noticeably edematous. There was 1 plus ankle and pretibial edema. The liver was at the costal margin and was nontender. There was no cyanosis. Systolic pulsations were noticeable in the neck veins. The lungs were clear. The pulmonic second sound was accentuated. No longer present was the loud snap of the mitral valve. There was a diastolic rumble heard between the mitral area and the xiphoid. The electrocardiogram showed "P wave abnormality and right axis deviation."
Surgical Treatment of Tricuspid Stenosis

On July 17, 1953, tricuspid commissurotomy was done, there being present a tight tricuspid stenosis. Using a mirror image technic of that used for mitral commissurotomy, the anterior commissure was opened with the guillotine knife converting an orifice measuring 13 mm. by 2 mm. to an orifice measuring 30 mm. by 10 mm. Both before and after operation there was an insignificant regurgitation present (fig. 2). Within one hour following operation most of the edema had disappeared and the patient made an uncomplicated recovery. Ten days following operation the patient was discharged from the hospital. The significant findings were as follows: The heart presented a regular rhythm; there was a very faint diastolic blow present to the left of the sternum at the fifth intercostal space; the a wave was absent in the neck veins; and there was no evidence of right heart failure.

At follow-up examination two and a half months after operation the following facts were established. There was no edema present; mercurial diuretics were unnecessary; there was no need for pillows while sleeping; the patient had resumed her occupation as a secretary.

Comment. Appreciating the widespread employment of commissurotomy in the treatment of mitral stenosis, we are faced with the question of how many cases have had unrecognized tricuspid stenosis of a degree serious enough to obviate a clinical cure. In view of a certain percentage of failures in otherwise adequately operated patients, attention might be directed with profit toward the possible coexistence of tricuspid stenosis. In such instances, cardiac catheterization usually will be decisive.

Table 2 indicates that the mitral valve is involved in 90 per cent of individuals dying as a result of chronic rheumatic valve disease, while the tricuspid valve is involved in 30 per cent. The mitral valve is singularly involved in 23 per cent, while there is no instance of singular involvement of the tricuspid valve. The incidence of coexistent involvement of the mitral and tricuspid valves, excluding aortic and pulmonic lesions, is 7 per cent.

Cooke and White report a total of 66 cases: 59 cases of solitary mitral valve involvement, and, in addition, seven cases having combined mitral and tricuspid involvement. Of these, there were four instances of mitral stenosis

Table 2.

<table>
<thead>
<tr>
<th>Year Reported</th>
<th>Authors Series</th>
<th>Total Autopsies with Valve Disease</th>
<th>Total Number with Tricuspid Disease</th>
<th>Total Number with Mitral Disease</th>
<th>Number with Mitral Disease Alone</th>
<th>Number with Tricuspid &amp; Mitral Disease Alone</th>
</tr>
</thead>
<tbody>
<tr>
<td>1924</td>
<td>Coombs¹</td>
<td>97</td>
<td>35 (35%)</td>
<td>97 (100%)</td>
<td>—</td>
<td>12 (12%)</td>
</tr>
<tr>
<td>1925</td>
<td>Thayer²</td>
<td>25</td>
<td>10 (40%)</td>
<td>20 (80%)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>1927</td>
<td>Van Glahn³</td>
<td>101</td>
<td>26 (26%)</td>
<td>57 (50%)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>1935</td>
<td>Bland et al.⁴</td>
<td>100</td>
<td>30 (30%)</td>
<td>98 (98%)</td>
<td>23 (23%)</td>
<td>5 (5%)</td>
</tr>
<tr>
<td>1941</td>
<td>Cooke &amp; White⁵</td>
<td>217</td>
<td>47 (22%)</td>
<td>206 (95%)</td>
<td>59 (22%)</td>
<td>7 (3%)</td>
</tr>
<tr>
<td>1943</td>
<td>Garvin⁶</td>
<td>119</td>
<td>43 (36%)</td>
<td>112 (93%)</td>
<td>28 (23%)</td>
<td>4 (4%)</td>
</tr>
<tr>
<td>1947</td>
<td>Aceves &amp; Carral⁷</td>
<td>147</td>
<td>49 (33%)</td>
<td>—</td>
<td>—</td>
<td>22 (15%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>806</td>
<td>240 (30%)</td>
<td>(90%)</td>
<td>(23%)</td>
<td>(7%)</td>
</tr>
</tbody>
</table>
combined exclusively with tricuspid stenosis, indicating that mitral valve disease, exclusive of aortic and pulmonic valve disease, is complicated by tricuspid stenosis in 6 per cent of the instances.

Unfortunately, none of the other series are detailed enough to allow appraisal of these relationships, and it is hoped that future reports will better illuminate this problem.

Conclusions

1. Some patients with mitral stenosis will be found to have an associated tricuspid stenosis. The incidence of this association is probably around 6 per cent.

2. A case history of one such instance is presented with surgical treatment by commisurotomy resulting in an apparent clinical cure.

3. Appreciation of certain criteria, which are discussed, together with right heart catheterization, will be an aid in the diagnosis of tricuspid stenosis.

Sumario Español

Entre los pacientes que sufren con enfermedad crónica valvular diagnosticados como estenosis mitral, hay algunos que tienen una estenosis tricúspide asociada. El diagnóstico de esta combinación de lesiones es difícil. La oportunidad singular de estudiar la hemodinámica de un caso “puro” de estenosis tricúspide se presentó cuando un paciente originalmente diagnosticado como estenosis mitral, tenía en lugar ambas lesiones y fue tratado por comisurotomía. Subsiguientemente estenosis de la tricúspide se sospechó clínicamente y se demostró por medio de estudios de cateterismo. Comisurotomía tricúspide se hizo cuatro años después de la comisurotomía mitral.

References

1 Coombs, C.: Rheumatic Heart Disease. New York, 1924.
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