Clinical Recognition of Tricuspid Stenosis

By Joseph K. Perloff, M.D., and W. Proctor Harvey, M.D.

Opinion differs regarding the accuracy with which a clinical diagnosis of tricuspid stenosis can be made. It has been stated that the recognition of tricuspid stenosis was clinically feasible only in advanced cases, and that the murmurs of tricuspid and mitral stenosis were indistinguishable. Bedside diagnoses were made in 12 of 15 cases in one series, however, and in 11 of 12 cases in another. There is evidence that the incidence of tricuspid stenosis has been underestimated suggesting that the lesion, particularly early in its natural history, is still being overlooked. These oversights may contribute to the failure of improvement in some patients after mitral valvulotomy. It must also be noted that in stenosis of the tricuspid valve the transvalvular gradient is commonly small and unless the diagnosis is actively sought for, it can be overlooked during routine venous catheterization. Clinical suspicion of this lesion may thus underlie the success of physiologic studies in establishing the diagnosis and in assessing its severity. It is the purpose of this paper to evaluate the validity of clinical criteria upon which recognition of tricuspid stenosis might be based, emphasizing particularly the physical signs and their mechanisms.

Materials and Methods

Thirteen patients were studied, all personally seen by the authors. Careful attention was directed to the peripheral arterial pulse, to the jugular venous pulse, to palpation of the precordium, and to auscultation of the heart. Five patients were seen at the Georgetown University Hospital, 6 at the Clinic of Surgery of the National Institutes of Health, 1 at the U. S. Naval Hospital, Bethesda, and 1 at Walter Reed Army Hospital. In 12 of the cases the diagnosis of tricuspid stenosis was made clinically either before catheterization or without knowledge of the physiologic data. In the thirteenth case the diagnosis was known beforehand. In one of the earlier patients in this series the diagnosis of tricuspid stenosis was made clinically, was missed at catheterization, but was proved at autopsy.

Aside from routine electrocardiograms, chest x-rays, and fluoroscopy of the heart, venous catheterization was performed in all patients, and left heart catheterization in 9. The diagnoses were further confirmed at surgery in 8 patients, and at autopsy in 2. The physiologic diagnoses of tricuspid stenosis were based principally on the criteria of Killip and Lukas. The most refined technic in our experience consisted of the use of a double-lumen catheter with simultaneous right atrial and right ventricular pressure pulses inscribed from the same base line on a photographic recorder after exercise and during deep respiratory excursions. The transvalvular gradient, exaggerated by exercise, was further magnified during inspiration. It should be emphasized that simple continuous recording of pressure while the catheter was pulled from the right ventricle into the right atrium was a relatively insensitive method and could occasionally miss significant tricuspid stenosis.

In 12 of the 13 patients thoracic wall logarhythmic phonocardiograms were taken with either a 3-channel Cambridge model or a Sanborn Twin-Beam recorder at paper speeds of 50 to 75 mm. per second. In 3 patients intracardiac phonocardiograms were taken with a catheter-tip microphone. Indirect carotid and jugular venous pressure pulses were taken with a pulse wave amplifier attached to the neck with a suction cup. In 8 patients, simultaneous right atrial and right ventricular pressure pulses were inscribed on a cathode-ray recorder or on a Sanborn Twin-Viso recorder. Simultaneous left atrial and left ventricular pulses were obtained by means of the bronchoscopic technic of left heart catheterization. Retrograde aortic catheterization for the detection of aortic insufficiency was done in the Clinic of Surgery of the National Heart Institute by the method of Braunwald and Morrow.


†The left heart catheterizations were done by Dr. Andrew G. Morrow, Chief, Clinic of Surgery, National Institutes of Health, Bethesda, Md.

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## Table 1
Clinical Data in Thirteen Cases of Tricuspid Stenosis

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Sex and current age (yr)</th>
<th>History of rheumatic fever</th>
<th>Age at onset of symptoms (yr)</th>
<th>Age at onset of symptoms (yr)</th>
<th>Awareness of &quot;A&quot; wave</th>
<th>Syncope</th>
<th>Orthopnea</th>
<th>Nocturnal dyspnea</th>
<th>Acute pulmonary edema</th>
<th>Hemoptysis</th>
<th>Fatigue</th>
<th>Chest pain</th>
<th>Edema, ascites, pleural effusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>34 F</td>
<td>Chorea</td>
<td>29</td>
<td>29</td>
<td>Mild</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Ill-defined</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>28 F</td>
<td>Sore throat</td>
<td>12</td>
<td>Asymptomatic</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>35 F</td>
<td>Scarlet fever age 10</td>
<td>Indefinite</td>
<td>10, mild dyspnea</td>
<td>Mild to moderate</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Innocent mammary-type</td>
<td>-</td>
</tr>
<tr>
<td>4</td>
<td>35 F</td>
<td>Age 7, 3 recurrences</td>
<td>20</td>
<td>31</td>
<td>Marked</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Ankle edema, hepatomegaly</td>
</tr>
<tr>
<td>5</td>
<td>30 F</td>
<td>Prob, age 13</td>
<td>19</td>
<td>24</td>
<td>Severe</td>
<td>-</td>
<td>Mild</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Edema, hepatomegaly</td>
</tr>
<tr>
<td>6</td>
<td>27 F</td>
<td>Polyarthritis age 9, chorea</td>
<td>17</td>
<td>&quot;Always&quot; dyspneic</td>
<td>Marked</td>
<td>3 Episodes</td>
<td>Mild</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Hepatomegaly, with onset atrial fibr.</td>
</tr>
<tr>
<td>7</td>
<td>19 M</td>
<td>Told of rheum, fever age 13</td>
<td>19</td>
<td>18</td>
<td>Mild</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Occas. RUQ discomfort on effort</td>
</tr>
<tr>
<td>8</td>
<td>32 F</td>
<td>Told of rhem, fever age 13</td>
<td>22</td>
<td>13</td>
<td>Moderate</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Ankle edema, mild hepatomegaly</td>
</tr>
<tr>
<td>9</td>
<td>23 M</td>
<td>Age 14</td>
<td>16</td>
<td>18</td>
<td>Marked</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Edeema, hepatomegaly</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>42 F</td>
<td>Age 7</td>
<td>16</td>
<td>22</td>
<td>Moderate</td>
<td>Once</td>
<td>During pregnancy</td>
<td>Twice during pregnancy</td>
<td>Freq. bouts during pregnancy</td>
<td>-</td>
<td>-</td>
<td>Mild hepatomegaly</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>29 F</td>
<td>Childhood</td>
<td>26</td>
<td>Marked</td>
<td>Repeated</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Since early childhood</td>
<td>Edema, ascites, hepatomegaly, pleural effusion</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>28 F</td>
<td>Questionable before atrial fibr.</td>
<td>26</td>
<td>Questionable before atrial fibr.</td>
<td>Moderate</td>
<td>Dizziness</td>
<td>-</td>
<td>During winter bronchitis</td>
<td>-</td>
<td>Streaked with bronchitis</td>
<td>+</td>
<td>Vague</td>
<td>-</td>
</tr>
<tr>
<td>13</td>
<td>32 F</td>
<td>Freq. upper respir. infec., tonsillectomy</td>
<td>29</td>
<td>&quot;as long as can recall&quot;</td>
<td>Marked</td>
<td>Effort weakness</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Easily for past 3 yrs.</td>
<td>Edema, ascites, hepatomegaly</td>
</tr>
</tbody>
</table>
Table 2

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Pure TS</th>
<th>TS with slight to moderate TI</th>
<th>PA pressure (mm. Hg)</th>
<th>Rt. atrial pressure (mm. Hg)</th>
<th>Tricuspid gradient (mm. Hg) or orifice size</th>
<th>MS with slight to moderate MI</th>
<th>LA or PCVP (mm. Hg)</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>+</td>
<td>16/6 (12)*</td>
<td>A 13-16</td>
<td>V 3-5</td>
<td>4-5</td>
<td>--</td>
<td>Post exercise PCVP 10</td>
</tr>
<tr>
<td>2</td>
<td>+</td>
<td>20/10 (13)</td>
<td>A 14-17</td>
<td>V 7-9</td>
<td>3-4</td>
<td>--</td>
<td>Post exercise PCVP 10</td>
</tr>
<tr>
<td>3</td>
<td>+</td>
<td>20/6 (11)</td>
<td>A 11</td>
<td>V 3-4</td>
<td>5-6</td>
<td>--</td>
<td>LA (6)</td>
</tr>
<tr>
<td>4</td>
<td>+</td>
<td>50/25 (33)</td>
<td>A 10-12</td>
<td>V 3-4</td>
<td>5</td>
<td>+</td>
<td>LA (22)</td>
</tr>
<tr>
<td>5</td>
<td>+</td>
<td>100/50 (67)</td>
<td>A 30-35</td>
<td>V 20-25</td>
<td>Fingertip</td>
<td>+</td>
<td>PCVP (20)</td>
</tr>
<tr>
<td>6</td>
<td>+</td>
<td>39/19 (25)</td>
<td>A 10</td>
<td>V 7</td>
<td>2 cm.²</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>+</td>
<td>25/10 (15)</td>
<td>A 18-20</td>
<td>V 5-7</td>
<td>3-4</td>
<td>+</td>
<td>LA (20)</td>
</tr>
<tr>
<td>8</td>
<td>+</td>
<td>32/10 (17)</td>
<td>A 10-12</td>
<td>V 3-4</td>
<td>Surgery, fingertip</td>
<td>A 25</td>
<td>V 12</td>
</tr>
<tr>
<td>9</td>
<td>+</td>
<td>85/40 (55)</td>
<td>A 16-20</td>
<td>V 14-19</td>
<td>5</td>
<td>Moderate A 35 V 40</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>+</td>
<td>42/23 (27)</td>
<td>A 11</td>
<td>V 4</td>
<td>5</td>
<td>+</td>
<td>LA (21)</td>
</tr>
<tr>
<td>11</td>
<td>+</td>
<td>66/32 (44)</td>
<td>Atrial fib.</td>
<td>V 15-19</td>
<td>.5 cm.²</td>
<td>Slight LA (30)</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>+</td>
<td>24/10 (18)</td>
<td>A 11-13</td>
<td>V 6</td>
<td>4-5</td>
<td>Slight A 12 V 10 (8)</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>+</td>
<td>67/41 (50)</td>
<td>Atrial fib. (9)</td>
<td></td>
<td>5</td>
<td>Autopsy, orifice 1½ fingers</td>
<td></td>
</tr>
</tbody>
</table>

*Figures in parentheses represent the mean value.

Abbreviations: TS, tricuspid stenosis; TI, tricuspid insufficiency; PA, pulmonary artery; MS, mitral stenosis; MI, mitral insufficiency; LA, left atrial; PCVP, pulmonary capillary venous pressure; AS, aortic stenosis; AI, aortic insufficiency. A = "a" wave; V = "v" wave.

Results

Age and Sex

There were 12 women and 1 man, ranging in ages from 19 to 42 years, with a mean age of 30 years.

History

There was a convincing history of antecedent rheumatic fever in 9 cases, a suggestive history in 3 cases (1 had chorea alone), and no historical evidence of rheumatic fever in 1 other case. One patient was completely asymptomatic, 5 patients were slightly to moderately symptomatic, 5 were markedly symptomatic, and 2 were incapacitated. The classic picture of tricuspid stenosis in the disabled, fibrillating patient with hepatomegaly, jaundice, edema, and ascites, was present in only 2 instances. The distribution and frequency of specific cardiovascular symptoms are seen in table 1.

Associated Valve Lesions

Coexisting mitral valve disease was present in all except 1 case, and the aortic valve was involved in all except 2. The tricuspid valve was purely stenotic in approximately half the cases (table 2). In 1 case studied clinically, physiologically, and at autopsy, it appeared that the presence of pulmonary hypertension...
Physiologic Data in Thirteen Cases of Tricuspid Stenosis

<table>
<thead>
<tr>
<th>Mitral gradient (mm. Hg) or orifice size</th>
<th>Pure AS</th>
<th>Pure AI</th>
<th>AS and AI</th>
<th>Aortic gradient (mm. Hg) or orifice size</th>
<th>Cardiac Index (L./Min./M.² BSA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>+ TS&gt;MS</td>
<td>Moderate</td>
<td></td>
<td></td>
<td>Rest 1.7</td>
<td>Exercise 1.6</td>
</tr>
<tr>
<td>+</td>
<td>Mild</td>
<td></td>
<td></td>
<td>Rest 2.5</td>
<td>Exercise 5.0</td>
</tr>
<tr>
<td>+</td>
<td>Moderate</td>
<td></td>
<td></td>
<td>Rest 2.0</td>
<td>Exercise 2.6</td>
</tr>
<tr>
<td>+</td>
<td>AS, trivial AI</td>
<td>70</td>
<td></td>
<td>Invalid</td>
<td></td>
</tr>
<tr>
<td>Surgery, less than fingertip 0.9 cm.²</td>
<td></td>
<td></td>
<td></td>
<td>Rest 1.7</td>
<td>Exercise 1.7</td>
</tr>
<tr>
<td>6</td>
<td>Moderate</td>
<td></td>
<td></td>
<td>Rest 2.5</td>
<td></td>
</tr>
<tr>
<td>Surgery, less than fingertip 18</td>
<td>Moderate</td>
<td></td>
<td></td>
<td>Rest 1.9</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>Slight</td>
<td></td>
<td></td>
<td>Rest 1.6</td>
<td></td>
</tr>
<tr>
<td>1 cm.²</td>
<td>+</td>
<td>Severe</td>
<td>AS, trivial AI</td>
<td>0.3 cm.²</td>
<td>Rest 1.5</td>
</tr>
<tr>
<td>10</td>
<td></td>
<td></td>
<td></td>
<td>Exercise 2.0</td>
<td>Invalid</td>
</tr>
<tr>
<td>Autopsy, orifice less than fingertip</td>
<td></td>
<td></td>
<td></td>
<td>Rest 1.66</td>
<td></td>
</tr>
</tbody>
</table>

had caused an anatomically stenotic tricuspid valve to become insufficient as well. This patient had the highest pulmonary arterial pressure in the series (100/50, mean 67). In a second case, considered to have mild, pure tricuspid stenosis, the valve became insufficient after the onset of rapid, atrial fibrillation and right ventricular failure, an observation similar to the experience of others.¹⁰ In 4 cases, the tricuspid stenosis was estimated to be of greater hemodynamic severity than the mitral stenosis, an incidence approximating that of Gibson and Wood.³ One of these patients had a calculated tricuspid orifice smaller than the calculated mitral orifice with surgical confirmation of these impressions. The other 3 patients (all of whom demonstrated mild to moderate aortic insufficiency) had normal postexercise mean left atrial pressures. One had no detectable transvalvular mitral gradient and no fluoroscopic evidence of left atrial enlargement. No patient in this series had isolated tricuspid stenosis. Only 5 instances of this occurrence were found in the literature.³–⁵, ¹¹, ¹²

Jugular Venous and Right Atrial Pressure Pulses

The jugular venous pulse was analyzed at the bedside according to the following method,
without which correlation with the right atrial pulse was occasionally poor. The internal jugular vein was observed with the upper half of the patient’s body elevated to the point that allowed the vein to empty and fill with the greatest amplitude (average 25° to 35° from horizontal). The contralateral carotid artery was gently palpated for timing purposes. The patient’s head was rotated slightly toward the side of observation in order to relieve tension of the sternocleidomastoid on the underlying internal jugular vein. Of the 11 patients with sinus rhythm the “A” waves were giant in 5, marked in 6, and modest in none (table 2 and fig. 1A). These represented right atrial “A’” waves of 14 to 20 mm. Hg in 4 of the patients with giant “A” waves, 30 to 35 mm. Hg in the fifth patient, and 10 to 13 mm. Hg in the 6 patients classified as marked. The V waves were inconspicuous, and the Y descents imperceptible except in 2 cases with pulmonary hypertension and right ventricular failure. Each of the 12 patients with established atrial fibrillation had an elevated V wave with a gentle Y descent, and the patient who began to fibrillate several weeks after catheterization had a similar jugular venous pulse (fig. 1B).

**Pulmonary Arterial Pressure**

In 6 patients the mean pulmonary arterial pressure was between 10 and 20 mm. Hg, in 3 between 20 and 35 mm. Hg, and in the remaining 4 it was 44, 50, 55, and 67 mm. Hg (table 2). In the latter 4 patients the mean left atrial pressures were 30, 22, 32, and 20 mm. Hg, respectively.
Palpation of the Precordium

A right ventricular lift was conspicuous in 3 patients, and moderate in a fourth, who developed increasing congestive heart failure after the onset of atrial fibrillation. The sound of pulmonary valve closure was palpable in only 2 instances. A lower left sternal edge diastolic thrill, which either appeared or increased with inspiration, was present in 4 cases. Palpation of the left ventricle and of mitral and aortic thrill's varied with the co-existing valvular diseases.

Auscultatory and Phonocardiographic Findings

The first heart sound was obviously accentuated in 7 cases, moderately accentuated in 3, and within normal limits in 3. In the case with no evident mitral valve disease, mitral valve closure was of normal intensity and tricuspid closure was moderately increased. The first sound was single in 6 instances and was split into mitral and tricuspid valve components in 7 instances. The second heart sound was single in 10 instances, and split into pulmonary and aortic components in 3 instances. One of these latter cases had aortic stenosis and the second sound was paradoxically split. In 3 instances, the single second sound in the second left interspace was sufficiently loud to suggest pulmonary hypertension. An additional sound consistent with the opening snap of the tricuspid valve was recorded in 2 patients (fig. 2) but was not appreciated by auscultation. Identification of this sound was based on the following criteria: (1) its occurrence 0.06 to 0.07 second after pulmonary valve closure, an interval corresponding to the estimated duration of right ventricular isometric diastole, (2) the failure of the interval between the single second sound and the additional sound to increase with inspiration as it might if these events were aortic and pulmonary valve closures, (3) the identification of normal inspiratory splitting and expiratory synchrony of the second sound in 1 of the 2 patients, and (4) the identification in both patients of a separate later sound with the timing of a mitral opening snap. The precedence in the cardiac cycle of tricuspid over mitral opening snap (first and fourth criteria) is at variance with statements of others and is based on the following observations. In the dog, isometric right ventricular diastole (the interval between closure of the pulmonary valve and opening of the tricuspid) is significantly shorter than isometric left ventricular diastole (the interval between closure of the aortic valve and opening of the mitral). The sequence of events of the cardiac cycle in the dog and in man is generally similar. Finally, the opening sound of the tricuspid valve that is believed to occur occasionally in atrial septal defect has an early timing consistent with the foregoing contentions.

In all patients the diastolic murmur of tricuspid stenosis was appreciated. This murmur was presystolic, with absent or trivial mid-diastolic components in the patients with sinus rhythm (fig. 3) and was mid-diastolic (rapid flow phase) in the 3 patients with
Figure 2
A woman, age 29, with atrial fibrillation, trivacular stenosis, and slight aortic insufficiency. Upper left tracing recorded between apex and lower left sternal edge: opening snap (OS) considered to be mitral. Upper right tracing recorded at lower right sternal edge: opening snap (OS) considered to be tricuspid. Lower tracing recorded at pulmonic area (PA) to illustrate inspiratory splitting of aortic (A₂) and pulmonic (P₂) components of the second heart sound.

Figure 3
A woman, age 32, with tricuspid stenosis, mitral stenosis, and pure aortic insufficiency. Note inspiratory augmentation of the presystolic murmur. Phonocardiogram recorded at the lower left sternal edge. PSM, presystolic murmur; S₁, first heart sound; S₂, second heart sound; OS, opening snap (here thought to be mitral); CAR, carotid pulse; DN, dicrotic notch.
atrial fibrillation (fig. 4). It was best heard at the lower left sternal edge, especially the fourth intercostal space, but occasionally in the third or fifth intercostal spaces. It characteristically increased during inspiration and decreased during expiration. The increment of change was often 2 to 3 grades. In several instances the murmur was virtually inaudible during expiration, but was grade III during inspiration. The mitral stenotic murmur was heard best at the cardiac apex. When the stethoscope was "inched" from this area to the left sternal edge, there was often an auscultatory hiatus in which the murmur of mitral stenosis became faint or absent, followed by the appearance of the murmur of tricuspid stenosis as the left sternal border was reached.

Intracardiac phonocardiograms (fig. 5) done on 3 patients revealed that the murmur was absent when the microphone was located on the atrial side of the tricuspid valve, but was grossly apparent when the catheter tip was advanced to the right ventricular side of the valve, where it occupied a position in the direction of turbulent flow. The murmurs recorded in the right ventricular cavity distal to the tricuspid valve increased with inspiration and decreased with expiration. They differed from the thoracic wall phonocardiograms in 2 respects: (1) the increased intensity of the presystolic component, and (2) the appearance of a mid-diastolic component.

**Hemodynamic Observations**

Simultaneous right atrial and right ventricular pressure pulses were recorded in 8 patients in this series, 6 with sinus rhythm (fig. 6), and 2 with atrial fibrillation (fig. 7). Simultaneous left atrial and left ventricular pressure pulses were recorded* in 5 additional patients with mitral stenosis, (3 with sinus rhythm, and 2 with atrial fibrillation) 1 of which was in this series. The tricuspid transvalvular gradient increased with inspiration and decreased with expiration. In 5 cases this was due both to a fall in right ventricular diastolic pressure and to a concomitant rise in right atrial pressure. In 2 cases it was due principally to a fall in right ventricular diastolic pressure, the right atrial pressure remaining unchanged or falling only slightly. In 1 case the gradient increased due to a rise.

*Published with the kind permission of Dr. Andrew G. Morrow, Chief, Clinic of Surgery, National Institutes of Health, Bethesda, Md.
A woman, age 34, with tricuspid and mitral stenosis. Upper tracings are thoracic wall phonocardiograms recorded at the lower left sternal edge (LSE) to illustrate inspiratory increase of the presystolic murmur (PSM). Lower series are intracardiac phonocardiograms. With the microphone in right ventricle (RV) the mid-diastolic and presystolic murmurs increase with inspiration. In the right atrium (RA) no diastolic murmur is recorded even during inspiration. The MSM is a midsystolic murmur across a stenotic aortic valve, well recorded in RA because of its anatomic proximity to the aorta. "X" is an artifact.

A woman, age 32, with tricuspid and mitral stenosis, and pure aortic insufficiency. Note selective inspiratory increase in tricuspid transvalvular gradient due to concomitant rise in right atrial (RA) pressure and fall in right ventricular (RV) pressure. RA and RV pressure pulses recorded simultaneously through a double-lumen catheter. (Courtesy, Dr. Andrew G. Morrow.)
A woman, age 29, with atrial fibrillation, trivalvular stenosis, and slight aortic insufficiency. The inspiratory fall and expiratory rise in right ventricular (RV) diastolic pressures are not paralleled by pressure changes of equal magnitude in the right atrium (RA), resulting in an inspiratory increase and expiratory decrease in tricuspid transvalvular gradient. The RA and RV pulses were recorded simultaneously through a double-lumen catheter. (Courtesy, Dr. Andrew G. Morrow.)

in right atrial pressure without change in right ventricular diastolic pressure. When similar pulses were recorded in 3 cases with the chest opened during surgery, these respiratory variations in transvalvular gradient failed to occur. The diastolic gradient across the mitral valve remained unchanged during the respiratory cycle, since the inspiratory and expiratory changes in the left atrial and left ventricular diastolic pressures were of equal magnitude (fig. 8).

The Electrocardiogram (fig. 9)

Eleven cases had sinus rhythm (1 patient began to fibrillate under observation) and 2 had established atrial fibrillation. Electrocardiographic evidence of right ventricular hypertrophy was unequivocally evident in none of the patients, although 4 had vertical electrical axes. Left ventricular hypertrophy occurred in 4 instances, and an additional patient had left axis deviation. When atrial enlargement was diagnosed according to the criteria of P-wave amplitude, P-wave configuration, or P/P-R ratio, isolated right atrial enlargement was present in 3 of the cases, and bialtrial enlargement was present in 7 cases. One patient had a QR pattern in V1, suggesting right atrial enlargement based on the observations of Sodi-Pallares.

X-rays (fig. 10)

The posteroanterior projection was found to be the most rewarding. In this view the portion of the right cardiac contour occupied by the right atrium was moderately to markedly enlarged, and the portion of the left cardiac contour occupied by the pulmonary artery segment was only slightly to modestly enlarged. Calcification of the tricuspid valve was not identified in any case.

Discussion

Although the reported incidence of tricuspid stenosis does not necessarily reflect the frequency with which this lesion is of hemo-
A woman, age 28, with mitral stenosis and mild aortic stenosis. The inspiratory fall and expiratory rise in diastolic pressure in the left ventricle (LV) is paralleled by pressure changes of equal magnitude in the left atrium (LA) leaving the mitral transvalvular gradient unchanged during respiration. BA, brachial artery. (Courtesy, Dr. Andrew G. Morrow.)

The first feature that might arouse suspicion is the history in a patient with mitral stenosis, who, although perhaps dyspneic and orthopneic, displays a relative paucity of acute paroxysmal symptoms such as pulmonary edema, paroxysmal nocturnal dyspnea, and the pulmonary apoplexy type of hemoptysis. This could result from an attenuating effect of the tricuspid obstruction on abrupt increases in blood flow into the lesser circulation. This assumption might also underlie previous observations that in more advanced cases of tricuspid stenosis incapacity may not be so pronounced as one would expect from the recurrent hepatomegaly, ascites, and edema. Although the suggestion of the "protective" role of the stenotic tricuspid valve has been commented upon, it appears that from the symptomatic point of view it may be the paroxysmal features of pulmonary symptomatology that are most frequently attenuated.

It is in the physical signs, however, that the highest index of suspicion is aroused. The peripheral arterial pulse (brachial and carotids were routinely palpated) was of interest in two respects. First, to emphasize that the prominent pulsation seen in the neck was venous and not arterial, and second, to detect evidence of aortic insufficiency (especially the pulsus bisferiens) as an aid in distinguishing a left parasternal early diastolic murmur from a Graham-Steell. The pulse most rewarding to analyze was that seen in the internal jugular vein. The typical tricuspid stenotic jugular venous pulse with sinus rhythm (fig. 1A) consisted of an "A" wave of unusual magnitude, occasionally reaching
Figure 9

Note evidence of right atrial enlargement in lead II and negative evidence of right ventricular hypertrophy in lead V1. In no. 9 the QR in V1 suggests right atrial enlargement according to criteria of Sodi-Pallares. In 10a and 10b (from the same patient) right atrial enlargement was difficult to assess because of changing P-wave configuration and P-R relationship.
the angle of the jaw, a small "V" wave, and a gentle, usually imperceptible "Y" descent. The patient may report awareness of this large venous pulse, which may become still more conspicuous during effort or excitement. Although it has been said that a giant "A" wave is of limited value in the diagnosis of tricuspid stenosis because of its occurrence in other conditions, we think that analysis of this pulse configuration in proper context can alone be virtually diagnostic of tricuspid valvular obstruction. In an acyanotic patient isolated "A" waves of giant proportion unassociated with "V"-wave elevation or increased rate of fall of the "Y" descent are seen only if the right atrium contracts against a resistance at the tricuspid orifice, or if it contracts against the resistance offered by a severely hypertensive but nonfailing right ventricle. If this pulse contour is observed, therefore, the diagnosis of tricuspid stenosis, pulmonary stenosis, or pulmonary hypertension can immediately be suspected. Pulmonic stenosis should cause no confusion in the cases under consideration. In the presence of pulmonary hypertension of sufficient severity to generate the giant "A" wave, one almost invariably can feel a heaving lift of the right ventricle in the third and fourth intercostal spaces along the left sternal edge, and a palpable shock of accentuated pulmonary valve closure. The finding of an isolated, dominant giant "A" wave in the jugular venous pulse unassociated with either a palpable right ventricle, a palpable pulmonary second sound, or a thrill of pulmonic stenosis, is in itself strong presumptive evidence that the resistance generating the "A" wave is at the level of the tricuspid valve. The availability of this sign is emphasized by the frequency with which sinus rhythm occurred when the diagnosis of tricuspid stenosis was made relatively early in the course of the disease. With atrial fibrillation analysis of the jugular ve-

Figure 10

Six representative x-rays (cases 2, 7, and 5 (upper); cases 4, 10, and 8 (lower)) illustrating mild to marked enlargement of the right atrial contour (RA) associated with only modest enlargement of the pulmonary artery segment (PA). Left atrial appendage (LA).
TRICUSPID STENOSIS

ous pulse is still valuable but more difficult to assess. The stenotic tricuspid valve elevates the right atrial pressure and impedes its rate of fall in a manner analogous to the effect of mitral stenosis on the left atrial pressure pulse.\(^{31}\) Hence, an elevated "Y" wave and a gentle "Y" descent in the jugular venous pulse are the only clues of tricuspid stenosis in the presence of atrial fibrillation (fig. 1B).

At this point in the physical examination one should carry palpation of the precordium to the lower left sternal edge to seek the tricuspid stenotic thrill. The fingertips should explore the area carefully being aware that the thrill not only may be exceedingly limited in location, but typically will increase with inspiration and decrease with expiration.

Auscultation is the most important phase of the examination and is even more rewarding when applied with the heightened index of suspicion allowed by the foregoing physical signs. Evidence of dominant mitral stenosis is almost always present. It appears that tricuspid stenosis occurs so uncommonly with dominant mitral insufficiency that the diagnosis of this combination should be considered with caution.\(^{9}\) The first heart sound at the cardiac apex and lower left sternal edge was either moderately accentuated or normal in 6 of the 13 cases. Absence of consistent, obvious accentuation may be related to the relatively frequent prolongation of P-R interval (fig. 9) associated with the right atrial enlargement\(^{25}\) of tricuspid stenosis. The first heart sound was often split (7 instances) probably due to concomitant delay in both mitral and tricuspid components (figs. 2 and 4). It is important to distinguish the tricuspid component of the first heart sound from a pulmonic ejection sound,\(^{32}\) which might occur with pulmonary hypertension. The ejection sound is high-pitched, clicking, best heard in the pulmonary area or subjacent intercostal space, and often decreases with inspiration and increases with expiration.

The second heart sound in tricuspid stenosis is important in two respects. Firstly, the intensity of the pulmonic component or the cumulative intensities of synchronous aortic and pulmonic components heard in the second left intercostal space should be used as evidence for or against pulmonary hypertension. Since aortic insufficiency is not uncommonly associated with tricuspid stenosis, the differentiation of the murmurs of aortic and pulmonic insufficiency should be re-emphasized. Secondly, in this series of cases respiratory splitting of the second heart sound occurred in only 3 instances. This suggests that in the presence of tricuspid valve obstruction the inspiratory augmentation of right ventricular filling may be impeded, and hence the inspiratory increase in right ventricular stroke volume and the inspiratory delay in pulmonary valve closure may not readily occur.\(^{33}\)

Following the second heart sound the tricuspid opening snap\(^{34}\) may be generated (fig. 2). Because the value of this sign has been emphasized,\(^{35}\) it was repeatedly sought for, but not convincingly appreciated by auscultation in any of our cases. It was, however, considered to have been recorded in two instances. Although the opening snap of the tricuspid valve can thus occasionally be recorded, its value in the clinical diagnosis of tricuspid stenosis is likely to be limited.

The most important auscultatory sign is the tricuspid stenotic murmur itself. Our studies suggest that in the majority of cases this murmur can be clearly distinguished from the accompanying murmur of mitral stenosis. Auscultation should begin at the cardiac apex, where the murmur of mitral stenosis is most evident, and proceed in small increments toward the lower left sternal edge. The mitral murmur will often fade between the apex and left sternal edge before the tricuspid murmur becomes evident. The area where the murmur of tricuspid stenosis should be sought is, then, along the lower left sternal edge, usually localized to the fourth or fifth intercostal spaces, but occasionally heard in the third interspace. It is imperative to emphasize this point, since it is still generally stated\(^{36}\) that the tricuspid area is along the right sternal edge or over the xiphoid. The anatomic position of the tricuspid valve, especially in the presence of right
atrial enlargement, together with the right-to-left direction of the jet across this stenotic valve, make the lower left sternal edge the appropriate area for auscultation of the murmur of tricuspid stenosis. Since the area in which the murmur is appreciated might be exceedingly limited, a careful geographic search of the region must be made.

In those cases of sinus rhythm the murmur was predominantly, if not exclusively, presystolic (fig. 3). A mid-diastolic component was either trivial or absent, although recordable with an intracardiac microphone in the right ventricle (fig. 5). Since the P-R interval is commonly prolonged in tricuspid stenosis, probably reflecting right atrial enlargement, the presystolic murmur is often remarkably discrete. The patient in this series in whom the auscultatory diagnosis of tricuspid stenosis was most difficult was the only one with a short P-R interval resulting in a very brief period between onset of presystolic murmur and first heart sound (fig. 9, parts 10a and 10b). In atrial fibrillation the tricuspid stenotic murmur begins earlier in diastole during the rapid flow phase (fig. 4). It may then continue with decreasing intensity up to the subsequent first heart sound.

The quality of the murmur of tricuspid stenosis was not uncommonly indistinguishable from that of the mitral stenotic murmur, but one occasionally got the impression that the tricuspid murmur was more superficial and higher pitched than the murmur of mitral stenosis. However, the only consistently reproducible way to distinguish these two stenotic murmurs was to analyze their respective variations in intensity during inspiration and expiration (figs. 3 and 4). Carvallo, in 1950, applied his observations on tricuspid insufficiency to the murmur of tricuspid stenosis, stating “the maneuver consists of making the patient inspire deeply and then hold the breath in post inspiratory apnea. This produces an increase in the intensity of the diastolic murmur.” It is this inspiratory augmentation of intensity that is the most valuable auscultatory sign in the diagnosis of tricuspid stenosis. It should be pointed out, however, that the most productive method of eliciting this sign is by listening while the patient is instructed to undertake moderately deep, slow, but continuous respiratory excursions. If the breath is held for any length of time in either inspiration, expiration, or in the respiratory mid-position, the sign may be lost. Although Carvallo’s basic observation is abundantly valid, it is during the active inspiratory effort that the murmur increases in intensity, and if the breath is held in inspiratory apnea the intensity may fade. The inspiratory augmentation may be of the magnitude of two or three grades, and not uncommonly may be grade III on inspiration but virtually inaudible on expiration. The intensity increase may be present for a series of cycles, or it may occur for only one or two beats at the very onset of inspiration. Since occasionally all auscultatory events increase with inspiration, it must be emphasized that it is the selective inspiratory augmentation of the murmur that is peculiar to tricuspid stenosis. This must occur without an increase in other acoustic events unless there is coexisting tricuspid insufficiency, in which case both tricuspid murmurs will wax and wane with respiration.

It has been stated that with the severest degree of tricuspid stenosis systemic venous pressure may already be so excessive that the inspiratory increase might be inconsequential, thus leaving the murmur uninfluenced by respiration. That this need not necessarily be a consequence of severity is suggested by observations in this and in other studies, indicating that the inspiratory increase in tricuspid gradient can be due solely to a fall in right ventricular diastolic pressure, the right atrial pressure remaining unaltered. The murmur of mitral stenosis either remains unchanged or gets fainter during inspiration. An exception to this might be the patient with mitral stenosis and marked sinus arrhythmia. With shortening of the diastolic filling period during the inspiratory increase in cardiac rate the mitral stenotic murmur might increase just as it increases when the rate is accelerated by other maneuvers.
casional instances have been reported in which the mitral stenotic murmur has increased with inspiration.\textsuperscript{10, 38} If it is assumed that tricuspid stenosis had not been overlooked in these cases and that this inspiratory augmentation was not associated with a sinus arrhythmia or accompanied by increased intensity of other auscultatory events, an explanation is not readily forthcoming. This discrepancy is, however, likely to be an uncommon occurrence.

The mechanism underlying the inspiratory increase in the murmur of tricuspid stenosis as opposed to the murmur of mitral stenosis was considered in the following fashion. All structures in the thorax should share equally the respiratory variations in intrathoracic pressure.\textsuperscript{39} Therefore, during inspiration as the pressure in the cardiac chambers decreases, the pressure in the right side of the heart falls below that in the extrathoracic systemic veins, but the pressure in the left side of the heart falls equally with that in the intrathoracic pulmonary veins. Hence, there is a selective inspiratory increase in systemic venous filling pressure and a selective inspiratory increase in right heart filling. This is thought to be the major cause of the increase in the right ventricular stroke volume that gives rise to the delay in pulmonary valve closure, resulting in normal inspiratory splitting of the second heart sound.\textsuperscript{40}

In tricuspid stenosis the inspiratory increase in right ventricular filling is altered by the valvular obstruction. When simultaneous right atrial and right ventricular pressure pulses are recorded through a double-lumen catheter, the transvalvular gradient is seen to increase during inspiration and to decrease during expiration (figs. 6 and 7). Since the inspiratory increase in gradient occurs in the presence of a constant orifice size, there must be an inspiratory increase in transvalvular flow, which is reflected in the inspiratory increase in tricuspid stenotic murmur.\textsuperscript{8, 41} As an augmented volume of blood enters the right atrium during inspiration, the stenotic lesion impedes its free entry into the right ventricle. Therefore, the pressure in the right atrium instead of falling in parallel with the diastolic pressure in the right ventricle, either falls only slightly, remains unchanged, or increases. These alterations were observed with either sinus rhythm or atrial fibrillation. When the respiratory fluctuations of intrathoracic pressure were abolished by opening the chest at operation, the respiratory effect on the tricuspid transvalvular gradient was abolished.

Further to establish that the diastolic stenotic murmur which possesses the property of selective inspiratory augmentation was located at the tricuspid orifice, 3 patients were studied by intracardiac phonocardiography (fig. 5). With the microphone lying in the right atrium no murmur was recorded, but when the microphone was advanced to a point just distal to the tricuspid valve, the stenotic murmur was grossly apparent, identifying it as tricuspid in origin. Respiratory tracings revealed that this murmur increased with inspiration and decreased with expiration in a fashion similar to what was considered to be the tricuspid stenotic murmur recorded by the thoracic wall microphone. In an application of the foregoing reasoning to the mitral valve, simultaneous left atrial and left ventricular pressures were recorded in the closed chest in 5 patients with mitral stenosis, 3 with sinus rhythm, and 2 with atrial fibrillation. The inspiratory fall and expiratory rise in intrathoracic pressure were reflected in these pulses with equal magnitude (fig. 8). Even though there were considerable respiratory changes in left ventricular diastolic and left atrial pressures, there was no change in transvalvular gradient. This is in accord with the observation that the mitral stenotic murmur does not selectively increase with inspiration.

Although the commonest cause of tricuspid stenosis is rheumatic fever (table 1), the lesion has been reported as a consequence of carcinoid,\textsuperscript{42} endocardial fibroelastosis,\textsuperscript{43} endomyocardial fibrosis,\textsuperscript{44} and systemic lupus erythematosus.\textsuperscript{3} In the presence of a normal valve the orifice may be obstructed by such a lesion as myxoma of the right atrium, which has
been reported to produce a murmur apparently identical in its respiratory variations with the murmur of organic tricuspid stenosis.\textsuperscript{45}

Not all tricuspid diastolic murmurs are caused by valvular obstruction. Flow murmurs across the tricuspid orifice may occur in atrial septal defect,\textsuperscript{21} but do not wax and wane with respiration. Occasionally rapid atrioventricular flow in the presence of tricuspid incompetence may generate a tricuspid diastolic murmur,\textsuperscript{46} a situation analogous to the short mitral diastolic murmur,\textsuperscript{47, 48} Fascinating in concept is what has been called the "right-sided Austin Flint" murmur in cases of pulmonary insufficiency.\textsuperscript{49} Finally, in some cases of right ventricular hypertrophy \textit{without} tricuspid incompetence a diastolic rumble has been described for reasons yet poorly defined.\textsuperscript{50}

The clinical diagnosis of tricuspid stenosis rests most securely on the physical signs. The electrocardiogram and x-ray were important, however, from confirmatory points of view. In evaluation of the large "A" wave in the jugular venous pulse it is necessary to exclude significant right ventricular hypertension. Absence of electrocardiographic evidence of right ventricular hypertrophy assists in this exclusion. The most consistent electrocardiographic pattern was the combination of negative evidence of right ventricular hypertrophy in lead V\textsubscript{1}, and positive evidence of right atrial enlargement in lead II (fig. 9). The most rewarding x-ray was the simple posteroanterior view (fig. 10). The combination of only slight to modest enlargement of the pulmonary artery segment associated with the contour of an enlarged right atrium along the lower cardiac silhouette was of value in suggesting that the right atrial enlargement was not due to significant pulmonary hypertension, and hence was of further help in assessing the large "A" waves.

In consolidation of the features in the foregoing discussion attention should be directed to the following points in the clinical recognition of tricuspid stenosis: (1) the patient is usually female, commonly considered to have rheumatic mitral stenosis; (2) there is a relative paucity of paroxysmal pulmonary symptoms, and occasionally the patient may be aware of the pulsating "A" wave in the neck; (3) examination of the jugular venous pulse in sinus rhythm reveals an abnormally large, often giant, "A" wave, a shallow "V" wave and gentle "Y" descent, or in atrial fibrillation reveals an elevated "V" wave with a gentle "Y" descent, these occurring in the absence of the physical, electrocardiographic, or radiologic signs of right ventricular hypertension; (4) dominant mitral stenosis is likely to be present, although the aortic valve may be either uninvolved, purely stenotic, purely insufficient, or a combination of both; (5) auscultation in the area along the lower left sternal edge will reveal principally or exclusively a presystolic murmur with sinus rhythm, or a mid-diastolic murmur with atrial fibrillation, each of which selectively increases with inspiration and decreases with expiration; auscultation of the second heart sound during active respiration generally will reveal absence of inspiratory splitting; (6) the electrocardiogram may reveal evidence of right atrial enlargement without right ventricular hypertrophy; and (7) a posteroanterior view of the chest may reveal only modest enlargement of the pulmonary artery segment in the presence of right atrial enlargement.

Summary

Clinical criteria for the recognition of tricuspid stenosis were studied in 13 cases, all personally observed by the authors. The results suggest that application of these criteria not only allows a confident diagnosis of tricuspid stenosis in a substantial majority of cases, but also allows the lesion to be recognized relatively early in its natural history.

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Addendum

Since this article was submitted for publication the authors have seen 2 more patients in whom tricuspid stenosis was diagnosed clinically and confirmed by cardiac catheterization. One patient, studied at Georgetown University Hospital, was a woman, age 42, with atrial fibrillation, coexisting mitral and aortic stenosis, and mild aortic regurgitation. The other case, studied at the National Heart Institute, was a woman, age 34, with sinus rhythm, coexisting mitral stenosis, and mild pure aortic regurgitation. The clinical and physiologic data in these additional cases were similar in all basic respects to the observations contained in this report.

Summario in Interlingua

Esseva studiate in 13 casos, omnes personalmente observate per le autores, le criterios clinic pro le recognition de stenosis tricuspidal. Le resultatos pare indicar le application del criterios assi trovate permitte non solmente le estabolimento del diagnose de stenosis tricuspidal con alte grades de confidentia in un forte majoritate del casos sed etiam le recognition del lesion a un periodo relativamente proecce de su historia natural.

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