Hemodynamic Studies in Tricuspid Stenosis of Rheumatic Origin

By M. Irené Ferrer, M.D., Régane M. Harvey, M.D., Marvin Kuschner, M.D., Dickinson W. Richards, Jr., M.D., and André Cournand, M.D.

Clinical data and hemodynamic measurements made by cardiac catheterization are presented in two patients with tricuspid stenosis of rheumatic origin. In one, pure tricuspid stenosis was proved by autopsy, and in the second the development of tricuspid stenosis and insufficiency was demonstrated by means of cardiorespiratory studies made 27 months apart. A detailed analysis of the pressure pattern characteristic of this type of valvular stenosis is given. The measurement of the stenosed tricuspid valve area by three methods is described and discussed.

The clinical features of tricuspid stenosis of rheumatic origin have been well described in the past and certain of the physical signs have been re-emphasized lately. Since there has been only one case to date in which some direct hemodynamic information was given, it was thought interesting to report a detailed study of two patients with this lesion. In one, the stenosis existed without insufficiency of the valve and in the presence of normal sinus rhythm; the diagnosis was eventually confirmed by necropsy. In the other subject, stenosis and insufficiency of the tricuspid valve were present along with auricular fibrillation. In both subjects the technic of cardiac catheterization was utilized to obtain the hemodynamic data. From these latter will be drawn certain physiologic conclusions pertinent to this unusual circulatory abnormality. The two cases will be presented separately.

Case I

The first patient, a 26 year old English-born housewife, had scarlet fever in childhood and occasional fleeting joint pains thereafter. At the age of 19 she was told she had a heart murmur although she was asymptomatic at that time. Dyspnea on exertion began at the age of 22 years and occasional ankle edema was noted during her twenty-fourth year. Six months prior to admission she developed marked congestive heart failure during an attack of influenza and this cleared after digitalization. She stopped taking digoxin two months before admission and one month later the signs of cardiac failure were again present and for these she sought hospitalization.

Physical examination revealed, in addition to rales in the chest, hepatomegaly and ankle edema, a regular sinus rhythm and an enlarged heart with only the murmur of mitral stenosis. There were no murmurs heard in the tricuspid region. The electrocardiogram showed right axis deviation with notching of the P waves and some T-wave abnormalities. Later in her course, after digitalization and recovery from congestive failure, the P-R interval was found to vary between 0.16 second and 0.22 second in the same tracing. The blood count and sedimentation rate were within normal limits. She remained afebrile throughout her course. When the patient had been up and about at home and was free of any signs of cardiac failure, she was readmitted and studied by cardiac catheterization. It should be noted particularly that the liver was no longer palpable at this time. The patient subsequently was operated upon and a mitral commissurotomy was performed. Cardiac ar-
rest occurred during fracture of the valve and although cardiac massage succeeded in restoring sinus rhythm the patient died of cerebral anoxia the next day.

Pathologic Examination

The pertinent findings at autopsy were as follows. The heart weighed 330 Gm. The epicardium, endocardium, and myocardium contained numerous small flame-shaped hemorrhages. Fine flakes of fibrin coated the epicardium at the base of the heart. The endocardium was thin and smooth except over the posterior portion of the left auricle where it was thickened and rugose. There was mild dilatation of the ventricles and moderate dilatation of the auricles, more marked on the right. The tricuspid valve was markedly stenosed. The orifice was reduced to an ovoid opening admitting the tip of an index finger and measuring 1 cm. by 2 cm. along the main axes. The free margins of the valve were rolled and inflexible but the leaflets were flexible and fell together readily. The chordae tendineae were somewhat shortened and thickened. There was advanced stenosis of the mitral valve which was funnel shaped with a small fixed orifice. The proximal portion of the anterior leaflet was flexible however. The chordae tendineae were thick and so extremely short that the valve edges were almost in direct contact with the papillary muscles. The anterolateral commissure of the mitral valve had been fractured, and there was a smaller splitting of the posteromedial commissure. The aortic valve had minimal changes but was apparently neither insufficient nor stenosed, and no insufficiency pockets were seen in the subjacent ventricular endocardium. The left ventricle measured 1 cm. in thickness and the right measured 0.3 to 0.5 cm.

The left lung was not fully expanded and the left pleural cavity contained 300 cc. of fluid and clotted blood. The lungs were rust colored and somewhat indurated. Numerous atheromatous plaques and yellow lipid streaks were seen in the large branches of the pulmonary artery.

Microscopic examination revealed thickening of the mitral, tricuspid and aortic valves by fairly dense, hyalinized fibrous tissue. Small thick-walled vessels extended into the leaflets of these valves. No verrucous endocarditis was seen on the valve surfaces but thrombus material consisting chiefly of fibrin was found on the mitral valve at the site of fracture. Several Aschoff bodies were present within the myocardium of the interventricular septum, and in the posterior wall of the left auricle Aschoff cells in characteristic palisade arrangement were found in the subendocardial fibro-elastic tissue. Focal hemorrhage and infiltration of polymorphonuclear cells were seen between the myofibrils of the ventricular myocardium and were attributed to cardiac massage.

Within the lungs, the alveolar capillaries were congested and the alveolar septa moderately thickened. Groups of alveoli contained pigment laden macrophages. There was mild medial hypertrophy of the walls of the small arterioles but arteriolar sclerosis was not present.

The final anatomic diagnosis was: Rheumatic endocarditis, inactive, of mitral, aortic and tricuspid valves with mitral stenosis and insufficiency and tricuspid stenosis; rheumatic myocarditis, active, of left ventricle; rheumatic auriculitis, active, of left auricle; chronic passive congestion of lungs, spleen and pancreas; severe chronic passive congestion of liver with "cardiac cirrhosis"; status postmitral commissurotomy.

Hemodynamic Data

The physiologic measurements were secured according to the techniques described in a previous paper. As can be seen in table 1, the resting cardiac output and stroke volume were greatly reduced and there was a moderate degree of pulmonary artery hypertension. The right auricular mean pressure greatly exceeded the right ventricular end diastolic pressure (see table 1), and, as is shown in figure 1, there was a higher pressure throughout diastole in the right auricle than in the right ventricle. The discrepancy in the diastolic pressure levels is made evident by examination of the normal pressure tracing (fig. 1), where these levels are almost the same. In both tracings (fig. 1) the pressure curves were secured as the cath-
TABLE 1.—Physiologic Data in a Patient (Case 662) with Mitral Stenosis, Tricuspid Stenosis and Normal Sinus Rhythm

<table>
<thead>
<tr>
<th>Cardiac Output (L/Min.)</th>
<th>Cardiac Index (L/Min./M²BSA)</th>
<th>Stroke Volume (cc)</th>
<th>Heart Rate</th>
<th>Art. Oxygen Sat. (%)</th>
<th>Oxygen Consumption (cc./Min./M²BSA)</th>
<th>Pressures in mm. Hg</th>
<th>TBV (cc/M²BSA)</th>
<th>PV (cc/M²BSA)</th>
<th>Hct %</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(a) At rest</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.48</td>
<td>1.64</td>
<td>28</td>
<td>80</td>
<td>96</td>
<td>113</td>
<td>105/69,87</td>
<td>44/26,33</td>
<td>2</td>
<td>—</td>
</tr>
<tr>
<td>2.68</td>
<td>1.77</td>
<td>30</td>
<td>89</td>
<td>94</td>
<td>117</td>
<td>103/60,77</td>
<td>41/22,28</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td>(b) After 9 minutes of leg exercise</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.83</td>
<td>1.87</td>
<td>28</td>
<td>100</td>
<td>93</td>
<td>223</td>
<td>121/73,97</td>
<td>62/34,42</td>
<td>1</td>
<td>—</td>
</tr>
</tbody>
</table>

M.²BSA = per square meter of body surface area. TBV = Total blood volume. s = systolic. d = diastolic. m = mean. PV = Plasma Volume. Hct = Hematocrit.

* The Z point is that point on the right auricular curve which corresponds in time to the onset of ventricular systole.

Fig. 1. Pressure tracing obtained in tricuspid stenosis (patient 662, upper frame) compared to a normal tracing (lower frame). In the upper row in each case are shown the blood pressure curves from the right ventricle (R.V.) and the right atrium (R.A.). These were secured as the catheter was withdrawn through the tricuspid valve. Below the right heart tracings are those of the brachial artery (B.A.) and the electrocardiographic lead II. The letter D indicates the end diastolic point on the right ventricular curve and a similar point in time on the right auricular curve is also labeled D. (This letter corresponds to the right auricular Z point.) The calibration scale is in millimeters of mercury. (For discussion see text.)

These curves were recorded using Statham gage pressure transducers and the cathode ray oscillographic technic for registering blood pressures on photographic paper.
eter was withdrawn from the right ventricle into the right auricle.

In figure 2, the right auricular and right ventricular tracings have been redrawn from the numbered beats of figure 1 as if they had been recorded simultaneously, in order to indicate the physiologic events occurring on either side of the stenosed valve. Particular care was taken to select beats in the same phase of respiration. A similar reconstruction was made from the normal tracing (see fig. 2). From the latter it is evident that once the normal tricuspid valve opens, the pressure curves in diastole in the right auricle and ventricle are almost superimposable. When tricuspid stenosis impedes the emptying of the right auricle there is an atrioventricular pressure gradient throughout ventricular diastole, and the end diastolic point in the right ventricle is actually lower than the end diastolic point as read from the right auricular pressure curve (the Z point, see table 1). Under normal circumstances these pressures are identical at the end of diastole, and hence the tricuspid valve closes as soon as isometric contraction begins. In the patient with tricuspid stenosis, however, since the right atrial pressure is much higher than is the end diastolic pressure in the right ventricle, this latter chamber must raise its pressure to the auricular level before the valve closes, and, therefore, "isometric contraction" is prolonged.

![Figure 2](image)

**FIG. 2.** Reconstruction of the right heart curves shown in figure 1. The right atrial (R.A.) and right ventricular (R.V.) curves have been redrawn and superimposed as if they had been recorded simultaneously. (For discussion see text.)

![Figure 3](image)

**FIG. 3.** Pressure tracings of the right atrium (R.A.) and brachial artery (B.A.) and electrocardiographic lead II in a patient (case 662) with tricuspid stenosis in the upper row (using the electrical recording device), and in a patient with tricuspid insufficiency (case 495) in the lower row (using Hamilton manometers). Both subjects were in normal sinus rhythm. The arrow indicates the onset of ventricular systole in each tracing and the letter A marks the peak of atrial systole. (For discussion see text.)

The right auricular pressure curve which appears in figure 3 was recorded at a greater amplification than in figure 1 in order to permit a more detailed analysis of the events occurring throughout the cardiac cycle in this chamber.
In this record a number of interesting features can be seen: (1) the peaks of atrial systole are very high, ranging from 9 to 12 mm Hg, (2) there is a marked fall in pressure during the ventricular contraction and the descent of the base of the ventricles, and finally (3) the opening of the tricuspid valve is not attended by a marked fall in pressure. Each of these will be discussed in turn.

The sharp fall in pressure following the high peak of atrial systole is much greater than is seen normally and suggests that the dynamic events are taking place in a distended right atrium. Impaired emptying of the right auricle in the presence of tricuspid stenosis is the obvious explanation for this distention.

Figure 3 clearly demonstrates a fall in auricular pressure during ventricular contraction (indicated by an arrow). One can therefore conclude that there was no regurgitation through the tricuspid valve and hence no insufficiency of the valve. This is made more evident when one compares the right atrial curves in this patient with that of a rheumatic cardiac with normal sinus rhythm and tricuspid insufficiency without tricuspid stenosis (fig. 3). In the latter very shortly after the onset of ventricular systole (also labeled with an arrow) there is a sharp rise in right atrial pressure.

There is not a marked drop in pressure in the atrium following the onset of the opening of the tricuspid valve, that is, following the V point (see figs. 2 and 3) such as takes place in the normal heart. The absence of a marked drop in pressure in this distended right auricle shows that the rate of flow across the valve in early diastole is reduced and the volume change is small.

From these facts it is obvious that right ventricular filling was not accomplished in large measure during early diastole. Indeed, examination of figure 2 suggests that ventricular filling took place throughout the diastolic period; a large atrioventricular pressure gradient favored continuous flow across the valve. The pulsatile nature of this continuous flow is suggested in this same figure 2, where it can be seen that the magnitude of the pressure gradient varied throughout the successive phases of diastole. For example, the gradient of pressure in early, mid and late diastole in each of the three labeled beats was 9, 5, 12; 9, 16, 13; 13, 7, 13 mm Hg respectively.

The cause of the continuous flow is easily understood when one considers that a stenosed valve cannot fall open in diastole and lie along the ventricular walls as do the normal leaflets. Hence the early inrush of blood to the ventricle, so important in normal ventricular filling, is prevented; blood is impounded in the atrium and pressure in this latter chamber remains high. This point is emphasized by comparing the pressure fall after the V point with that of the patient with tricuspid insufficiency whose right atrium was also distended but in whom early diastolic atrial emptying did occur (Fig. 3).

Analysis of pressure curves and blood flow data at rest have yielded interesting physiologic information in this patient. Measurements made during supine leg exercise also afforded some pertinent facts (table 1). The response to exercise in this woman was very abnormal in that the cardiac output and hence pulmonary blood flow did not increase significantly, despite which she had a sharp rise in pulmonary hypertension and heart rate. The rise in pulmonary artery pressures can be ascribed to the shortened left ventricular diastolic filling time accompanying the rise in heart rate in the presence of mitral stenosis. It is of interest that in this patient there was no change in the right ventricular end diastolic pressure during exercise, while in 11 other patients with rheumatic heart disease and no tricuspid stenosis there was a rise in this pressure during exercise although not necessarily to abnormal levels. The absence of an early rise in the right ventricular diastolic pressure was presumably due to the fact that much of the increased venous return early in exercise could not cross the stenosed tricuspid valve and reach the right ventricle in the presence of a shortened diastolic filling time. It is obvious from these observations that this woman was greatly handicapped in the performance of any exertion, since venous return to both right and left ventricles was impeded.
HEMODYNAMIC STUDIES IN TRICUSPID STENOSIS

Calculations of Tricuspid Valve Area

Since the autopsy specimen of the heart afforded us the opportunity of examining the tricuspid valve directly, an attempt was made to measure the area of this valve orifice by three different methods, recognizing the fact that each method has its own limitations. Therefore the results obtained, although not precise in any instance, may indicate the limits within which such measurements can be made with this type of material.

In the first instance, the pathologist assumed the role usually taken by the surgeon doing a commissurotomy and used his finger tip to measure the valve orifice at autopsy. The inherent error of this method is that the orifice may be overstretched by this maneuver and hence the orifice area over estimated. Secondly a Kodachrome photograph (fig. 4) of the ventricular side of the valve was projected, the elliptic form of the opening was traced on paper and the area of the orifice calculated by planimetric integration of this tracing. This photograph of itself, however, may have been misleading because the edges of the valve may have fallen closer together than they were in life in as much as we are no longer dealing with a fluid dynamic system. This area then would be too small.

Thirdly, the area of the tricuspid valve orifice was calculated according to the formulas suggested by Gorlin and Gorlin. The data used are given in table 2. As can be seen,
valve had an elliptic orifice at the base of a conical valve and the flow across it was presumably pulsatile.

This patient, a 47 year old male, had been having increasing dyspnea for several months when he was first seen in December 1949. At this time a diagnosis of mitral stenosis and insufficiency of rheumatic origin was made. No other murmurs were heard. The first catheterization study (table 4) was performed when the patient had normal sinus rhythm and demonstrated a normal resting cardiac output and blood volume, with severe pulmonary hypertension as inferred from the height of the right ventricular systolic pressure. At this time (Jan. 15, 1950) the end diastolic pressure in the right ventricle and the right auricular mean pressure were almost identical and were normal. It is important to note that the right ventricular end diastolic and right auricular Z-point pressures were the same. In October 1950 a palpable and slightly pulsatile liver was noted, and in May 1951 the patient was found to have auricular fibrillation. Two months later, in July 1951, peripheral edema and marked hepatomegaly were present. Congestive failure continued despite therapy until February 1952 when edema and liver size decreased. Two episodes of peripheral arterial emboli—one to a radial and one to a small retinal artery—occurred in March and April 1952. During his hospitalization for this latter event, a second cardiac catheterization was performed. A murmur over the tricuspid area had been heard

In table 3 are given the average measurements calculated from the hydraulic formulas and the values obtained by the other two methods. The valve area arrived at by hydraulic formulas was not the same as the one estimated by an exploring finger but did approach the valve area calculated from the autopsy photograph. It is difficult to be certain which of the three most nearly approximates the correct measurement of the functioning valve orifice. To attempt a reconciliation of these different estimates of valve size would not then appear particularly profitable.

**Case II**

The previous case presentation, with autopsy confirmation of the diagnosis, served to establish the hemodynamic pattern of tricuspid stenosis. On the strength of such a pattern, tricuspid stenosis was diagnosed in a second subject even though there is no anatomic confirmation of the lesion.

**Table 2.—Data Used in Estimation of Tricuspid Valve Area According to Hydraulic Formulas**

<table>
<thead>
<tr>
<th></th>
<th>Cardiac Output (cc./min.)</th>
<th>Diastolic Filling Period (sec. per beat)</th>
<th>Heart Rate</th>
<th>Diastolic Filling Period (sec. per min.)</th>
<th>T.V.F. (cc. per sec.)</th>
<th>Pressures in mm. Hg</th>
<th>T.V.A (cm.²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beat #1</td>
<td>2480</td>
<td>0.40</td>
<td>35.2</td>
<td>70.4</td>
<td>7.60</td>
<td>0</td>
<td>0.55</td>
</tr>
<tr>
<td></td>
<td>2680</td>
<td>0.40</td>
<td>35.2</td>
<td>76.0</td>
<td>7.60</td>
<td>0</td>
<td>0.60</td>
</tr>
<tr>
<td>Beat #2</td>
<td>2480</td>
<td>0.39</td>
<td>33.2</td>
<td>74.8</td>
<td>8.50</td>
<td>0</td>
<td>0.58</td>
</tr>
<tr>
<td></td>
<td>2680</td>
<td>0.39</td>
<td>33.2</td>
<td>80.7</td>
<td>8.50</td>
<td>0</td>
<td>0.62</td>
</tr>
<tr>
<td>Beat #3</td>
<td>2480</td>
<td>0.40</td>
<td>35.2</td>
<td>70.4</td>
<td>4.75</td>
<td>-3.54</td>
<td>0.54</td>
</tr>
<tr>
<td></td>
<td>2680</td>
<td>0.40</td>
<td>35.2</td>
<td>76.0</td>
<td>4.75</td>
<td>-3.54</td>
<td>0.55</td>
</tr>
<tr>
<td><strong>Average</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td><strong>0.58</strong></td>
</tr>
</tbody>
</table>

T.V.F. = Tricuspid Valve Flow. T.V.A. = Tricuspid Valve Area.

**Table 3.—Various Estimations of Tricuspid Valve Area**

<table>
<thead>
<tr>
<th>From Hydraulic Formulas¹</th>
<th>By Pathologist's Finger</th>
<th>From Photograph of Autopsy Specimen</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.58 cm.²</td>
<td>1.65 cm.²</td>
<td>0.68 cm.²</td>
</tr>
<tr>
<td>(0.54 to 0.62 cm.²)</td>
<td>(0.66 to 0.69 cm.²)</td>
<td></td>
</tr>
</tbody>
</table>
HEMODYNAMIC STUDIES IN TRICUSPID STENOSIS

TABLE 4.—Physiologic Data at Rest in a Patient (Case 559) with Mitral Stenosis and Insufficiency, Tricuspid Stenosis and Insufficiency and Auricular Fibrillation, before and after the Development of the Tricuspid Lesion

<table>
<thead>
<tr>
<th>Date</th>
<th>Cardiac Output (L./Min.)</th>
<th>Cardiac Index (L./Min./M.²BSA)</th>
<th>Stroke Volume (cc.)</th>
<th>Heart Rate</th>
<th>Art. Oxygen Sat. (%)</th>
<th>Oxygen Consumption (cc./Min./M.²BSA)</th>
<th>Pressures in mm. Hg</th>
<th>TBV (cc/M.² BSA)</th>
<th>PV (cc/M.² BSA)</th>
<th>Heart %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/15/50</td>
<td>4.24</td>
<td>2.92</td>
<td>58</td>
<td>80</td>
<td>97</td>
<td>111</td>
<td>106/69,86</td>
<td>70/30</td>
<td>2/2</td>
<td>2680</td>
</tr>
<tr>
<td>N.S.R.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1365</td>
</tr>
<tr>
<td>4/28/52</td>
<td>3.75</td>
<td>2.56</td>
<td>58</td>
<td>65</td>
<td>94</td>
<td>121</td>
<td>109/63,71,37</td>
<td>73/30</td>
<td>8/7</td>
<td>2920</td>
</tr>
<tr>
<td>Aur. Fib.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1563</td>
</tr>
</tbody>
</table>

For symbols see table 1.

inconstantly during this time and was considered by most observers to be high pitched, musical and systolic in time.

Comparison of the first and second hemodynamic studies, made 27 months apart, was most revealing (table 4). The cardiac output was lower and blood volume only slightly higher in the second observations and there was no change in right ventricular systolic pressure. The end diastolic pressure in the right ventricle was exactly the same as on the first examination but the right auricular mean and Z-point pressures were much higher. Thus a pressure gradient between diastolic auricular and ventricular pressures had developed since the first study and was interpreted as due to tricuspid stenosis. A comparison of the two auricular pressure curves (fig. 5) further demonstrated that the function of the tricuspid valve had been compromised, because, in addition to the tricuspid stenosis, the second curve (April 28, 1952) showed a well marked regurgitant wave during ventricular systole, typical of tricuspid insufficiency. It would thus appear definite that a double tricuspid valvular lesion had developed in this man in the interval between the two catheterizations. The question might be raised as to the level of right ventricular end diastolic pressure in tricuspid insufficiency and whether this lesion, proved in this case by the pressure curve, could of itself alone account for the hemodynamic changes found. It is known that tricuspid insufficiency is almost always found with auricular fibrillation, but in a large number of rheumatic patients with tricuspid insufficiency alone the right auricular mean and Z-point pressures and the end diastolic right ventricular pressure were either identical or

Fig. 5. Pressure tracings in the right atrium and peripheral artery before (upper curves) and after (lower curves) the development of tricuspid stenosis and insufficiency with auricular fibrillation. The arrow indicates the onset of ventricular systole in each tracing. B.A. is the brachial artery and F.A. the femoral artery. (For discussion see text.)

Hamilton manometers were used to record the upper curves and the electrical recording device the lower curves.
matched each other within 1 or 2 mm. Hg. This was true regardless of whether these pressures were 3 mm. Hg or as high as 22 mm. Hg (authors' unpublished observations in 11 cases). Therefore the gradient found in this patient could not be laid to insufficiency of the valve and is interpreted as evidence of tricuspid stenosis.

In conclusion, it should be pointed out that in the past the diagnosis of tricuspid valvular lesions by means of clinical criteria alone has not only been unsatisfactory but in all likelihood has been possible only in the advanced stages of involvement. This latter inference is reinforced by the fact that in a recent paper eight cases of tricuspid insufficiency were discovered in whom the clinical criteria were equivocal or absent. Furthermore, in the two patients described in the present report, it would not have been possible to diagnose the tricuspid stenosis on clinical grounds in either instance. It is obvious then, particularly since surgical intervention is available for at least partial correction of stenotic valves, that considerable attention and careful analysis should be given to the right atrial pressure curves in all subjects undergoing cardiac catheterization in order to uncover these lesions.

SUMMARY
1. Clinical data and hemodynamic measurements made by cardiac catheterization are presented in two patients with tricuspid stenosis of rheumatic origin. In one, pure tricuspid stenosis was proven by autopsy and in the second the development of tricuspid stenosis and insufficiency was demonstrated by means of cardiocirculatory studies made 27 months apart.
2. A detailed analysis of the pressure pattern characteristic of this type of valvular stenosis is given.
3. The measurement of the stenosed tricuspid valve area by three methods is described and discussed.

ACKNOWLEDGMENTS
The authors gratefully acknowledge the help and cooperation of Dr. E. H. Luckey, Dr. H. B. Kirkland and the staff of the Second (Cornell) Medical Division, Bellevue Hospital, in making one of these patients (case 662) available for study.

REFERENCES
Hemodynamic Studies in Tricuspid Stenosis of Rheumatic Origin
M. IRENE FERRER, REJANE M. HARVEY, MARVIN KUSCHNER, DICKINSON W. RICHARDS, JR. and ANDRE COURNAND

Circ Res. 1953;1:49-57
doi: 10.1161/01.RES.1.1.49
Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1953 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circres.ahajournals.org/content/1/1/49

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation Research 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 Research is online at:
http://circres.ahajournals.org/subscriptions/