Tricuspid Stenosis

A Difficult Diagnosis in the Presence of Atrial Fibrillation

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Roman W. DeSanctis, M.D., and W. Gerald Austen, M.D.

Tricuspid stenosis is a surprisingly common lesion, and indeed has been found at autopsy in approximately 5 to 10% of all patients with rheumatic heart disease.1-4 Although the degree of tricuspid valve narrowing was considered to be inconsequential in the majority of these reported cases, nevertheless a considerable number of patients showed obvious hemodynamically significant tricuspid stenosis which had not been suspected during life.

With the advent of cardiac surgery, the detection of tricuspid stenosis has become extremely important, for not only are adequate means of dealing with tricuspid valve disease now available, but the failure to recognize and correct associated tricuspid stenosis has occasionally been responsible for the failure of patients to improve after surgery for left-sided lesions.1, 5, 6

Perloff and Harvey in an excellent review7 have concluded that the diagnosis of tricuspid stenosis can be made in most patients by careful clinical examination, particularly in the presence of normal sinus rhythm. These authors also pointed out, however, that with atrial fibrillation the manifestations of tricuspid stenosis become more subtle. In the presence of this arrhythmia, evaluation of jugular venous pulsation, auscultation of the murmur of tricuspid stenosis, and demonstration of a gradient on pullback recordings from right ventricle to right atrium become more difficult. Hence the diagnosis may be overlooked.

The purpose of the present report is to emphasize the clinical and hemodynamic problems encountered in making the diagnosis of tricuspid stenosis in the presence of atrial fibrillation and to point out some maneuvers which facilitate recognition of this lesion at cardiac catheterization. The value of simultaneously recorded right atrial and right ventricular pressures, the marked variability of the tricuspid valve gradient during respiration and with atrial fibrillation, and the role of exercise and amyl nitrite in augmenting barely detectable resting gradients across the tricuspid valve will be demonstrated and discussed.

Methods

Ten patients with significant tricuspid stenosis were found among the 415 patients with rheumatic heart disease undergoing cardiac catheterization at the Massachusetts General Hospital from January 1, 1962, to June 30, 1965. Each patient received a complete cardiac evaluation including 12-lead electrocardiogram and cardiac fluoroscopy. Right heart catheterization was performed in the conventional manner; left heart catheterization, when necessary, was carried out by retrograde arterial or transseptal techniques. Simultaneous pressures across the tricuspid valve were obtained either with a no. 7 or 8 double-lumen catheter* or through a standard right heart and transseptal left heart catheter positioned in the right ventricle and right atrium, respectively. The catheters were connected to matched pressure systems consisting of P23Db Statham pressure transducers and Sanborn amplifiers. All pressures were recorded on a Sanborn 550 Polygraph recorder. Cardiac output was measured by the Fick principle. Tricuspid valve area was estimated by the Gorlin formula.8 Pulmonary arteriolar resistance (PAR) was cal-

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culated by the method of Wood.9 Surgery was performed on eight patients utilizing total cardiopulmonary bypass with a Kay-Cross disc oxygenator.

Results

Clinical, hemodynamic, and operative data are presented in tables 1 and 2.

Age and Sex

The group averaged 47 years of age; all patients were female.

Clinical History

The duration of symptoms ranged from 1 to 15 years. Exertional dyspnea and easy fatigability were the most frequent complaints. Symptoms of severe right-sided failure had been noted for at least a year prior to catheterization in nine patients. Recurrent pulmonary edema occurred only in R.R., the patient with the least severe disease of the tricuspid valve. Mitral commissurotomy had been performed in five cases from 3 to 12 years previously. One patient had improved markedly after operation, three had noticed only moderate improvement in symptoms, and the fifth patient, operated on three years previously, had experienced no significant change in her symptoms.

Physical Examination

Prominent V waves, usually increased by inspiration, were observed in the neck veins of five patients; only minimal abnormalities of the jugular venous pulses could be detected in the five remaining cases. Palpation of the cardiac impulse suggested enlargement of the left ventricle in four cases, the right ventricle in two, and both ventricles in four. Auscultatory findings were consistent with mitral stenosis in every case. In two cases an apparently separate diastolic ruffle of grade 1 to 2 intensity (on basis of 1 to 6), increasing with inspiration and suggesting tricuspid stenosis, was heard at the lower left sternal border. These two patients also had loud pansystolic murmurs and thrills, similarly affected by respiration, in the same area. A murmur suggesting tricuspid regurgitation was present in four other cases. Two patients had murmurs consistent with mitral regurgitation; eight patients had a murmur indicative of aortic valve disease.

Electrocardiogram

By definition, atrial fibrillation was present in all 10 patients. Left ventricular hypertrophy was present in two and right ventricular hypertrophy in one. One patient had biventricular hypertrophy. The remaining six patients presented no definite evidence of hypertrophy of either ventricle. A qR pattern in lead V1 was not observed.

Chest Film

Cardiac enlargement was found in every patient although a marked degree of enlargement was observed in only three. In general, changes secondary to the left-sided lesions dominated the radiographic picture in all except one patient (E.M.) in whom gross right atrial enlargement was the most prominent finding. Mild-to-moderate bulging of the lower right cardiac border suggested right atrial enlargement in the other nine patients. No calcification in the region of the tricuspid valve was ever detected.

Associated Lesions

All ten patients had severe mitral stenosis. Other lesions consisted of mitral regurgitation in two, severe aortic stenosis in five, and significant aortic regurgitation in one patient.

Cardiac Catheterization Findings

The pertinent results are summarized in

![Figure 1](http://circ.ahajournals.org/)

*Figure 1*  
Pullback record from right ventricle to right atrium in patient A.M. The cardiac rate averaged 60 beats per minute and there was no definite gradient across the tricuspid valve.
### Table 1

**Clinical and Operative Data on Ten Patients with Significant Tricuspid Stenosis**

<table>
<thead>
<tr>
<th>Patient, age &amp; sex</th>
<th>Clinical history</th>
<th>Physical examination</th>
<th>ECG</th>
<th>Tricuspid stenosis suspected</th>
<th>Operative findings</th>
<th>Associated lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.M. 51 F</td>
<td>Biventricular failure, 6 yr.</td>
<td>Prominent V wave in neck; LV lift; RV lift (?)</td>
<td>AF</td>
<td>No</td>
<td>Moderately severe</td>
<td>Severe MS; mild AS</td>
</tr>
<tr>
<td></td>
<td>Mitral commissurotomy, 3 yr ago; no clinical improvement</td>
<td>Gr 3 apical diastolic rumble &amp; apical &amp; lower LSB pansystolic murmurs, P₂**, no OS</td>
<td></td>
<td></td>
<td>TS &amp; TR</td>
<td></td>
</tr>
<tr>
<td>M.B. 57 F</td>
<td>Left-sided failure, 15 yr.</td>
<td>Neck veins unremarkable; LV lift. Gr 3 apical diastolic murmur, LSB diastolic blow &amp; lower LSB pansystolic murmur; gr 1 lower LSB diastolic rumble</td>
<td>AF</td>
<td>Yes</td>
<td>—</td>
<td>Severe AR &amp; MS</td>
</tr>
<tr>
<td></td>
<td>Mitral commissurotomy, 12 yr ago; recurrent symptoms with biventricular failure, 3 yr</td>
<td></td>
<td>LVH</td>
<td>Yes</td>
<td>Severe</td>
<td>AS &amp; MS</td>
</tr>
<tr>
<td>J.D. 28 F</td>
<td>Dyspnea, 2 yr; biventricular failure with pulmonary emboli, 6 mo</td>
<td>Prominent V waves in neck; RV lift. Gr 4 apical diastolic rumble &amp; aortic systolic &amp; lower LSB pansystolic murmurs; gr 2 lower LSB diastolic rumble with thrill</td>
<td>AF</td>
<td>Yes</td>
<td>Severe</td>
<td>Severe</td>
</tr>
<tr>
<td></td>
<td></td>
<td>RVH</td>
<td></td>
<td></td>
<td>TS &amp; TR</td>
<td></td>
</tr>
<tr>
<td>M.G. 43 F</td>
<td>Dyspnea with progressive left-sided failure, 13 yr.</td>
<td>Prominent V waves in neck; RV lift; Gr 3 apical diastolic rumble, apical &amp; lower LSB pansystolic &amp; aortic systolic murmurs</td>
<td>AF</td>
<td>No</td>
<td>Severe TS; moderate TR</td>
<td>Severe AS, MS &amp; MR</td>
</tr>
<tr>
<td></td>
<td>Biventricular failure, 1 yr</td>
<td></td>
<td>LVH?</td>
<td>No</td>
<td>Severe</td>
<td>Severe AS &amp; MS</td>
</tr>
<tr>
<td>M.I. 49 F</td>
<td>Dyspnea &amp; fatigue; occ. angina, 1 yr</td>
<td>Neck veins unremarkable; slight LV lift. Gr 2 apical diastolic rumble &amp; aortic systolic murmur</td>
<td>AF</td>
<td>No</td>
<td>Severe</td>
<td>Severe AS &amp; MS</td>
</tr>
<tr>
<td>L.M. 56 F</td>
<td>Dyspnea with angina &amp; paroxysmal flutter, 10 yr; intermittent failure in previous year</td>
<td>Neck veins unremarkable; LV lift. Gr 3 aortic systolic &amp; gr 1 aortic diastolic murmurs &amp; gr 1 apical diastolic rumble</td>
<td>AF</td>
<td>No</td>
<td>Moderately severe TS</td>
<td>Severe AS &amp; MS</td>
</tr>
<tr>
<td>E.M. 49 F</td>
<td>Biventricular failure, 6 yr.</td>
<td>Prominent V waves in neck; LV &amp; RV lift. Gr 4 aortic systolic &amp; gr 3 lower LSB pansystolic murmurs</td>
<td>AF</td>
<td>No</td>
<td>Severe</td>
<td>Severe MS; mild AS</td>
</tr>
<tr>
<td></td>
<td>Mitral commissurotomy, 5 yr ago; symptom free for 2 yr; then progressive right-sided failure</td>
<td></td>
<td>RVH</td>
<td>No</td>
<td>TS &amp; TR</td>
<td>mild AS</td>
</tr>
</tbody>
</table>
D.P. 39 F Dyspnea, 7 yr, with congestive failure for 1 yr.
Good history of progressive orthopnea & nocturnal dyspnea
Slightly prominent V waves in neck with slow Y descent;
RV lift. Gr 2 apical diastolic rumble; also heard well at lower LSB
AF No Severe TS Severe MS

R.R. 45 F Intermittent congestive failure, 10 yr. Mitral commissurotomy 8 yr ago with transient improvement. Recurrent pulmonary edema, 2 yr
Prominent V waves in neck;
LV & RV lift. Gr 2 apical diastolic & apical & LSB pansystolic murmurs & LSB diastolic blow. P₂⁺⁺;
loud OS
AF No — Severe MS; moderate MR

R.S. 53 F Dyspnea, 15 yr. Mitral commissurotomy, 12 yr ago; improvement for 5 yr; then recurrent failure with edema
Neck veins unremarkable;
LV & RV lift. Gr 3 apical diastolic rumble & apical pansystolic & aortic systolic murmurs
AF RVH² No Severe TS Severe AS & MS

AS = aortic stenosis; AF = atrial fibrillation; LSB = left sternal border; LVH = left ventricular hypertrophy; MR = mitral regurgitation; MS = mitral stenosis; RVH = right ventricular hypertrophy; TR = tricuspid regurgitation; TS = tricuspid stenosis.
Grading of murmurs is on basis of 1 to 6.

Table 2

<table>
<thead>
<tr>
<th>Patient</th>
<th>PAm</th>
<th>PAR</th>
<th>RVed</th>
<th>Ram</th>
<th>Tricuspid Diastolic Mean Gradient</th>
<th>End-diastolic gradient</th>
<th>CI</th>
<th>Tricuspid valve area, cm²</th>
<th>RV—RA pullback showed TS</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.M.</td>
<td>33</td>
<td>8</td>
<td>/0-9</td>
<td>9</td>
<td>Inspiration 5 4 — — — — — — —</td>
<td>0-4 1.5 1.0 NO</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M.B.</td>
<td>23</td>
<td>4</td>
<td>/0-7</td>
<td>8</td>
<td>6 1 4 — — — — — — — — —</td>
<td>0-4 1.5 0.6 YES</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J. D.</td>
<td>75</td>
<td>42</td>
<td>/0-10</td>
<td>18</td>
<td>15 5 10 — — — — — — —</td>
<td>0-7 1.7 1.2 YES</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M.G.</td>
<td>26</td>
<td>2</td>
<td>/0-6</td>
<td>8</td>
<td>8 2 — — — — — — — —</td>
<td>0-4 1.5 0.9 NO</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M.L.</td>
<td>30</td>
<td>4</td>
<td>/0-3</td>
<td>6</td>
<td>7 1 4 — — — — — — —</td>
<td>0-5 2.4 1.4 NO</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L.M.</td>
<td>30</td>
<td>4</td>
<td>/0-5</td>
<td>6</td>
<td>7 1 4 — — — — — — —</td>
<td>2-8 1.5 0.6 YES</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>E.M.</td>
<td>50</td>
<td>18</td>
<td>/0-10</td>
<td>16</td>
<td>12 8 10 — — — — — —</td>
<td>0-6 2.0 1.3 NO</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D.P.</td>
<td>20</td>
<td>10</td>
<td>/0-2</td>
<td>4</td>
<td>6 2 4 7 — — — — — —</td>
<td>0-3 2.2 1.3 NO</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R.R.</td>
<td>53</td>
<td>10</td>
<td>/0-9</td>
<td>10</td>
<td>5 1 3 7 — — — — — —</td>
<td>0-6 1.5 0.9 NO</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R.S.</td>
<td>28</td>
<td>4</td>
<td>/0-5</td>
<td>9</td>
<td>9 2 6 10 — — — — — —</td>
<td>0-6 1.5 0.9 NO</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

PAm = pulmonary artery mean pressure; PAR = pulmonary arteriolar resistance units; RVed = right ventricular end-diastolic pressure; Ram = right atrial mean pressure; CI = cardiac index.
table 2. Pullback records from right ventricle to right atrium demonstrated a distinct tricuspid valve gradient in the basal state in only three of the ten cases. The difficulty in detecting tricuspid stenosis by the pullback technique is illustrated in figure 1 in which a gradient across the tricuspid valve is difficult to appreciate. Simultaneous right atrial and right ventricular pressures were obtained in nine patients (the initial patient in the series was incompletely evaluated). In every instance a mean diastolic gradient was demonstrated on the simultaneous tracings. This gradient averaged 5.6 mm Hg for the group (range 3 to 10). With normal inspiration the mean gradient rose to an average of 8.3 mm Hg (range 5 to 15), and during expiration it decreased to an average of 2.6 mm Hg (range 1 to 8, fig. 2). The changing gradient during the respiratory cycle was effected primarily by a change in the right ventricular diastolic pressure, which fell during inspiration and rose with expiration. The response of right atrial mean pressure to respiration was unpredictable but such changes in pressure, if they did occur, were small when compared to those observed in right ventricular diastolic pressure. End-diastolic pressure gradients ranged from 0 to 13 mm Hg depending on the length of the previous diastolic filling period as well as the severity of the stenosis (fig. 3). In the five patients who could perform exercise, the resting gradients were significantly increased (average increase 4.3, range 3 to 6 mm Hg).

Inhalation of amyl nitrite effected an in-

![Figure 2](image-url)

**Figure 2**

*Effect of respiration on the gradient across the tricuspid valve measured through a no. 8 double-lumen catheter (upper record). Effect of amyl nitrite on the gradient (lower record).*
crease of similar magnitude in the two patients in whom it was employed (fig. 2). The cardiac index was moderately lowered in three cases but markedly reduced in the remaining seven. Elevation of the pulmonary arteriolar resistance was found in four patients, all of whom had hemodynamic evidence of right ventricular failure.

**Operative Findings**

Exploration of the tricuspid valve confirmed the preoperative diagnosis of significant tricuspid stenosis in all eight patients undergoing corrective surgery. Four patients were judged to have moderate to severe tricuspid regurgitation associated with the obstructive lesion.

**Discussion**

The detection of tricuspid stenosis in the presence of atrial fibrillation is difficult for several reasons: (1) The accompanying mitral valve disease which is usually present may produce virtually identical auscultatory findings and hence obscure those of the tricuspid disease. An increase in the intensity of the diastolic murmur during inspiration is very helpful since it points to the murmur's being of tricuspid valve origin. However, this change in the tricuspid murmur often is not present or cannot be detected by ordinary auscultation. (2) The characteristic large, and occasionally giant, A waves in the neck veins and right atrium of patients with tricuspid stenosis are lost after the onset of atrial fibrillation. The resultant venous wave form, although frequently abnormal, is of diagnostic help only if a slow Y descent can be demonstrated. The problem may be further complicated by the distortion of the venous pulse by large V waves secondary to associated tricuspid regurgitation. (3) The cardiac output is usually considerably reduced and the mean diastolic gradient is correspondingly small despite the presence of severe tricuspid stenosis. The resulting murmur is of low intensity, maximal in early diastole when the gradient is the largest, and may be obscured by the murmur of mitral stenosis. Such small pressure differences are difficult to detect on routine pullback tracings across the tricuspid valve. This difficulty is enhanced by the rise and fall of intracardiac filling pressures during the respiratory cycle and by the marked variability of these pressures in the presence of atrial fibrillation. With long diastolic filling periods, particularly with greatly diminished cardiac output, adequate time for atrial decompression results in equalization of atrial and ventricular pressures in the terminal phases of diastole and thereby effectively masks tricuspid valve obstruction.

*Figure 3*

Simultaneous right atrial and right ventricular pressures illustrating the effect of variable diastolic filling periods on the tricuspid gradient.
on a pullback tracing. (4) With significant tricuspid regurgitation, it may be difficult to interpret the significance of an early diastolic rumble at the lower left sternal border since such a murmur may be produced either by increased flow across a slightly deformed tricuspid orifice or by actual anatomic tricuspid valve obstruction.

This group of ten patients illustrates the difficulty encountered in detecting tricuspid stenosis either by physical examination or by the usual hemodynamic techniques in the presence of atrial fibrillation. In eight of the ten patients, the lesion was not suspected clinically prior to cardiac catheterization. In the first patient of the series (A.M.), cardiac examination suggested tricuspid regurgitation. The pullback tracing from right ventricle to right atrium at cardiac catheterization, however, did not show a characteristic wave form of tricuspid regurgitation in the right atrial pressure curve or evidence of a gradient across the tricuspid valve. Nevertheless, subsequent surgical exploration of the tricuspid valve revealed moderately severe tricuspid stenosis and regurgitation. Following this experience it became the policy of the laboratory to evaluate tricuspid valve function by simultaneous measurement of right ventricular and right atrial pressures. In the 42 months since that time significant tricuspid stenosis associated with atrial fibrillation has been detected in nine additional cases, in six of which it would probably have been overlooked if this maneuver had not been used. In five the diagnosis was confirmed by provoking an increase in a small tricuspid gradient by augmenting intrathoracic venous return with deep inspiration or exercise. The value of such maneuvers has been stressed previously by Killip and Lukas.10 Our studies also show that the pullback record will be most helpful when obtained at the height of inspiration or during exercise when both venous return and rate are increased. The experience with amyl nitrite, although limited, suggests that this agent may be useful in enhancing the gradient across the tricuspid valve by increasing venous return as well as increasing heart rate. Such an agent may be particularly applicable to the patient who is too ill to perform muscular exercise.

The practical importance of assessing tricuspid valve function is in the selection of the proper surgical treatment for the patient. The failure to recognize significant organic tricuspid valve disease may result in an increased operative mortality and has been shown on occasion to explain why a patient does not improve following adequate corrective surgery for left-sided valve lesions.1 Demonstration of tricuspid stenosis, regardless of its severity, constitutes strong evidence in favor of organic tricuspid disease and should lead to a careful exploration of the tricuspid valve at the time of surgery and operative correction if the lesion is judged to be significant.

Summary

The present report concerns ten patients with significant tricuspid stenosis and atrial fibrillation. The diagnosis could be made clinically in only two patients and by pullback record from right ventricle to right atrium in three, including the two mentioned previously. Careful hemodynamic assessment of tricuspid valve function by using simultaneously recorded right atrial and right ventricular pressures resulted in the detection of significant tricuspid stenosis in the nine patients on whom these measurements were made. The influence of atrial fibrillation and respiration on the tricuspid gradient and the role of exercise and amyl nitrite in augmenting barely detectable resting gradients are demonstrated.

References

4. Lambrew, C. T., and Goldsmith, E. I.: Tri-
TRICUSPID STENOSIS


Warnings to the Young Auscultator

I would, therefore, make the following remarks, as warnings to the young auscultator. First. Be careful about inferring too much from physical signs alone. Second. Do not risk your reputation by too nice a diagnosis. Third. If you make an error, comfort yourself with the belief, that, perhaps, part of the difficulty lies inherent in the subject, and not wholly in your blundering ear and judgment. Fourth. If you make many errors, still be hopeful, at thinking how much more clear-sighted you are than those who have gone before you. Believe that auscultation teaches very much although it may be imperfect. Learn humility; but do not despair of becoming more accurate in the future. Fifth above all, do not, from the physical signs alone, imagine that you have found out a severe organic disease of the heart, when perhaps, nothing but a functional derangement really exists, that will mock your fatal prognosis.—Henry I. Bowditch. The Young Stethoscopist. New York, Hafner Publishing Company, 1964, p. 157. (Original publication, 1846)
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