Tricuspid Stenosis
Physiologic Criteria for Diagnosis and Hemodynamic Abnormalities

By Thomas Killip III, M.D., and Daniel S. Lukas, M.D.

Physiologic criteria for the diagnosis of tricuspid stenosis have not been satisfactorily established. In an attempt to define these criteria the pressure gradients across the tricuspid valve during right ventricular diastole were analyzed in 10 patients with tricuspid stenosis and compared with the gradients in a group of patients who did not have this lesion. The diagnostic importance of the mean gradient is emphasized. The hemodynamic abnormalities associated with the tricuspid stenosis are presented and discussed.

Lesions of the tricuspid valve are frequently unrecognized until they are associated with advanced signs and symptoms. The problems involved in the diagnosis of tricuspid insufficiency have recently been emphasized.

Similar difficulty prevails with regard to tricuspid stenosis. Information about the clinical and physiologic spectrum that can be produced by this disease is scant. Since surgical therapy is indicated in selected cases, the need for precision in diagnosis and evaluation of the functional severity of this lesion is apparent.

The present paper contains the data on 10 patients with tricuspid stenosis and emphasizes the hemodynamic criteria necessary for diagnosis and the physiologic alterations produced by this deformity. The clinical aspects of these patients are discussed in a separate communication.

Materials and Methods

In 9 of the 10 patients the diagnosis of tricuspid stenosis was suspected clinically, in the other it was made from the hemodynamic data and confirmed at autopsy. Of the 5 patients who were in normal sinus rhythm, the lesion was isolated in FC, combined with mitral stenosis in ER, RP, and JP, and combined with mitral and aortic stenosis in EH. All 5 of the patients with atrial fibrillation had associated tricuspid insufficiency and mitral stenosis and insufficiency. Of this group, SG and LR did not have aortic valvular disease; RR and JF had a double aortic lesion, and AN had aortic stenosis. JP was 14 weeks pregnant at the time of study. All 10 patients were female. Their ages ranged from 23 to 52 and averaged 37 years. Following diagnostic studies JP and RP underwent mitral valvuloplasty; SG had a mitral and a tricuspid valvuloplasty, and FC underwent a tricuspid valvuloplasty.

Cardiac catheterization was performed on all patients (table 1). It was repeated 13 months after tricuspid valvuloplasty in FC, following a 29-month interval in JF and after 39 months in RR. The technic of catheterization and the accessory analytic methods employed were as previously described. Pressures were measured with a Sanborn electromanometer and recorded by a 4-channel Polyviso recorder. In recording right atrial and ventricular diastolic pressures a sensitivity of 40 mm Hg per 4-cm deflection was utilized, since it provides records that are easily and accurately read and yet are free of the artifacts frequently recorded at higher sensitivities. Right ventricular and atrial pressures were determined in succession through a single-lumen catheter except in patient LR, in whom they were recorded simultaneously through a double-lumen catheter.

The mean pressures in right atrium and right ventricle during ventricular diastole and the gradient during flow across the tricuspid valve were determined by graphic integration—points read on the pressure curves for every 0.04 second of ventricular diastole were averaged. Sufficient cardiac cycles were analyzed to include an entire respiratory cycle. The peak of the R-wave in lead II of the simultaneously recorded electrocardiogram was used as a reference point to plot the beginning and end of ventricular diastole on the atrial curve. The ventricular pressure at right ventricular end-diastole (RVed) and the corresponding pressure point on the right atrial curve (so-called Z-point) were also analyzed.

The areas of the tricuspid and mitral valve orifices were calculated from the formula of Gorlin and Gorlin, with the recommended correction factors. The diastolic filling period was determined from the

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Table 1.—Data Obtained at Cardiac Catheterization in Ten Patients with Tricuspid Stenosis

<table>
<thead>
<tr>
<th>Patient</th>
<th>O2 Consumption ml/min./M.2</th>
<th>Cardiac index L./min./M.4</th>
<th>Cardiac rate</th>
<th>Pressures in mm. Hg</th>
<th>Pulmonary vascular resistance (dynes-sec.-cm.-2)</th>
<th>Mitral valve area (cm.2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>F. C.</td>
<td>R</td>
<td>1.91</td>
<td>88</td>
<td>6</td>
<td>15/5, 10</td>
<td>140</td>
</tr>
<tr>
<td></td>
<td>E</td>
<td>2.07</td>
<td>126</td>
<td>11</td>
<td>23/12, 15</td>
<td>130</td>
</tr>
<tr>
<td>F. C.*</td>
<td>R</td>
<td>1.72</td>
<td>90</td>
<td>7</td>
<td>17/8, 13</td>
<td>10</td>
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<tr>
<td></td>
<td>E</td>
<td>2.71</td>
<td>140</td>
<td>13</td>
<td>26/17, 22</td>
<td>100</td>
</tr>
<tr>
<td>E. R.</td>
<td>R</td>
<td>1.52</td>
<td>84</td>
<td>8</td>
<td>24/9, 14</td>
<td>136</td>
</tr>
<tr>
<td></td>
<td>E</td>
<td>1.90</td>
<td>112</td>
<td>17</td>
<td>32/16, 26</td>
<td>136</td>
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<tr>
<td>R. F.</td>
<td>R</td>
<td>2.48</td>
<td>68</td>
<td>5</td>
<td>24/9, 14</td>
<td>57</td>
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<tr>
<td></td>
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<td>2.53</td>
<td>88</td>
<td>7</td>
<td>35/20, 28</td>
<td>74</td>
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<tr>
<td>J. P.</td>
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<td>90</td>
<td>6</td>
<td>29/15, 21</td>
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<td>42/26, 33</td>
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<tr>
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<td>80</td>
<td>8</td>
<td>25/14, 26</td>
<td>97</td>
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<tr>
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<td>3.73</td>
<td>100</td>
<td>10</td>
<td>39/14, 26</td>
<td>146</td>
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<tr>
<td>S. G.</td>
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<td>1.69</td>
<td>110</td>
<td>21</td>
<td>45/27, 27</td>
<td>292</td>
</tr>
<tr>
<td></td>
<td>E</td>
<td>—</td>
<td>110</td>
<td>29</td>
<td>50/31, 42</td>
<td>—</td>
</tr>
<tr>
<td>R. R.</td>
<td>R</td>
<td>2.28</td>
<td>76</td>
<td>14</td>
<td>48/22, 30</td>
<td>165</td>
</tr>
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<td>18</td>
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<td>R. R.*</td>
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<td>85</td>
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<td>55/36, 45</td>
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<tr>
<td>A. N.</td>
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<td>100</td>
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<td>45/18, 27</td>
<td>263</td>
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<td>L. R.</td>
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<td>70</td>
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<td>45/18, 27</td>
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<td>—</td>
<td>32</td>
<td>19</td>
<td>67/—</td>
<td>—</td>
</tr>
<tr>
<td>J. F.</td>
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<td>1.76</td>
<td>92</td>
<td>9</td>
<td>57/28, 38</td>
<td>310</td>
</tr>
<tr>
<td>J. F.*</td>
<td>R</td>
<td>1.71</td>
<td>90</td>
<td>15</td>
<td>123/55, 71</td>
<td>1809</td>
</tr>
</tbody>
</table>

* Second Study  R: Rest  E: Exercise

right ventricular pressure tracing. For calculation of the tricuspid orifice area this interval was measured from the point during isometric relaxation at which right atrial and ventricular pressures were equal to the point during isometric contraction at which they again equalized (figs. 3 and 5).

Control Data

To determine the range in diastolic atrioventricular gradient that may be encountered in applying the present method to nonstenotic tricuspid valves, the catheterization data at rest of 14 patients with rheumatic heart disease who eventually came to autopsy and were demonstrated not to have tricuspid stenosis were reviewed. All had mitral stenosis and many had additional valvular lesions, including severe tricuspid insufficiency. Nine patients had atrial fibrillation, 5 normal sinus rhythm. The mean right atrioventricular diastolic gradient at rest was -0.1 mm. Hg (S.D. ± 1.0) with a range of -2.1 to +1.4 (fig. 1).

Since satisfactory studies during exercise were available on only 4 of the autopsied patients, atrioventricular gradients during exercise from an additional 7 patients with rheumatic heart disease who did not have any clinical stigmata of tricuspid stenosis3 were also determined. In the combined series of 11 patients the mean gradient was +0.5 mm. Hg (S.D. ± 1.1), with a range of -1.6 to +2.2 (fig. 1).

In the patients studied at rest the mean Z-RVed gradient was -1.0 (S.D. ± 1.1) mm. Hg, with a range of -3.8 to +2.4. During exercise the mean Z-RVed gradient was -2.3 mm. Hg (S.D. ± 1.9), with a range of -4.8 to +1.0.

Results

Atrioventricular Gradient

At rest the mean right atrial pressure during ventricular diastole significantly exceeded the mean right ventricular diastolic pressure in all 10 patients with tricuspid stenosis (table 2). In general, the gradients were small, ranging from 2.9 to 11.9 mm. Hg (fig. 1). In 9 of the 10 patients it was less than 7 mm. Hg; in 4 it was less than 5 mm. Even the smallest gradients, however, were readily appreciated by gross inspection of the curves* when the pressures in the right atrium and ventricle were recorded at

* When records for the control series were reviewed a previously unrecognized gradient in the tracings of JF was visually apparent. Examination of the post-mortem specimen revealed a moderately severe stenosis of the tricuspid valve. This experience added considerable confidence in the validity of the present methods.
the same sensitivity (fig. 2). The largest gradients at rest were noted in the patients with atrial fibrillation, all of whom had associated tricuspid insufficiency.

The manner in which the gradient varied during the course of ventricular diastole depended on the rhythm. In normal sinus rhythm it was usually small early in diastole, but became considerable during atrial contraction, which produced a pressure wave of exceptionally large amplitude (figs. 2 and 3). In atrial fibrillation a constant level of right atrial pressure during diastole and an early diastolic dip in right ventricular pressure gave rise to a gradient that was largest early in diastole (figs. 4 and 5).

During exercise the gradient increased in every patient in whom adequate studies were available (table 2, fig. 6). The increase resulted from a rise in mean atrial pressure, an augmented amplitude of atrial contraction when present, and an accentuated dip in right ventricular pressure early in diastole.

Respiration often produced striking effects on the mean diastolic gradient, which was maximal during inspiration and minimal during expiration (fig. 7). In general, right atrial pressure varied little with the phases of respiration, whereas right ventricular diastolic pressure fell with inspiration and rose with expiration.

Right atrial pressure at the Z-point was higher than the RVed pressure in all patients, but did not provide an index of the level of the mean diastolic gradient. Indeed in 4 patients, including FC, who required subsequent tricuspid valvuloplasty, this difference was no greater than in the control series. Exercise increased the Z-RVed difference in all but 1 patient. This marked disparity in pressures at the onset of right ventricular contraction was especially striking in those with normal sinus rhythm and was caused by the onset of ven-
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Note

Functional systole curves of tricuspid m. Hg traction.

In patients with normal sinus rhythm the atrial V-wave was very small, but the fall in atrial pressure when the tricuspid valve opened appeared to be normal. The rate of decline of the regurgitant wave in those patients with associated insufficiency did not differ sufficiently from that observed in the control series to be useful as a criterion of tricuspid stenosis.

Tricuspid Orifice Area

The calculated area of the tricuspid orifice ranged from 0.7 to 1.7 cm² in the 5 patients with normal sinus rhythm (table 2). The areas calculated at rest and during exercise checked within 0.2 cm². Tricuspid valvuloplasty was performed on FC, in whom the calculated area was 1.1 cm². At operation an experienced cardiac surgeon estimated the area as “not more than 1 square centimeter.”

Less reliance can be placed upon the valve areas in patients with associated tricuspid insufficiency, since the rate of regurgitant flow and therefore the total forward flow across the valve are not known. Tricuspid valvuloplasty was recommended and performed in SG because the clinical and hemodynamic observations indicated a predominant tricuspid stenosis and the calculated area of 0.6 cm² was therefore considered reasonably reliable. At operation the valve area was estimated by the surgeon to be between 0.5 and 1.0 cm² and only a slight regurgitant stream was noted.

The cardiac output at rest was reduced in all but 1 patient (table 1). The single exception, JP, was 14 weeks pregnant at the time of study. Although her output was within the accepted normal range, it may possibly be abnormal when compared to the reported values for the first trimester of pregnancy. The increase in cardiac output with exercise was subnormal for the degree of work per-
formed, as judged by oxygen consumption, in all but 1 patient, EH.

There was a significant correlation (r = 0.90, p < .01) between tricuspid valve area per square meter of body surface and the cardiac index (fig. 8). The data include those patients who had evidence of tricuspid insufficiency on the atrial pressure curve. This good correlation may indicate either that these patients had relatively little insufficiency, so that the valve area calculations were reasonably accurate, or that the valve area as calculated gives a good estimate of the reduction in output secondary to combined tricuspid stenosis and insufficiency. The correlation was less significant (r = -0.5 or less) when the valve size per square meter was compared to each of the following variables: mean gradient, log of the mean gradient, right atrial mean pressure, and right atrial mean pressure during ventricular diastole.

**Associated Hemodynamic Abnormalities**

The associated abnormalities delineated by cardiac catheterization were to a large extent a reflection of the accompanying multivalvular

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**TABLE 2.—Data Pertaining to Pressure Gradients in mm. Hg, Flows and Valve Areas in Ten Patients with Tricuspid Stenosis**

<table>
<thead>
<tr>
<th>Patient</th>
<th>State</th>
<th>Atrial diastolic mean</th>
<th>Ventricular diastolic mean</th>
<th>Atrialventricular gradient</th>
<th>Flow, ml./diastolic sec.</th>
<th>Tricuspid valve area cm.²</th>
<th>Ventricular end-diastolic</th>
<th>Atrial Z point RVed difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>F. C.</td>
<td>R</td>
<td>6.3</td>
<td>1.6</td>
<td>4.7</td>
<td>103</td>
<td>1.1</td>
<td>3.2</td>
<td>1.1</td>
</tr>
<tr>
<td>F. C.*</td>
<td>R</td>
<td>9.5</td>
<td>5.4</td>
<td>4.4</td>
<td>81</td>
<td>0.9</td>
<td>6.6</td>
<td>-2.9</td>
</tr>
<tr>
<td>E. R.</td>
<td>R</td>
<td>8.6</td>
<td>2.1</td>
<td>6.5</td>
<td>73</td>
<td>0.7</td>
<td>4.2</td>
<td>2.0</td>
</tr>
<tr>
<td>R. P.</td>
<td>R</td>
<td>5.8</td>
<td>2.9</td>
<td>2.9</td>
<td>93</td>
<td>1.2</td>
<td>1.8</td>
<td>3.2</td>
</tr>
<tr>
<td>J. P.</td>
<td>R</td>
<td>7.4</td>
<td>3.8</td>
<td>3.6</td>
<td>108</td>
<td>1.5</td>
<td>4.2</td>
<td>0.2</td>
</tr>
<tr>
<td>E. H.</td>
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<td>12.1</td>
<td>4.4</td>
<td>7.7</td>
<td>169</td>
<td>1.3</td>
<td>3.4</td>
<td>9.4</td>
</tr>
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<td>6.8</td>
<td>220</td>
<td>1.7</td>
<td>8.0</td>
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<td>9.3</td>
<td>11.9</td>
<td>121</td>
<td>0.6</td>
<td>7.1</td>
<td>12.7</td>
</tr>
<tr>
<td>R. R.*</td>
<td>R</td>
<td>17.0</td>
<td>7.8</td>
<td>9.2</td>
<td>119</td>
<td>0.9</td>
<td>10.8</td>
<td>6.0</td>
</tr>
<tr>
<td>A. N.</td>
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<td>17.6</td>
<td>11.0</td>
<td>6.6</td>
<td>132</td>
<td>1.2</td>
<td>12.6</td>
<td>4.4</td>
</tr>
<tr>
<td>L. R.</td>
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<td>4.2</td>
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<td>158</td>
<td>1.4</td>
<td>8.4</td>
<td>3.2</td>
</tr>
<tr>
<td>E</td>
<td>E</td>
<td>15.7</td>
<td>8.0</td>
<td>7.7</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>J. F.</td>
<td>R</td>
<td>8.6</td>
<td>3.5</td>
<td>5.1</td>
<td>78</td>
<td>0.8</td>
<td>4.5</td>
<td>2.1</td>
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<tr>
<td>J. F.*</td>
<td>R</td>
<td>12.3</td>
<td>7.8</td>
<td>4.5</td>
<td>84</td>
<td>0.9</td>
<td>10.0</td>
<td>1.3</td>
</tr>
</tbody>
</table>

* Second study  R: Rest  E: Exercise

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**TABLE 3.—Hemodynamics in Tricuspid Stenosis: Comparison of Eight Patients with Pure Mitral Stenosis and Normal Rhythm to Three Patients with Mitral and Tricuspid Stenosis and Normal Rhythm**

<table>
<thead>
<tr>
<th></th>
<th>Mitral stenosis mean ± S.D.</th>
<th>Mitral and tricuspid stenosis (mean ± S.D.)</th>
<th>Significance of difference between means (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitral valve area, cm²/M² B.S.A.</td>
<td>0.72 ± 0.02</td>
<td>0.72 ± 0.01</td>
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<tr>
<td>Cardiac output, L/min./M² B.S.A.</td>
<td>3.03 ± 0.54</td>
<td>2.35 ± 0.83</td>
<td>&lt;.05 &gt; .02</td>
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<tr>
<td>Cardiac output, ml./diastolic sec./M² B.S.A.</td>
<td>90 ± 13</td>
<td>62 ± 17</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Pulmonary artery mean pressure, mm. Hg.</td>
<td>35 ± 5.5</td>
<td>16.3 ± 4.0</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Pulmonary “capillary” mean pressure, mm. Hg.</td>
<td>23 ± 2.5</td>
<td>12.3 ± 3.2</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Pulmonary vascular resistance, dynes-sec.-cm.⁻¹</td>
<td>270 ± 83</td>
<td>93 ± 40</td>
<td>&lt;.05 &gt; .02</td>
</tr>
</tbody>
</table>
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FIG. 6 Top. Relation between rate of blood flow (ordinate) and mean gradient (abscissa) across tricuspid valve at rest and during exercise in tricuspid stenosis. Calculated area of tricuspid orifice is included for each patient. In each instance gradient and flow increased during exercise.

FIG. 7 Middle. Variation during a respiratory cycle in mean diastolic gradient across tricuspid valve in patient (ER) with tricuspid stenosis. Points connected by a vertical line represent mean pressures (ordinate) in right atrium (RA) and ventricle (RV) for a single diastolic interval. Note relatively constant atrial pressure. During inspiration RV pressure falls and gradient increases; with expiration pressure rises and gradient decreases.

FIG. 8. Bottom. Relation between calculated tricuspid valve area per square meter (abscissa) and cardiac index (ordinate) in 10 patients with tricuspid stenosis. r = 0.90.

disease (table 1). Pulmonary "capillary" and arterial hypertension were present at rest or during exercise in all 9 patients with multivalvular disease. Pulmonary vascular resistance was normal in 5. Of the 4 patients with mitral stenosis as the only associated lesion 3 were in normal sinus rhythm and had mitral valve indices of 0.7 cm²/M² of body surface. These 3 patients were compared statistically to a series of 8 patients with a similar degree of mitral stenosis, normal sinus rhythm, and no other valvular involvement. The data, tabulated in table 3, demonstrate that the cardiac index, flow per diastolic second, pulmonary artery mean pressure, pulmonary "capillary" pressure, and pulmonary vascular resistance were significantly lower in the patients with stenosis of both mitral and tricuspid valves.

In patient SG, who had atrial fibrillation, a similar modifying effect of the tricuspid lesion on the hemodynamic alterations produced by mitral stenosis is apparent. She had less elevation of pulmonary vascular pressures and resistance than would be expected for the degree of mitral stenosis present (table 1).

Sequential Studies

Three patients were catheterized twice (tables 2 and 3). The data of RR obtained 39 months apart are virtually identical. In JF a 6-fold increase in pulmonary vascular resistance occurred in 29 months and pulmonary arterial pressure rose markedly although the cardiac index remained the same. This patient died 6 months after the second study and the clinical and hemodynamic diagnoses were confirmed at necropsy. In each of the 2 patients the estimated tricuspid valve orifice areas derived from the 2 different studies agreed within 0.1 cm² (table 2). Studies repeated on FC 13 months following tricuspid valvuloplasty revealed the functional orifice to be unchanged although at operation 1 commissure had apparently been fractured successfully. However, in contrast to the first study, cardiac output increased...
appropriately with exercise. The elevated pulmonary “capillary” pressure present on the second study is interpreted as possible evidence of an elevated left ventricular diastolic pressure secondary to myocardial insufficiency, since the patient had no clinical or radiographic evidence of mitral or aortic valvular involvement.

**DISCUSSION**

*Physiologic Criteria for Diagnosis*

Although the tricuspid valve is ideally located for study by catheterization of the right heart, there has not been agreement about the hemodynamic criteria for the diagnosis of stenosis of this valve. Ferrer and co-workers, reporting 2 cases, emphasized the gradient across the valve at the onset of right ventricular systole. McCord, Swan, and Blount reporting 3 cases, only 1 of whom had surgically significant stenosis, stated “... the increase in the early diastolic gradient is the most dependable hemodynamic characteristic of surgical tricuspid stenosis.” In Gibson and Wood’s study of 12 cases the largest gradient across the valve at any moment during ventricular diastole was utilized as the evidence for stenosis.

On the basis of hydrodynamic principles Gorlin and Gorlin have defined the variables that influence the flow of blood across stenotic valves. They clearly emphasized the importance of the mean pressure gradient across an obstructed valve and indicated its relationship to the rate of flow and the size of the orifice.

Use of the mean gradient throughout the period of ventricular filling as the criterion for the diagnosis of tricuspid stenosis avoids pitfalls inherent in emphasizing any single point on the pressure curves during ventricular diastole. An instantaneous gradient is not only subject to several limitations as a diagnostic criterion but cannot be related readily to other physiologic parameters. A gradient during early diastole is particularly unreliable, since a 3 to 4 mm Hg difference between right atrium and ventricle at this point in the cardiac cycle has been noted in our laboratory in patients with rheumatic heart disease proved at autopsy not to have tricuspid stenosis. A similar difference between left atrial and ventricular pressures early in diastole has been observed in pure mitral insufficiency. A gradient at the end of diastole (Z-RVed) greater than 1.2 mm (2 standard deviations on the positive side of the mean value), as the control data from patients at rest indicate, would be unlikely in the absence of tricuspid stenosis; however, several patients in the present study had normal Z-RVed differences in spite of tricuspid stenosis. Any momentary gradient may be produced by artifacts, such as overshoot, in the recorded pressures. Such artifacts are minimized by integration of the pressures throughout diastole. Finally, in stenosis with atrial fibrillation a salient momentary gradient may be absent, despite a small but significant mean gradient.

When records are obtained and analyzed by the present method, a mean gradient greater than 1.9 mm Hg at rest and 2.6 mm during exercise (2 standard deviations on the positive side of the mean control value) would be most unlikely in patients with rheumatic heart disease without tricuspid obstruction. The gradients observed in tricuspid stenosis (table 2) are smaller than the left atrioventricular pressure differences noted in tight mitral stenosis, which is usually associated with a pulmonary “capillary” pressure of around 28 mm Hg and a resulting gradient of 23 mm (a left ventricular mean diastolic pressure of 5 mm Hg is assumed). The largest resting gradient across the tricuspid valve in the present series was only 11.9 mm Hg. The most obvious explanation for this difference is that the tricuspid orifices are larger than the mitral orifices. An additional factor is the lower cardiac output in tricuspid stenosis than in uncomplicated mitral stenosis (table 3). For any given degree of stenosis of an atrioventricular valve the smaller the blood flow the smaller the gradient across the valve.

Exercise provides an excellent test for evaluating a small resting gradient. There was some uncertainty about the significance of the 2.9 mm Hg value in RP at rest, but the prompt rise to 5.6 mm during exercise confirmed the
presence of tricuspid stenosis (fig. 6). The invariable increase in gradient is due to the augmentation of flow across the valve resulting from both the rise in cardiac output and the decrease in diastolic filling period that are associated with exercise. A greater pressure head is required to drive more blood in less time across the stenotic valve.

A stenotic ativoventricular valve, unlike the normal valve, probably opens during ventricular isometric relaxation and closes during ventricular isometric contraction. It is preferable to analyze the gradient throughout the period of functional rather than true diastole, which is set-off between the 2 points at which right atrial and ventricular pressures are identical (figs. 3 and 5). Sufficient cardiac cycles must also be analyzed to cover at least 1 complete respiratory cycle, since the gradient varies with alterations in intrathoracic pressure (fig. 7).

The accentuation of the murmur of tricuspid stenosis with inspiration, a phenomenon described by Rivero Carvallo,14 is a reflection of the increase in gradient and flow that occurs with inspiration. In this lesion the right ventricular pressure follows the change in intra thoracic pressure (fig. 7), falling during inspiration and rising during expiration. The atrial pressure, however, remains relatively constant, probably because the increased venous return into the atrium with inspiration exceeds the augmented outflow across the stenotic valve. The change in the intensity of murmurs over the tricuspid area with inspiration is a sign of cardinal diagnostic importance in patients with rheumatic heart disease.5, 15

The manner in which the gradient varied during the course of ventricular filling in atrial fibrillation and sinus rhythm was clearly reflected in the auscultatory phenomena.3 The thrills and murmurs over the tricuspid area in atrial fibrillation were early and middiastolic; in normal sinus rhythm they were presystolic. During these phases of the cardiac cycle the gradients were maximal and were probably associated with greatest and most turbulent flow across the stenotic valve.

Critique of Orifice Calculation

Calculation of the functional orifice of the tricuspid valve by the formula of Gorlin and Gorlin6 is useful in evaluating the degree of obstruction from a set of measured values for flow and gradient. Data are not available to determine precisely the correction factor for turbulence and orifice contraction needed in the calculation, but certain information suggests that the value of 1.0 as originally proposed6 is satisfactory. Ferrer and co-authors10 demonstrated that the area determined by planimetry of a scale postmortem photograph of the valve in a case of tricuspid stenosis agreed within 0.1 cm.2 of the calculated area. In the 2 patients from the present series who underwent tricuspid valvuloplasty and in the single case reported by Gorlin and Gorlin the calculated area agreed well with the estimate made from digital palpation by the surgeons, although it must be conceded that the surgeon’s finger is not a precise planimeter.

The calculated area should be considered as a functional rather than an exact anatomic size, but this does not detract from the usefulness of the calculation by the Gorlin formula. Extensive experience has indicated that the formula frequently aids greatly in the preoperative and postoperative evaluation of patients with mitral stenosis. The data from the patients with tricuspid stenosis indicate its value in this lesion. The close correspondence between the areas at rest and during exercise (table 2) is impressive evidence of its reliability in relating flow and gradient to valve size. Without calculation of valve area it would be difficult to compare the physiologic data from one patient to the data from another or to compare repeated studies in the same patient. This is well illustrated by the postoperative study in FC (tables 1 and 2).

When there is associated tricuspid insufficiency, calculations of the orifice area yield falsely small values, since the rate of forward flow across the valve is greater than the cardiac output by an amount related to the regurgitant flow, which is unknown. When methods become available to measure the regurgitant flow,16
more precise estimations of the degree of stenosis and insufficiency will be possible.

Effects of Tricuspid Stenosis on Dynamics of Mitral Stenosis

The hemodynamic data of the 3 patients with combined mitral and tricuspid stenosis were significantly different from a group of patients with uncomplicated mitral stenosis of comparable severity (table 3). The lower pulmonary vascular pressures in the group with tricuspid stenosis are secondary to the lower flow and pulmonary vascular resistance. It is possible that the hemodynamic evidence of mitral stenosis might be obscured at rest by the effects of tricuspid stenosis. Such was the case in RP (table 1), in whom pulmonary venous hypertension was observed only during exercise. An important implication of these observations is that patients with tricuspid stenosis must be studied completely at rest and during exercise before the presence of significant mitral stenosis can be ruled out.

Major Hemodynamic Alterations

Tricuspid stenosis produces 2 major hemodynamic alterations, a reduction in cardiac output and an increase in right atrial pressure. Both these effects on cardiac function are of importance in the production of the major symptoms, fatigue and edema, and must be considered in attempts to define a "critical" area, if one exists, for the tricuspid valve.

The data from FC illustrate the ability of tricuspid stenosis alone to reduce cardiac output (table 1 and fig. 8). This reduction in output is due to the inability of the right atrium to propel blood past the obstructed valve at the normal rate. When tricuspid and mitral stenosis occur together, the cardiac output is lower than would be expected on the basis of the existing degree of mitral stenosis alone (table 3). The probable explanation is that the elevated pressure necessary to maintain flow across the obstructed mitral valve is created to a great extent by the pumping action of the right ventricle, whereas only the right atrium performs this function for the tricuspid valve. In obstruction of the tricuspid valve, therefore, blood flow is limited proximal to the right ventricle. Restriction of blood flow at rest and during exercise is probably the outstanding factor responsible for fatigue. The regression formula derived from the data in figure 8 suggests that a tricuspid orifice smaller than 1.0 cm.$^2$/M.$^2$ of body surface may cause reduction of cardiac output. It would be of great interest to evaluate the pumping action of the right atrium by comparing the hemodynamic effects of normal rhythm and atrial fibrillation in a patient with tricuspid stenosis.

The level of atrial pressure in tricuspid stenosis is influenced by the diastolic pressure in the right ventricle, and the systolic insufficiency wave, if one is present, as well as by the gradient. The mean diastolic gradient depends on the flow per diastolic second and the valve size. At any particular rate of flow the smaller the orifice area, the larger is the gradient. Similarly for any given degree of stenosis, the larger the flow, the larger is the gradient.

Elevation of right atrial pressure and consequently of pressures throughout the systemic venous bed is a potent factor in the production of edema. In this regard it is pertinent that the incidence of edema and ascites in patients with tricuspid insufficiency is 7 times greater when the right atrial mean pressure (measured in the edema-free state) is above 10 mm. Hg than when it is below this level. Since the diastolic gradient across the stenotic valve is only 1 of the factors responsible for elevation of right atrial pressure, it is not possible to select a valve area that might be likely to be associated with edema, particularly in patients with multivalvular disease. However, if right ventricular mean diastolic pressure is normal (1–2 mm. Hg) and tricuspid insufficiency is not present, a right atrial pressure in excess of 10 mm. Hg would be required to maintain a normal resting output across a tricuspid orifice smaller than 0.7 cm.$^2$/M.$^2$ body surface or 1.3 cm.$^2$ for a patient of average size (1.7 M.$^2$).

Surgical Implications

A practical application of the hemodynamic data is in the selection of patients who may benefit from tricuspid valvuloplasty. The
importance of the mean diastolic gradient in the diagnosis of tricuspid stenosis by physiologic means has been emphasized and the range of gradient in the absence of tricuspid obstruction has been defined for this laboratory. Since absolute values will vary with the techniques of study, the range of normal must be determined for the individual laboratory. If significant abnormalities in gradient and flow across the tricuspid valve are found, surgery may be indicated. In interpreting the data the influence of coexisting valvular lesions and the state of myocardial function must be considered. The final decision to perform surgery should be made only after a complete and multidisciplined clinical and hemodynamic appraisal of the patient.

SUMMARY

Cardiac catheterization in 10 patients with tricuspid stenosis revealed an abnormal mean pressure gradient from right atrium to ventricle during ventricular diastole. The gradient was significantly in excess of the difference of $-0.1 \pm 1.0$ mm Hg at rest and $+0.5 \pm 1.1$ mm Hg during exercise found in patients with rheumatic heart disease and no tricuspid stenosis. An elevated mean gradient, widening with exercise, was considered the most reliable physiologic criterion for the diagnosis of tricuspid stenosis.

The difference in pressures across the stenotic tricuspid valve increased with inspiration and decreased with expiration. It was largest during atrial contraction in normal sinus rhythm and early in diastole in atrial fibrillation.

The major effects of the lesion on the circulation were a restriction of cardiac output and elevation of right atrial pressure. The decrease in cardiac output correlated well with the degree of constriction of the tricuspid orifice as calculated by the formula for valve area. The cardiac output, pressures and resistance in the pulmonary vascular bed were lower in 3 patients with associated mitral stenosis when compared to a group of patients with a similar degree of mitral stenosis but no involvement of the tricuspid valve.

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SUMMARIO IN INTERLINGUA

Catheterisation cardiac in 10 patientes con stenosis tricuspide revelava anormal valores medie del gradient de pression inter le atrio e le ventriculo dextere durante le diastole ventricular. Le gradient excedeva significativamente le correspondente valor de $-0.1 \pm 1.0$ mm Hg in statu de reposo e de $+0.5 \pm 1.1$ mm Hg in statu de exercitio trovate in patientes con rheumatic morbo cardiac sed sin stenosis tricuspide. Un elevate valor medie del gradiente, crescente con exercitio, eseva considerate como le plus secur criterio physiologic in le diagnose de stenosis tricuspide.

Le differencia transvalvular del pressiones observate in casos de stenotic valvulas tricuspid se augmentava con inspiration e se reduciva con expiration. Illo eseva le plus pronunciate durante le contraction atrial in normal rhythm sinusal e durante le prime phases diastolic in fibrillation atrial.

Le major effectos del lesion super le circulation eseva un restriction del rendimento cardiac e un elevation del pression dexteroatrial. Le reduction del rendimento cardiac eseva ben correlationate con le grado de constriction del orificio tricuspide calculate per medio del formula pro le area valvular. Le rendimento cardiac e le pression e resistentia in le vasculatura pulmonar eseva plus basse in 3 patientes con associate stenosis mitral que in un gruppo de patientes con simile grados de stenosis mitral sed sin affection del valvula tricuspide.

REFERENCES


3 Killip, T., III, and Lukas, D. S.: Clinical features of tricuspid stenosis. To be published.

Science has taught me the opposite lesson. She warns me to be careful how I adopt a view which jumps with my preconceptions, and to require stronger evidence for such belief than for one to which I was previously hostile.—Huxley.
Tricuspid Stenosis: Physiologic Criteria for Diagnosis and Hemodynamic Abnormalities
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