Traumatic Tricuspid Insufficiency*
An Underdiagnosed Disease

Gerard Champsaur, M.D.; Xavier Andre-Fouet, M.D.; and
Patrice Rueff, M.D.

Twelve cases of traumatic tricuspid insufficiency (only three of which already have been published), collected in the same city, are reported. This figure strongly contrasts with the limited number of cases published to date in the literature.

Traumatic tricuspid insufficiency (TI) usually is thought to be a rare condition. However, it appears that the frequency of this entity has been underestimated since we have been able to collect 12 cases in a medium-sized city. The aim of this study is to describe these cases briefly and to discuss the available diagnostic approaches and the treatment of this interesting valvular lesion.

Patients

Our group of patients (Table 1) consists of ten men and two women, aged an average of 39 years. All but one were injured in a motor vehicle accident and sustained multiple lesions, in particular rib fractures (ten of 12), cranial trauma with loss of consciousness (nine of 12), and less frequently, abdominal trauma and limb fractures.1,2

The diagnosis of traumatic TI was established within five days after the trauma in four cases, because of the occurrence of right heart failure, which led promptly to operation (patients 1 and 2) or because of development of an obvious tricuspid regurgitant murmur (patients 7 and 9). Three other cases (patients 10, 11 and 12) were recognized between 15 and 40 days after trauma, also because a regurgitant murmur became evident. In the other five cases (patients 3 to 6 and 8), the diagnosis was delayed (six months to 19 years after the trauma) but was then made because of the development of symptoms (typically dyspnea) or other cardiac abnormalities, such as cardiomegaly or right bundle branch block.

A murmur of tricuspid regurgitation was heard in all patients, (grade 1 or 2 in 11 cases), but it was heard only during deep inspiration in two cases. Among the other physical signs, hepatomegaly (seven of 12 cases), occasionally with systolic pulsations (four of 12), jugular venous distension (five of 12) and marked venous V waves (eight of 12) were the most frequently observed. One patient developed transient peripheral edema and acities during her stay in the Intensive Care Unit. We never observed cyanosis.

On a chest x-ray film, the heart was observed to be enlarged in nine out of 12 cases. On the electrocardiogram, all patients demonstrated sinus rhythm. Right bundle branch block was noted in eight cases; it was complete in six and incomplete in two. In one patient, the ECG suggested right atrial enlargement, while low voltage was noted in another. In two patients, the ECG was entirely normal.

Tricuspid Insufficiency was confirmed by phonocardiography and jugular venous pulse recordings in eight cases, by echocardiography in ten cases, by right-heart catheterization and right ventriculography in seven cases. Doppler echocardiography was not available to us at the time.

Table 1—Case Reports

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Diagnostic Year</th>
<th>Age at Diagnosis, yr</th>
<th>Interval Between Trauma and Diagnosis</th>
<th>Interval Between Trauma and Surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>1*</td>
<td>M</td>
<td>1972</td>
<td>47</td>
<td>5 days</td>
<td>5 days</td>
</tr>
<tr>
<td>2*</td>
<td>F</td>
<td>1977</td>
<td>22</td>
<td>2 days</td>
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<td>3</td>
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<td>12 yr</td>
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<tr>
<td>5†</td>
<td>M</td>
<td>1973</td>
<td>64</td>
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<tr>
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<td>7</td>
<td>M</td>
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<td>53</td>
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<td>8</td>
<td>M</td>
<td>1979</td>
<td>20</td>
<td>6 mo</td>
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<td>9</td>
<td>M</td>
<td>1980</td>
<td>43</td>
<td>1 day</td>
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<tr>
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<td>M</td>
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<tr>
<td>12</td>
<td>F</td>
<td>1984</td>
<td>47</td>
<td>15 days</td>
<td>...</td>
</tr>
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</table>

*From Latarjet et al.
†From Saint-Pierre et al.

*From the Departments of Cardiology and Cardiovascular Surgery, Hôpital de la Croix-Rousse and Hôpital Cardiovasculaire et Pneumologique Louis Pradel, Lyon, France. Manuscript received September 11; revision accepted March 2.
In the four patients who underwent tricuspid valve replacement, the diagnosis of traumatic TI was confirmed. A pericardial laceration was observed in three cases, while distension of the right-heart cavities was evident in all cases. The tricuspid lesions included rupture of the anterior papillary muscle (one case), rupture of anterior or septal chordae tendineae (one case each) and dislocation of the anterior or septal leaflets (one case each). In patient 1, an associated posterior rupture of the ascending aorta also was found at surgery and sutured. This patient received a Starr-Edwards 4M prosthesis, while patients 2, 3 and 4 received bioprostheses.

Patient 1 died during surgery, patient 3 committed suicide, patient 5 died without witness. All other patients were alive and in NYHA class 1 or 2 in 1985.

**DISCUSSION**

While cardiac contusion is frequently observed at the autopsy of patients who died after motor vehicle accidents, traumatic TI is considered to be rare. In their review published in 1980, Sbar and Harrison collected 45 cases of traumatic TI from the literature, including 41 cases occurring after blunt chest trauma. We have found 40 additional cases. Thus, the total number of reported cases appears to be fewer than 100. Since we have observed 12 cases in a period of 12 years in a city of about 1 million inhabitants, we think that the frequency of traumatic TI is probably underestimated, and that many cases must go undetected because of the difficulty of diagnosis either in the acute or in the chronic phase.

In the acute phase, thoracic lesions are the rule, and are often associated with cranial trauma, limb fractures or abdominal lesions; emergency surgery often is required. In this context, it is not surprising that TI may go undetected.

In the chronic phase, symptoms and signs often are subtle or atypical. Some patients remain asymptomatic because the regurgitation is only mild or moderate. In others, dyspnea and fatigue appear, but are nonspecific. The murmur may be absent, or present only during inspiration; it may be best heard at cardiac apex because of the right heart dilatation, then simulating mitral insufficiency. Hepatic enlargement, venous distension, systolic pulsations of the liver and the jugular veins also are inconstant. Cyanosis—secondary to right-to-left shunting through a stretched foramen ovale—is misleading.

However, the diagnosis of TI seems to be easier at present due to improvements in posttraumatic intensive care and advances in diagnostic techniques. So all four of our patients whose TI was diagnosed late (four to 19 years) suffered their accidents before 1970. By contrast, in all the patients injured after 1977, the diagnosis was made within six months after the acci-

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>RVEDD (mm)*</th>
<th>LVEDD (mm)†</th>
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<th>LVEDD</th>
<th>Interventricular Septal Motion</th>
<th>Tricuspid Valve</th>
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<td>35</td>
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<tr>
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<tr>
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<td>54</td>
<td>0,46</td>
<td>Hypokinetic</td>
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<td></td>
</tr>
<tr>
<td>4 Before operation</td>
<td>52</td>
<td>42</td>
<td>1,24</td>
<td>...</td>
<td>Anterior leaflet systolic luxation in right atrium</td>
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<tr>
<td>After operation</td>
<td>30</td>
<td>48</td>
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<tr>
<td>6</td>
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<td>40</td>
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<td>7</td>
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<td>35</td>
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<tr>
<td>8</td>
<td>41</td>
<td>50</td>
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<td>26</td>
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<td>0,81</td>
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<td>...</td>
<td></td>
</tr>
</tbody>
</table>

*RVEDD: right ventricular end diastolic diameter.
†LVEDD: left ventricular end diastolic diameter.

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Traumatic Tricuspid Insufficiency (Gayet et al)
dent, often by means of echocardiography. This technique discloses right-heart overload and demonstrates tricuspid regurgitation by systolic appearance of contrast in the inferior vena cava. Doppler echocardiography is still more sensitive; the velocities in the jet can be measured by the continuous wave mode, whereas the regurgitation can be graded semiquantitatively from the distance of intrusion of the jet into the right atrium by the pulsed-wave mode. Color-coded Doppler probably will further improve this sensitivity.

The traumatic origin of TI may be overlooked, especially in case of a remote accident. Echocardiography then can be of great help by demonstrating the precise mechanism of TI (Table 2). Two-dimensional echo can show prolapse or subluxation of a leaflet into the right atrium during systole (Fig 1). On M-mode recordings, one sometimes can observe a slow and irregular diastolic fluttering of tricuspid leaflets (Fig 2).

Most of the time, traumatic TI is mild or moderate and is well tolerated over years. However, more severe regurgitation sooner or later leads to symptoms, and may even warrant surgery. Our patients 3 and 4, who were operated on late after their accidents, had a resting cardiac index of less than 2l/min/sq m, which put severe limitations on exercise capacity. After operation, the cardiac index returned to normal values. Our patients 1 and 2 had operations in the first days of their accident. We could not find such cases reported in the literature, and we cannot be certain if supportive medical management could have made surgery avoidable. However, their tricuspid lesions were quite severe, and we suspect that they probably would have required tricuspid replacement rather soon.

Surgical repair of traumatic TI rarely has been attempted. Most of the time, the valve is replaced. Usually, preference is given to bioprostheses because of their lower thrombotic risks. Their most worrisome complication—primary dysfunction—might become less prevalent now that leaflet degeneration in children...
and young adults is well-established. Moreover, this risk may be lower in the tricuspid (rather than mitral or aortic) position, as suggested by Cohen et al. Finally, the mortality of reoperation is low, probably because bioprosthetic dysfunction develops slowly.

In conclusion, our experience favors the hypothesis that frequency of traumatic TI is much higher than reflected by the number of reported cases. Systematic search of its symptoms and signs, and the availability of noninvasive tools, especially echocardiography and Doppler ultrasound, should lead to its more frequent recognition.

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REFERENCES