Tricuspid Insufficiency

A Study of Hemodynamics and Pathogenesis

By Charles E. Hansing, M.D., and George G. Rowe, M.D.

SUMMARY

The clinical diagnosis of the presence and severity of tricuspid insufficiency is difficult and is complicated by the transient nature of this abnormality as well as its usual occurrence in subjects with other valve disease. This paper presents correlative hemodynamic data from 100 consecutive catheterized patients with valvular heart disease in 90 of whom the presence of tricuspid insufficiency was sought by the indicator-dilution technic. Tricuspid regurgitation was present in 28 of the 90 patients. Subjects were grouped into 20 controls without tricuspid insufficiency and those with mild, moderate, and severe tricuspid insufficiency. The hemodynamics of these groups were compared by the analysis of variance to determine which parameters relate most closely to tricuspid insufficiency. The results are interpreted to confirm that tricuspid insufficiency results from those factors which overdistend the right side of the heart, and by permitting blood to escape retrograde from the right ventricle serves as a safety valve preventing progressive overload of the pulmonary circulation.

Additional Indexing Words:
Tricuspid insufficiency  Cardiac catheterization  Indicator-dilution curves

Hemodynamics  Right heart failure

It has long been felt that the weak anatomic support structure of the tricuspid valve leads to frequent occurrence of tricuspid insufficiency in congestive heart failure.1-4 In addition to trauma, congenital, and rare causes of tricuspid insufficiency, rheumatic valvular disease may involve the tricuspid leaflets causing stenosis and insufficiency, usually in the presence of more severe aortic and mitral valve abnormalities.5 With the recent advances in cardiovascular surgery the presence or absence of tricuspid valve regurgitation has become of more than academic interest both in planning the surgical approach to the heart and determining which procedure to undertake. Furthermore, in patients with mitral stenosis and tricuspid insufficiency, the presence of a loud systolic murmur may lead to the erroneous diagnosis of mitral insufficiency6 and to misunderstanding of the nature of the clinical problem.

The following report is based on 100 consecutive cardiac catheterizations done for valvular disease in which 90 patients were studied for tricuspid insufficiency by the indicator-dilution technic.

Materials and Methods

For this study, records were chosen from 100 consecutive cardiac catheterizations on patients with suspected aortic or mitral valve disease. In each case, right heart, as well as retrograde and transseptal left heart, catheterization was done. Patients with congenital heart disease in which no tricuspid valve disease was suspected were not included.

To detect tricuspid insufficiency, a catheter was placed with its tip in the right ventricle just downstream from the tricuspid valve. A transseptal catheter was withdrawn from the right ventricle until it lay in the right atrium just upstream from the tricuspid valve (fig. 1). The pressures from both catheters were observed for...
Cardiac catheter placement for dye curve detection of tricuspid insufficiency. Dye is injected into the right ventricle. Blood is withdrawn from the right atrium and femoral artery through equally sensitive cuvette densitometers by constant withdrawal syringes.

Of the 100 consecutive cases, indicator-dilution curves were obtained across the tricuspid valve in 90 patients. In the other 10 patients, dye curves were not done for a variety of reasons which would not affect the validity of this study. The hemodynamic data for those patients with tricuspid insufficiency are presented in table 1, along with the hemodynamic data for the first 20 consecutive patients in this series who did not have tricuspid insufficiency. These “controls” are utilized as a group of representative patients from this laboratory for comparison with those who had tricuspid insufficiency. They were taken, rather than all of those without tricuspid insufficiency, to keep the groups roughly comparable in size and to avoid the mathematical manipulation of such large numbers. Those with tricuspid insufficiency...
TRICUSPID INSUFFICIENCY

Table 1

Hemodynamic Changes with Tricuspid Insufficiency

<table>
<thead>
<tr>
<th>Subjects*</th>
<th>RAV</th>
<th>RAM</th>
<th>RV, S/ED</th>
<th>PAM</th>
<th>LAM</th>
<th>CI</th>
<th>TPuR</th>
<th>PAR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>7</td>
<td>6</td>
<td>40/7</td>
<td>25</td>
<td>18</td>
<td>3.0</td>
<td>435</td>
<td>114</td>
</tr>
<tr>
<td>Mild</td>
<td>11</td>
<td>8</td>
<td>53/10</td>
<td>35</td>
<td>28</td>
<td>2.5</td>
<td>719</td>
<td>161</td>
</tr>
<tr>
<td>Moderate</td>
<td>15</td>
<td>11</td>
<td>73/13</td>
<td>40</td>
<td>26</td>
<td>2.5</td>
<td>744</td>
<td>300</td>
</tr>
<tr>
<td>Severe</td>
<td>19</td>
<td>13</td>
<td>73/16</td>
<td>50</td>
<td>27</td>
<td>1.9</td>
<td>2129</td>
<td>780</td>
</tr>
</tbody>
</table>

*The control subjects are those without tricuspid insufficiency whereas mild, moderate, and severe refer to the quantity of tricuspid insufficiency as judged by dye curves.

Abbreviations: RAV = height of the right atrial V wave; RAM = right atrial mean pressure; RV, S/ED = right ventricle pressure, systolic/end-diastolic pressure; PAM = pulmonary artery mean pressure; LAM = left atrial mean pressure; CI = cardiac index; TPuR = total pulmonary resistance; and PAR = pulmonary arteriolar resistance. All pressures are expressed in mm Hg and resistances are expressed in c g s units.

were divided into groups of mild, moderate, and severe by inspection of the indicator-dilution curves as illustrated in figure 1.

Analysis of variance for a complete design was used to compare statistically the data for patients with and without tricuspid insufficiency and for comparison within the group of patients with tricuspid insufficiency. The data were analyzed for the parameters listed in table 2. Pulmonary arteriolar resistance was calculated using mean pulmonary artery and left atrial pressures which were not recorded simultaneously. This gave a value of zero or less when the left atrial mean pressure was greater than the pulmonary artery pressure. Such data were recognized as impossible and due to error attributable to the nonsteady state from which the data were collected. This was corrected for in the statistical analysis by using the log (x + 1) format for analyzing the total pulmonary resistance and pulmonary arteriolar resistance.

Results

Of the 90 patients who had a successful indicator-dilution study of the tricuspid valve, 28 were judged by the morphology of the dye curves to have tricuspid insufficiency and 20 who had normal dye curves in this respect were taken as controls. By a comparison of the magnitude of the deflection of the dye curve obtained from the right atrium and that obtained from the systemic artery, 11 were classified as having mild tricuspid insufficiency, while 10 were moderate, and seven were severe (table 1). As a general rule, the more severe the heart disease as judged clinically and hemodynamically the more severe the tricuspid regurgitation.

More detailed analysis of those patients with tricuspid insufficiency revealed that only three had normal pulmonary artery pressure, and of these one had severe pulmonic valvular insufficiency, one had organic tricuspid valvular disease with stenosis, and one had severe aortic insufficiency. All 28 patients with tricuspid insufficiency had elevated right ventricular end-diastolic pressure. Ten had atrial fibrillation, and of these five had evidence of organic tricuspid valvular disease with a right atrioventricular diastolic gradient. Atrial fibrillation did not invariably produce tricuspid insufficiency since this arrhythmia was present in four of the 20 control patients. There were no instances of isolated tricuspid insufficiency, but one patient had heart failure due to coronary artery disease rather than valvular or congenital heart disease. Of the 28 patients with tricuspid insufficiency, five had tricuspid stenosis, 14 had mitral stenosis, 24 had mitral insufficiency, 10 had aortic stenosis, and 19 had aortic insufficiency. Two patients had congenital heart disease.

In table 2 are shown the results of comparing, by analysis of variance, various hemodynamic parameters in those without tricuspid insufficiency, and those with mild, moderate, and severe tricuspid insufficiency. It is clear from tables 1 and 2 that those factors which relate to the amount of distention of the right side of the heart are related in degree to the amount of tricuspid insufficiency, while...
Table 2

Analysis of Variance between Amount of Tricuspid Regurgitation and Other Parameters*

<table>
<thead>
<tr>
<th>Parameter</th>
<th>F value</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left ventricular end-diastolic pressure</td>
<td>1.00</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Left atrial mean pressure</td>
<td>3.10</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Cardiac index</td>
<td>4.66</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Log pulmonary arteriolar resistance +1</td>
<td>5.34</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Right ventricular systolic pressure</td>
<td>6.15</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Right atrial mean pressure</td>
<td>6.24</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Right atrial V wave</td>
<td>6.57</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Pulmonary artery mean pressure</td>
<td>7.19</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Log total pulmonary resistance +1</td>
<td>7.34</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Right ventricular end-diastolic pressure</td>
<td>7.57</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

*The manner of accomplishing these statistical comparisons is described in the text. Pressures were measured in mm Hg and resistances in c g s units. The parameters have been arranged in order from the least to the greatest F value, and the significance of the relation is expressed in the right-hand column.

left atrial mean and left ventricular end-diastolic pressures are not. It should be noted that the relationship of tricuspid insufficiency and cardiac index is inverse, i.e., the greater the tricuspid leak, the lower the cardiac index (table 1).

Discussion

Tricuspid insufficiency has been recognized clinically since 1836 when Benson9 described the jugular venous wave abnormalities in a case proven at autopsy. In the following year King10 stated that “on occasion of most copious influx (the right ventricle) becomes dilated: upon which the curtains of the tricuspid valve are drawn aside, an aperture of reflux is produced, and the force of the ventricle is diverted from the pulmonary circulation.” The clinical observation4, 5, 11 that patients with pulmonary hypertension had less orthopnea and paroxysmal nocturnal dyspnea once they developed tricuspid insufficiency would seem to support King’s concept. The present data also tend to support this concept showing a higher pulmonary artery pressure and a decreased cardiac index in those groups where tricuspid insufficiency was more severe while left atrial mean pressure did not rise significantly. This is consistent with the hypothesis that rising pulmonary arteriolar resistance by producing pulmonary hypertension, right atrioventricular distention, and tricuspid insufficiency, diminishes forward flow and progressive overloading of the left atrium and pulmonary capillary bed.

Duroziez is credited with the description of the physical findings of tricuspid insufficiency in congestive heart failure. Included were a xiphoid systolic murmur, enlarged right atrium, distended neck veins with systolic pulsation, hepatic enlargement and pulsation, and peripheral cyanosis.12 In spite of the general agreement about these signs the clinical diagnosis is frequently difficult. The characteristic murmur with its inspiratory accentuation may be missed easily in the presence of other systolic murmurs8 or in mild tricuspid insufficiency. Jugular venous V waves are not always present, may be obscured by distended neck veins, or confused with the CV waves in atrial fibrillation. The hepatic pulsation is readily detected only with severe tricuspid insufficiency and may be confused with a transmitted pulse from the aorta or right ventricle.11 Furthermore, the presence and severity of tricuspid insufficiency are remarkably labile, being greatly influenced by exercise, deep breathing, emotions, and the degree of cardiac compensation.1, 11 For these reasons objective confirmation of the diagnosis has long been sought.

In 1908 MacKenzie,13 using jugular phlebograms, presented what he considered to be stages of severity of tricuspid regurgitation. In
TRICUSPID INSUFFICIENCY

more recent years, investigators studying the right atrial pressure curves have inferred the relatively common occurrence of tricuspid regurgitation in severe congestive heart failure and with rheumatic disease of the valve.\textsuperscript{1, 4, 5, 11, 14} It is generally agreed that the initial change in right atrial curves is a decrease in the depth of the X descent which eventually almost disappears as the V wave becomes larger, leading to “ventricularization” of the curve with severe tricuspid insufficiency. It has frequently been felt that pressure-curve analysis misses some mild cases of tricuspid insufficiency,\textsuperscript{4, 5} and one study of right atrial pressure recordings taken at the time of surgery on six patients with moderate-to-severe insufficiency, as judged by the surgeon, produced normal pressure curves.\textsuperscript{15} The large CV waves seen in the right atrium in atrial fibrillation have led to some confusion as to whether all patients with atrial fibrillation have tricuspid regurgitation,\textsuperscript{9} or whether it occurs only in the presence of atrial fibrillation plus some myocardial insufficiency.\textsuperscript{1, 11} The data presented here would support the latter opinion and support the reservations concerning diagnosis of tricuspid insufficiency from pressure curves alone although there was a significant relation between the V wave and the severity of tricuspid insufficiency.

Angiocardiology has been unreliable in demonstrating tricuspid insufficiency. The high-pressure delivery of contrast material into the right ventricle may cause the catheter to regurgitate into the right atrium or stimulate premature ventricular contractions which produce insufficiency. Only if adequate opacification is achieved and no regurgitation is seen can one state confidently that tricuspid insufficiency is absent.\textsuperscript{16}

In the presence of tricuspid regurgitation when indicator is injected into the right ventricle and sampled from a systemic artery, there is a spread in the curve which differs from that seen with reduction in cardiac output.\textsuperscript{1} However, such curves are also distorted by insufficiency of the other valves and intracardiac shunts, and subsequent model testing did not confirm the fundamental premise of the method since there was not a quantitative relation between the variance of the curve and the amount of insufficiency when the characteristics of the model were changed.\textsuperscript{17} With development of indocyanine-green dye, a more sensitive indicator-dilution method was established in which dye was injected into the right ventricle and sampled from the right atrium.\textsuperscript{18, 19} Rather gross quantitation of regurgitation is estimated by comparison of the right atrial deflection with that obtained from a systemic artery.\textsuperscript{19} These methods utilized either two venous catheters with one in the right ventricle and one in the right atrium\textsuperscript{19} or a no. 9 double-lumen catheter.\textsuperscript{18} A large double-lumen catheter is required in order for blood to be aspirated sufficiently rapidly from the right atrium to get a good dye curve with a reasonable appearance time. In this laboratory the catheter passed from the arm to the right ventricle is a very pliable no. 6 chosen to avoid displacement of the tricuspid valve leaflets, and to decrease the number of premature contractions when the right ventricle impales itself on the catheter tip with each systole. With such a pliable catheter to avoid catheter motion and premature contractions, dye injection must be done slowly and carefully. The transseptal catheter’s natural curve allows its tip to be positioned near the tricuspid valve orifice. Independent positioning of the injecting and sampling catheter orifices facilitates their optimum placement in relation to the tricuspid valve and may constitute some advantage over a double-lumen catheter. Since the exact anatomic position of the tricuspid valve cannot be discerned fluoroscopically, prolonged observation of the pressure curves from the right atrium and the right ventricle are used to determine that neither catheter orifice crosses the tricuspid valve. If there is doubt as to whether this has occurred, the dye curve is repeated until consistent results are obtained. With the precautions cited above no indocyanine-green dye appears early in the right atrium in virtually all of those patients with normal right heart pressures. In an occasional patient a trace of dye appears, and
they are classified with the group without tricuspid insufficiency.

Quantitation of regurgitant flow would depend on instantaneous and complete mixing of the dye with the right ventricular blood or predictable mixing with a particular fraction of this blood, similar mixing of the regurgitated dye with the blood in the right atrium, and knowledge of the volume of blood contained in the right atrium. Since none of these mixing conditions is possible and the right atrial volume effectively varies continuously with the systolic and diastolic flow of blood, accurate quantitation of regurgitation is impossible. It seems reasonable to try to determine whether mild, moderate, or severe tricuspid insufficiency is present by comparison of the right atrial and femoral artery dye curves (fig. 2). This would appear to be the most sensitive method at present for determining the presence and severity of tricuspid insufficiency in a cardiac catheterization laboratory. A much more accurate phasic quantitation of tricuspid regurgitation can be measured in dogs by chronic surgical implantation of electromagnetic flow probes on the atrial side of the tricuspid valve (Folts JD, Young WP, Ravan M, Rowe GG: Unpublished data.)

The presence of tricuspid insufficiency in 28 of the present 90 patients who were studied at rest after optimal hemodynamic conditions had been established supports the clinical impression that the tricuspid valve is very frequently incompetent in congestive failure.10, 11.15 Differentiation of functional tricuspid incompetence which is secondary to dilatation of the right ventricle and tricuspid annulus with congestive failure, from incompetence which is due to organic disease of the valve, cannot be made at the time of catheterization. The presence of a tricuspid diastolic gradient (tricuspid stenosis), however, strongly suggests organic tricuspid valve disease. The detection and rough quantitation of tricuspid insufficiency and/or stenosis should be of interest to the cardiac surgeon and may help direct his approach to the heart. However, the final decision concerning surgical treatment of tricuspid insufficiency, whether it be valve replacement, annuloplasty, or no operative intervention, must still be made at operation following relief of the other cardiac abnormalities.20

Acknowledgment

The help and advice of Professor J. H. Torrie, Professor of Agronomy, in the statistical methods used in this study are gratefully acknowledged.

References

10. King TW: An essay on the safety valve function in the right ventricle of the human heart and on gradations of this function in the circulation of warm blooded animals. Guy Hosp Rep 2: 104, 1837
12. Dutroize P: Du Retreccissement de La Tricuspide Gaz Hop Paris 41: 310, 1868

Circulation, Volume XLV, April 1972
TRICUSPID INSUFFICIENCY

Detection of pulmonic and tricuspid valvular regurgitation by means of indicator solutions. Circulation 20: 561, 1959


18. Collins NP, Braunwald E, Morrow AG:...
Tricuspid Insufficiency: A Study of Hemodynamics and Pathogenesis
CHARLES E. HANSING and GEORGE G. ROWE

Circulation. 1972;45:793-799
doi: 10.1161/01.CIR.45.4.793

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1972 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/45/4/793

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/