Isolated Tricuspid Insufficiency

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SUMMARY
Isolated tricuspid insufficiency can be a well-tolerated lesion, as illustrated by a 32-year follow-up in one patient reported here. A second case of recent onset is also described. The 13 reported cases of isolated tricuspid insufficiency due to trauma can be divided into two groups: ruptured papillary muscle or ruptured chordae tendineae and/or valve. All patients with ruptured papillary muscle were dead or had surgery within 4 months after trauma. The earliest surgery in the chordae tendineae group was 1½ years after trauma. Physical findings in isolated tricuspid insufficiency are distinctive; the electrocardiogram usually shows incomplete right bundle-branch block, and the chest roentgenogram shows cardiomegaly. Indicator-dilution curves from the right atrium and pulmonary artery are useful for confirmation of the diagnosis. Surgery is indicated when papillary muscles are ruptured, but conservative treatment is probably usually indicated in the other patients.

Additional Indexing Words:
Cardiac catheterization  Cardiac surgery  Prosthetic heart valves  Trauma

Isolated tricuspid insufficiency is an uncommon, but often well-tolerated lesion. All reported cases of traumatic tricuspid insufficiency\textsuperscript{1-10} are reviewed, and two additional cases are reported, with one patient alive and well 32 years after the onset of isolated tricuspid insufficiency.

Case Summaries

Case 1
E.W., a 70-year-old retired Navy Chief Petty Officer continues to work full-time and is asymptomatic. In 1938, his anterior chest was thrown with considerable force against the steering wheel of his car. The patient himself noted markedly pulsatile neck veins, which had not been present before the accident. In 1966, one episode of atrial flutter (with shortness of breath) was successfully converted with digoxin and quinidine to a normal sinus rhythm with no recurrences.

Physical Examination
Jugular venous pressure was moderately elevated, with very prominent v waves. The first heart sound was decreased in intensity. A soft fourth heart sound was audible only with inspiration along the lower left sternal border. A grade 2/6 holosystolic murmur was loudest at the lower sternal border and increased to grade 3/6 with inspiration. With the Valsalva maneuver the murmur disappeared; upon release of the Valsalva maneuver, the murmur returned almost immediately. The liver was palpable 3 cm below the right costal margin with slight systolic pulsation. There was no edema or ascites, and the remainder of the physical examination was normal.

A phonocardiogram with jugular venous pulse tracing is shown in figure 1, an electrocardiogram in figure 2, and a chest roentgenogram in figure 3. (Right atrial enlargement was suspected on the chest roentgenogram, but pericardial effusion and Ebstein's anomaly were additional considerations).

Cardiac Catheterization
The right atrial pressure was elevated, with an 18-mm Hg v wave. The right ventricular end-diastolic pressure was 14 mm Hg. The pulmonary artery pressure was 31/18 mm Hg. There was no gradient across the tricuspid or pulmonic valve. Simultaneous recording of pressures and intracardiac electrocardiograms demonstrated simulta-
neous change of right ventricular pressure and electrical potential to right atrial pressure and electrical potential. A pulmonary artery indicator-dilution curve, with tricarbocyanine green dye, was normal. However, right ventricular and right atrial dye curves revealed prolonged disappearance slopes consistent with marked tricuspid insufficiency. An intracardiac phonocatheter localized the systolic murmur to the tricuspid valve. Also, a mid-diastolic murmur was recorded, which was considered consistent with a diastolic flow murmur across the tricuspid valve. A left-to-right shunt was excluded. A right ventricular cineangiogram revealed marked reflux of contrast material into an enlarged right atrium. The tricuspid valve was not displaced downward into the right ventricle.

Case 2

T.L., a 24-year-old, active duty, Navy man was thrown from a motorcycle in March, 1969, sustaining nonpenetrating chest trauma. Immedi-
right ventricle to right atrium demonstrated simultaneous change from right ventricle pressure and electrical potentials to right atrial pressure and electrical potentials. No left-to-right shunt was detected by oxygen saturation data. Left heart catheterization was normal. A pulmonary artery dye curve was normal. However, indicator-dilution curves with tricarbocyanine green dye injected into the right atrium or right ventricle revealed prolonged disappearance slopes, consistent with marked tricuspid regurgitation. A right ventricular cineangiogram revealed marked reflux of contrast material into the right atrium; the tricuspid annulus was in a normal position.

Discussion

There are 15 reported cases\(^1\text{e}-10\) of isolated traumatic tricuspid insufficiency, including our two cases (table 1).\(^1\text{e}-10\) These cases can be divided into two groups: (1) ruptured papillary muscle\(^1\text{e}-5\) and (2) ruptured chordae tendineae and/or valve,\(^5\text{e}-10\) on the basis of time from trauma to surgery and/or death. The five patients with ruptured papillary muscle were either dead or had operations less than 4 months posttrauma. The earliest surgery in the ruptured chordae tendineae group was 1½ years posttrauma. The latter group generally followed a benign course as emphasized by the 70-year-old man reported here, who we assume has ruptured chordae tendineae since he is alive and asymptomatic 32 years after the onset of traumatic tricuspid insufficiency.

Etiology and Pathophysiology

Most of the reported cases have been due to automobile accidents.\(^2\text{e}, 5, 7\text{e}-10\) Any violent compression of the heart during diastole, associated with some obstruction to great vessel outflow, can cause rupture of the chordae tendineae or papillary muscle with resultant insufficiency.

Symptoms

Symptoms are frequently absent or mild, and onset of symptoms may be delayed, especially in the chordae tendineae group. In the ruptured papillary muscle group, symptoms usually begin soon after the trauma, but two patients in the ruptured chordae tendineae group also had onset of symptoms immediately after trauma.\(^7\text{e}, 8\) One patient in

Figure 3
Chest X-ray—cardiac enlargement with a globular contour, probable right atrial enlargement.

right after the accident a murmur was noted which was not present previously. In addition, an electrocardiogram showed nonspecific ST-segment and T-wave changes (considered due to myocardial contusion). When he was reevaluated in September, 1969, he was asymptomatic.

Physical Examination

Jugular venous pressure was elevated, with prominent v waves. A grade 3/6 holosystolic murmur was heard over the lower sternal border; the murmur became louder with inspiration. After a Valsalva maneuver, the murmur returned almost immediately. The remainder of the physical examination was normal.

Electrocardiograms showed an incomplete right bundle-branch block. Chest roentgenograms were normal.

Cardiac Catheterization

The right atrial pressure was elevated, with a v wave of 18 mm Hg. The right ventricular end-diastolic pressure was elevated to 15 mm Hg. The pulmonary artery pressure was 25/13 mm Hg. There was no gradient across the tricuspid or pulmonic valve. Simultaneous intracardiac electrocardiogram and pressure recording from the
Table 1

Reported Cases of Traumatic Tricuspid Insufficiency

<table>
<thead>
<tr>
<th>Case</th>
<th>Author</th>
<th>Age</th>
<th>Sex</th>
<th>Time from trauma to surgery, autopsy, or follow-up</th>
<th>RA wave (mm Hg)</th>
<th>RV wave (mm Hg)</th>
<th>Pathology</th>
<th>Procedure</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Kleberger¹</td>
<td>?</td>
<td>M</td>
<td>Immediate</td>
<td></td>
<td></td>
<td>Rupt pap muscle</td>
<td></td>
<td>Died</td>
</tr>
<tr>
<td>2</td>
<td>Parmley et al.²</td>
<td>28</td>
<td>M</td>
<td>4 months</td>
<td></td>
<td></td>
<td>Rupt pap muscle</td>
<td>Repair</td>
<td>Died 12 hr p.o.</td>
</tr>
<tr>
<td>3</td>
<td>Osborn et al.³</td>
<td>33</td>
<td>M</td>
<td>3 months</td>
<td>19.5</td>
<td>20/5</td>
<td>Rupt pap muscle</td>
<td>Repair</td>
<td>Improved</td>
</tr>
<tr>
<td>4</td>
<td>Aleksandrow et al.⁴</td>
<td>53</td>
<td>M</td>
<td>6 weeks</td>
<td>8</td>
<td>13/0</td>
<td>Rupt pap muscles</td>
<td></td>
<td>Died after cath</td>
</tr>
<tr>
<td>5</td>
<td>Jahnke et al.⁵</td>
<td>30</td>
<td>M</td>
<td>2 months</td>
<td>19</td>
<td>20/9</td>
<td>Rupt pap muscles</td>
<td></td>
<td>Hufnegg valve</td>
</tr>
<tr>
<td>6</td>
<td>Todd⁶</td>
<td>21</td>
<td>M</td>
<td>3 years</td>
<td></td>
<td></td>
<td>Rupt chordae</td>
<td></td>
<td>Died</td>
</tr>
<tr>
<td>7</td>
<td>Bjork⁷</td>
<td>44</td>
<td>F</td>
<td>1½ years</td>
<td>18</td>
<td>24/8</td>
<td>Rupt chordae, plus 2 leaflets</td>
<td>S-E valve</td>
<td>Good</td>
</tr>
<tr>
<td>8</td>
<td>Brandenburg et al.⁸</td>
<td>42</td>
<td>M</td>
<td>24 years</td>
<td>28</td>
<td>28/12</td>
<td>Rupt chordae</td>
<td>S-E valve</td>
<td>Fair</td>
</tr>
<tr>
<td>9</td>
<td>Shabetai et al.⁹</td>
<td>45</td>
<td>M</td>
<td>10 years</td>
<td>15</td>
<td>15/5</td>
<td>Valve destroyed</td>
<td>S-E valve</td>
<td>Died 9 months p.o.</td>
</tr>
<tr>
<td>10</td>
<td>Salzer et al.¹⁰</td>
<td>34</td>
<td>M</td>
<td>15 years</td>
<td>15</td>
<td>25/8</td>
<td>Dilated annulus</td>
<td>S-E valve</td>
<td>Died 10 months p.o.</td>
</tr>
<tr>
<td>11</td>
<td>Jahnke et al.¹¹</td>
<td>30</td>
<td>M</td>
<td>5 years</td>
<td>12</td>
<td>12/2</td>
<td></td>
<td></td>
<td>Good</td>
</tr>
<tr>
<td>12</td>
<td>Jahnke et al.¹¹</td>
<td>32</td>
<td>M</td>
<td>3 years</td>
<td>18</td>
<td>20/5</td>
<td></td>
<td></td>
<td>Good</td>
</tr>
<tr>
<td>13</td>
<td>Jahnke et al.¹¹</td>
<td>36</td>
<td>M</td>
<td>3 years</td>
<td>11</td>
<td>17/1</td>
<td></td>
<td></td>
<td>Good</td>
</tr>
<tr>
<td>14</td>
<td>Morgan, Forker⁸</td>
<td>70</td>
<td>M</td>
<td>32 years</td>
<td>18</td>
<td>31/14</td>
<td></td>
<td></td>
<td>Good</td>
</tr>
<tr>
<td>15</td>
<td>Morgan, Forker⁸</td>
<td>24</td>
<td>M</td>
<td>1 year</td>
<td>18</td>
<td>25/15</td>
<td></td>
<td></td>
<td>Good</td>
</tr>
</tbody>
</table>

Abbreviations: Cath = heart catheterization; M = male; F = female; pap = papillary; p.o. = postoperative; RA = right atrium; RV = right ventricle; rupt = ruptured; S-E = Starr-Edwards.
*Assumed ruptured chordae tendinae.
each group developed cyanosis immediately after trauma, secondary to a right-to-left shunt at the atrial level.4, 8

**Physical Findings**

The physical findings are distinctive and should be looked for in a patient with a history of blunt trauma. The venous pressure will be elevated, with prominent systolic venous pulsations in the neck and, possibly, an enlarged pulsatile liver. The systolic murmur at the lower left sternal border usually increases on inspiration. A useful test is the Valsalva maneuver; the murmur of tricuspid insufficiency will return in about 1 sec on release of the Valsalva, whereas left heart murmurs will usually not return for 3 sec or longer. The findings in the ruptured chordae and ruptured papillary muscle groups may be similar. Although not mentioned in any reports, a prominent fourth heart sound (with increase in inspiration) and a prominent diastolic flow murmur along the lower left sternal border would be expected to be present in the severe acute tricuspid insufficiency due to ruptured papillary muscle.

**Electrocardiographic Findings**

Seven of the 15 electrocardiograms are available for review.2, 4, 8-10 Three of the seven showed left axis deviation (between -30 and -60 degrees),2, 5, 10 while one had a frontal QRS axis of -100 degrees.8 None showed definite right axis deviation.

The most common reported electrocardiographic finding was incomplete (and occasionally complete) right bundle-branch block, present in 12 of 13 cases. P waves suggestive of right atrial enlargement were mentioned only once;5 in fact, P waves were usually more suggestive of left atrial enlargement, as in our case 1. There was a tendency to low voltage of the QRS complexes, especially in lead V1. The electrocardiograph was normal in only one patient.3

**Radiologic Findings**

Cardiac enlargement was almost always present, but the chest roentgenograms usually suggested generalized cardiac enlargement rather than localized right atrial and/or right ventricular enlargement. A good example was our case 1. Besides our case 2, only one other chest roentgenogram was completely normal.2

**Hemodynamic Data**

Prominent right atrial v waves were present, and ranged from 8 to 28 mm Hg; they were similar in the two groups. Two authors emphasized that the right ventricular end-diastolic pressure is always normal or only slightly elevated.5, 9 Three patients with a moderately elevated right ventricular end-diastolic pressure are now known. Two of these three patients are long-term survivors, i.e., 24 and 32 years, but our second patient had the highest known pressure, and he was in the first year posttrauma.

Besides marked reflux from the right ventricle to the right atrium on cineangiogram, the diagnosis of tricuspid insufficiency can be documented by normal indicator-dilution curves from the pulmonary artery, with dye curves from the right atrium and right ventricle showing a prolonged disappearance slope. Tricuspid insufficiency by cineangiogram has sometimes been attributed to the catheter across the tricuspid valve; the catheter cannot be responsible when indicator-dilution curves show the abnormal curve from the right atrium and the normal curve from the pulmonary artery.

 Intracardiac phonocardiography is also useful in the localization of the murmur in the tricuspid valve. Because the physical findings, electrocardiogram, and chest roentgenograms can mimic Ebstein's disease, this needs to be excluded by demonstrating the normal position of the tricuspid annulus with angiography and/or simultaneous pressure and electrocardiographic change from the right ventricle to the right atrium.

**Prognosis and Surgery**

Traumatic tricuspid insufficiency due to ruptured chordae tendineae is a relatively benign condition with a possibility of long-term survival without surgery. The follow-up in our patient (case 1), who is alive and asymptomatic 32 years after the onset of

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traumatic tricuspid insufficiency, indicates that all patients may not require surgery, which is contrary to the experience of all previous authors. One patient had tricuspid valve replacement 24 years after trauma, and another, 10 years after the accident. The earliest surgery performed on a patient with ruptured chordae was 1½ years posttrauma. Four patients in the ruptured chordae group had tricuspid valve replacement, all with Starr-Edwards mitral prosthetic valve. Two died in the first postoperative year.

Of the five patients in the ruptured papillary muscle group, one died immediately after trauma, one died 2 days after cardiac catheterization, and the other three patients had tricuspid valve surgery 2 to 4 months posttrauma. Two patients had a repair of the ruptured papillary muscle without valve replacement; one died 12 hr postoperatively secondary to hemorrhage and the other improved, but still had tricuspid insufficiency. The one patient with valve replacement was alive 9 months after surgery.

References
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