The Diagnosis of Tricuspid Insufficiency
Clinical Features in 60 Cases with Associated Mitral Valve Disease

By Gonzalo Sepulveda, M.D. and Daniel S. Lukas, M.D.

In a series of 146 patients with mitral stenosis studied by cardiac catheterization, 60 were found to have right atrial pressure curves diagnostic of tricuspid insufficiency. The clinical and hemodynamic features of these patients are reviewed. The oft-cited, classic manifestations of tricuspid insufficiency were found with relative infrequency and the diagnosis had been made clinically in less than one fourth of the cases. Modification of criteria for the diagnosis of tricuspid insufficiency is suggested.

INSUFFICIENCY of the tricuspid valve (T.I.) produces characteristic alterations in the right atrial and peripheral venous pressure curves.\(^1\)\(^-\)\(^3\) The essential component of these alterations is an increase in pressure during ventricular systole. Clinical diagnosis of the lesion has been dependent on recognition of this systolic pulsation in the systemic veins, chiefly those of the neck and liver.

The right atrial pressure curve that was originally described by Bloomfield and associates\(^4\) as diagnostic of tricuspid insufficiency has been found with surprising frequency among patients with advanced mitral disease.\(^5\)\(^-\)\(^6\) Failure to recognize the tricuspid lesion clinically in a very large proportion of the cases suggested that the present diagnostic criteria may be too stringent. Accordingly, with the purpose of identifying the most common manifestations of tricuspid insufficiency, a review was made of the clinical features of 60 patients with chronic rheumatic heart disease and predominant mitral valvular involvement in whom associated tricuspid insufficiency was demonstrated by cardiac catheterization. In addition the hemodynamic data obtained in these patients was analyzed and compared with those from a group of patients without tricuspid insufficiency but with a similar degree of mitral involvement.

Material and Methods

The material for this study consisted of 60 patients with chronic rheumatic heart disease and moderate to severe mitral stenosis. Almost all were undergoing evaluation for mitral valvuloplasty. Some degree of mitral insufficiency was present in 41 and at valvuloplasty was usually estimated to be mild. Ten patients had an associated aortic lesion. The average age was 40.6 years (range, 21 to 60). The 40 females had an average age of 41.9 years (range, 24 to 60); the 20 males, 38 years (range, 21 to 52).

In the analysis of the clinical features particular attention was paid to the following criteria listed by The Criteria Committee of the New York Heart Association as diagnostic of tricuspid valve disease:\(^7\): cyanosis, often combined with icterus; distended neck veins, which may show unusually marked pulsations; rare orthopnea; frequent auricular fibrillation; enlarged liver, which may show unusually marked pulsations; recurrent ascites; enlargement of the right atrium and clear lung fields at the roentgenologic examination; right axis deviation of QRS in the electrocardiogram. Particularly suggestive of tricuspid insufficiency are: systolic pulsation of the cervical veins and often of the veins of the extremities and systolic expansile pulsation of the liver. The Committee states: “The clinical diagnosis of tricuspid valve deformity, although difficult, can be made correctly in an appreciable number of instances. It should not be made in the presence of congestive failure, since then tricuspid incompetency is frequently present. It should be suspected in a rheumatic patient with mitral stenosis who has persistently enlarged liver, engorged neck veins and recurrent ascites, but who is free of orthopnea. Murmurs rarely contribute to the diag-

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nosis of this valvular deformity since it is difficult or impossible to distinguish them from the murmurs of mitral stenosis and insufficiency which are almost invariably present in these patients.

The clinical signs reported herein were observed during hospitalization of each patient for cardiac catheterization. All patients were receiving digitalis; those who had cardiac failure were under as optimum medical control as was deemed possible at time of catheterization.

Conventional roentgenograms of the chest, available in 59 cases, were examined with regard to overall cardiac size, right atrial enlargement and pulmonary vascular congestion. An estimate of the degree of enlargement of the right atrium was based on the distance the atrial border protruded to the right from the midline of the thorax in the posteroanterior roentgenogram. The distance was 4 cm. or less for slightly enlarged atria, 4 to 6 cm. for moderate enlargement, 6 to 8 cm. for severe enlargement and over 8 cm. for giant-sized atria. These measurements were selected after review of 24 available angiocardiograms which provided an accurate delineation of the right atrium.

Cardiac catheterization, recording of pressures and determination of cardiac output were performed by methods previously described. Exercise was performed with the patient in the recumbent position and consisted of flexion and extension of the legs at the rate of one cycle per two seconds for five minutes. All right atrial pressure tracings were carefully examined; only those with an unequivocal pattern of tricuspid insufficiency were included. An associated tricuspid stenosis was ruled out by comparing the Z point (point on the right atrial curve that corresponds in time to the onset of ventricular systole) with the right ventricular end-diastolic pressure and by comparing the mean right atrial pressure during ventricular diastole to the mean right ventricular diastolic pressure. Two patients with a mean pressure gradient greater than 2 mm. Hg (6 mm. in one and 17 mm. in the other) from right atrium to right ventricle during diastole were not included because of this evidence of tricuspid stenosis. Both tricuspid and mitral stenoses of one patient were treated surgically.

Results

Clinical Features (fig. 1)

Chronic Auricular Fibrillation. Fibrillation of 6 months to 15 years duration was present in 58 (96.7 per cent) of the 60 patients. Two had normal sinus rhythm. Of 146 patients with rheumatic mitral lesions studied in this laboratory, the incidence of tricuspid insufficiency in those with atrial fibrillation was considerably and very significantly greater (p < 0.01) than in those with normal sinus rhythm (fig. 2).

Enlargement and Pulsation of the Liver. Hepatomegaly that persisted in spite of the absence of peripheral edemas or attainment of “dry weight” by salt restriction and mercurial diuretics was found in 53 (88.3 per cent) of the patients. Definite systolic pulsation of the liver was present in only nine (15 per cent). In more than two-thirds of those with hepatomegaly the liver edge was more than two fingerbreadths below the right costal margin in the midclavicular line.

Right-sided Failure, as manifested by the chronic need for mercurial diuretics to control peripheral edemas, was present in 41 (68.3 per cent) patients. It was considered severe (two or more mercurial injections per week) in 21

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**Fig. 1.** Incidence of clinical features in 60 cases of tricuspid insufficiency. Scale in per cent. Number of cases is encircled.

**Fig. 2.** The incidence of auricular fibrillation (A.F.) and normal sinus rhythm (N.S.R.) and tricuspid insufficiency (T.I.) among 146 cases of rheumatic mitral disease.
and moderate (one or less mercurial per week) in 20.

**Distension and Pulsation of Neck Veins.** Twenty-nine (48.3 per cent) patients had definite distension of these veins in the sitting position, but only seven (11.7 per cent) had associated systolic pulsations. As will be seen subsequently, patients with pulsating neck veins had a higher mean and wider pulse pressure in the right atrium than those without.

*Edema* of the ankles or pretibial region of slight degree was present on admission in 22 (36.7 per cent) patients. Treatment with a low salt diet and mercurial injections resulted in disappearance of the edema in all prior to cardiac catheterization.

*Ascites* was present on admission in 11 (18.3 per cent) patients. Only three gave a history of chronic recurrent ascites that required frequent paracenteses for control.

*Murmur of Tricuspid Insufficiency,* that is a systolic murmur loudest at the lower left border of the sternum and of different quality than the apical murmur of mitral insufficiency, was infrequently found. In all 11 (18.3 per cent) patients with such a murmur the clinical diagnosis of tricuspid insufficiency was made.

*Jaundice* was observed in only two patients. Their plasma bilirubin was 2.9 and 3.4 mg. per 100 cc. Both had advanced signs of tricuspid insufficiency.

*Orthopnea* of variable degree was present in 50 patients (83.4 per cent) and was not related to the degree of tricuspid insufficiency as estimated either clinically or physiologically. Of the 10 patients (16.6 per cent) without orthopnea only one had classic clinical signs of tricuspid insufficiency.

*Cyanosis* was present in only eight (13.3 per cent) patients.

**Clinical Diagnosis of Tricuspid Insufficiency** was made in only 14 (23.3 per cent) patients. All had two or more of the following signs: systolic pulsation of the veins and liver, ascites, murmur of tricuspid insufficiency.

**Electrocardiographic Alterations**

Electrocardiographic tracings were available in 58 cases with complete unipolar leads in 53. In 32 (60.4 per cent) of the tracings, QRS in lead V₁ was less than 7 mm. (0.7 millivolts) in amplitude (figs. 3 and 4). This incidence of a low amplitude QRS in V₁ was statistically significant (p < 0.01) as compared with a group of 15 patients with mitral stenosis, auricular fibrillation and no tricuspid insufficiency, in which the change occurred only once. It was found only twice in a group of 20 patients with mitral...
stenosis and normal sinus rhythm. The small complex in V1 was of the rsR' or incomplete right bundle branch block pattern in 14 instances (fig. 3).

The onset of the intrinsicoid deflection was normal (less than 0.03 second) in 14 (26.4 per cent) cases and prolonged on the average to 0.051 second in 39 (73.6 per cent). The average time for the whole series, 0.041 second, was longer than the average time of 0.027 second found in 15 patients with mitral stenosis, auricular fibrillation and no tricuspid insufficiency.

Right axis deviation was present in 25 (43.1 per cent). Eighteen (34 per cent) had a typical10 or highly suggestive pattern of right ventricular hypertrophy. In 31 (58.5 per cent) the S waves were prominent in four or more precordial leads.

Roentgenograms and Angiocardiograms

Right atrial enlargement of moderate to giant degree was present in 50 (84.7 per cent) instances. It was slight in the remaining cases. Overall cardiac enlargement was the rule and in general a close relationship between the size of the atrium and degree of cardiac enlargement was found (fig. 5).

The angiocardiograms confirmed these findings with one exception; a right atrium considered to be slightly enlarged in the conventional roentgenograms was found to be within normal limits. The angiocardiographic “jet sign” (fig. 6), a filling defect produced in the opacified right atrium by the regurgitant blood stream,11 was found in only seven of the most severe cases. In all instances the angiocardiograms revealed a large left atrium, distended pulmonary veins and arteries—the pulmonary congestive phenomena typical of advanced mitral disease. Angiocardiography was of aid in distinguishing between right and left atrial enlargement, particularly in those cases in which the left atrium protruded so far to the right as to form most of the right cardiac border and thereby obscure the true size of the right atrium (fig. 6).

Hemodynamic Data

A right atrial pressure curve with the typical pattern of tricuspid insufficiency and two normal tracings are shown for comparison in fig-
FIG. 7. Right atrial tracings and simultaneously recorded lead II from patients with mitral stenosis. In A, atrial contraction wave (normal rhythm) is followed by a sharp decrease in pressure occurring during ventricular systole. In B, atrial systole is absent (auricular fibrillation) and decrease in pressure during systole is less pronounced. In C, decrease of pressure during ventricular contraction is replaced by a sharp increase in pressure that persists throughout systole and has a peak-plateau contour characteristic of tricuspid insufficiency. Calibration in mm. Hg. Left scale applies to A. and B.; right to C.

FIG. 8. Right atrial pressure tracing and lead II during exercise from a 39 year old male with advanced mitral stenosis and organic tricuspid insufficiency (autopsy). The dome of the regurgitant wave reaches a level of 53 mm. Hg.

during the resting state and 53 mm. Hg during exercise in one of the patients in this series (fig. 8). The invariable increase in height of the regurgitant pressure wave during exercise was often helpful in confirming a pattern of tricuspid insufficiency that was not very definite at rest (fig. 9).

In table 1 the hemodynamic data obtained in the 60 patients with tricuspid insufficiency is statistically compared with similar data from
GONZALO SEPULVEDA AND DANIEL S. LUKAS

Fig. 9. Right atrial pressure tracings and lead II during rest (A.) and exercise (B.) from a 48 year old female with tight mitral stenosis. The tracing at rest is suggestive but not diagnostic of tricuspid insufficiency. During exercise pressure increases briskly and characteristic contour appears.

Table 1.—Hemodynamics in Mitral Disease. Comparison of a Group of 60 Patients With Tricuspid Insufficiency and a Group of 20 With Auricular Fibrillation and No Tricuspid Insufficiency

<table>
<thead>
<tr>
<th></th>
<th>T.I.</th>
<th>No T.I.</th>
<th>Significance of Difference Between Means: p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac output, L./min./sq.M. B.S.A.</td>
<td>Mean and std. deviation</td>
<td>Mean and std. deviation</td>
<td></td>
</tr>
<tr>
<td>Rest</td>
<td>1.94 ± 0.42</td>
<td>2.03 ± 0.21</td>
<td>&lt;0.5 &gt; 0.4*</td>
</tr>
<tr>
<td>Exercise</td>
<td>2.29 ± 0.62</td>
<td>2.72 ± 0.30</td>
<td>&lt;0.02 &gt; 0.01</td>
</tr>
<tr>
<td>Right ventricular pressure, mm. Hg</td>
<td>75/8 ± 28/5</td>
<td>45/5 ± 18/2</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Rest</td>
<td>93/12 ± 32/5</td>
<td>64/5 ± 25/4</td>
<td>&lt;0.5 &gt; 0.02</td>
</tr>
<tr>
<td>Exercise</td>
<td></td>
<td>30-101/0-13</td>
<td></td>
</tr>
<tr>
<td>Right atrial mean pressure, mm. Hg</td>
<td>10 ± 6</td>
<td>4 ± 2</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Rest</td>
<td>16 ± 8</td>
<td>6 ± 2</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Exercise</td>
<td></td>
<td>3-10</td>
<td></td>
</tr>
<tr>
<td>Pulmonary vascular resistance, dynes-sec.-cm.⁻¹</td>
<td>643 ± 493</td>
<td>286 ± 154</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Rest</td>
<td>110-2105</td>
<td>117-594</td>
<td></td>
</tr>
<tr>
<td>Mitral valve area, sq. cm.</td>
<td>0.9 ± 0.4</td>
<td>1.0 ± 0.3</td>
<td>&lt;0.4 &gt; 0.3*</td>
</tr>
<tr>
<td></td>
<td>0.5-2.0</td>
<td>0.6-1.8</td>
<td></td>
</tr>
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</table>

* Not significant.

20 patients with mitral disease, auricular fibrillation and no evidence of tricuspid insufficiency. The mean calculated mitral valve orifice area was the same in both groups. Pulmonary vascular resistance was significantly higher in the tricuspid insufficiency group. The mean right atrial pressure in those with tricuspid insufficiency was 10 mm. Hg at rest and 17 mm. during exercise. Both values were definitely higher than those in the group without tricuspid insufficiency, in which the resting value was 4 mm. and 6 mm. during exercise. Cardiac output during exercise tended to be lower in the group with tricuspid insufficiency.

An attempt was made to determine whether there is any relation between the mean pressure or pulse pressure in the atrium and the appearance of systolic pulsations of the neck.
veins and liver. The mean atrial pressure (14 ± 8 mm. Hg) and pulse pressure (11 ± 6 mm. Hg) in the group with pulsation of the veins and liver were greater than in those without these signs, in whom mean pressure was 9 ± 5 mm. and pulse pressure 7 ± 3 mm. Both differences were statistically significant (p < 0.01 and p < 0.02 > 0.01, respectively). The clinical diagnosis of tricuspid insufficiency was made more often when both these pressures were high.

Since venous hypertension is an essential feature of tricuspid insufficiency, it was considered worthwhile to compare the clinical features of 22 patients with mean atrial pressures greater than 10 mm. Hg (a definitely elevated level corresponding to 136 mm. H₂O) with 37 whose pressures were below this level. There is a definite trend for the various manifestations of the disease to be more frequently encountered in the group with higher pressures (fig. 10). The difference in incidence of these signs in the two groups was statistically significant in the case of edema (p < 0.01), ascites (p < 0.01), liver pulsation (p < 0.02 > 0.01) and pulsation of the neck veins (p < 0.05 > 0.02). Also the pulmonary vascular resistance was greater in the group with higher atrial pressures (p < 0.02 > 0.01).

**Comments**

The right atrial pressure tracings of fully 41.6 per cent of patients with chronic rheumatic heart disease and advanced mitral valvular involvement were found to be diagnostic of tricuspid insufficiency. What proportion had organic alterations in the valve as opposed to a purely functional insufficiency cannot be stated with certainty since it is difficult to distinguish the two conditions by either clinical or physiologic means. Only in the rare case in which signs of tricuspid stenosis accompany those of insufficiency is an organic lesion of the valve certain. The problem is somewhat complicated by the fact that an early tricuspid diastolic murmur may occur in functional tricuspid insufficiency.12

It has been suggested that a regurgitant wave of very large amplitude in the right atrial pressure curve is indicative of organic tricuspid disease.18 In some of our patients the magnitude of the right atrial pressure and the severity of the clinical picture did indeed suggest an organic lesion. Autopsy in one revealed definite chronic rheumatic tricuspid valvulitis. In another, however, the tricuspid leaflets were normal and in a third they were only slightly thickened. Of four patients with moderate or severe clinical and physiologic tricuspid insufficiency studied at necropsy by Müller and Shillingford12 only one had rheumatic disease of the valve. The others demonstrated hypertrophy and dilatation of the right atrium and ventricle and a dilated valve ring.

Organic tricuspid disease is by no means rare. It has been found in almost one third of the cases of chronic rheumatic heart disease studied at autopsy.11-20 A mitral lesion was present in 90 per cent of the cases but in only 23 per cent did it occur as an isolated lesion.

Multiple factors contribute to the development of functional tricuspid insufficiency or the aggravation of a minor organic leak in severe mitral stenosis. Obstruction at the mitral orifice and pulmonary arteriolar sclerosis combine to produce a degree of pulmonary arterial and right ventricular hypertension that is more pronounced than in most other forms of acquired heart disease.1-5, 21-23 Stretching of the tricuspid valve ring follows the inevitable hyper-
trophy and dilatation of the right ventricle that has been chronically subjected to such a large work load and perhaps damaged by previous rheumatic myocarditis. In this regard it is significant that the patients with tricuspid insufficiency had higher pulmonary vascular resistances and right ventricular pressures than those without insufficiency but with atrial fibrillation and a similar degree of mitral stenosis. Because of the magnitude of the pressure gradient across the tricuspid valve during ventricular systole, a large regurgitant flow may occur through a relatively small insufficient orifice. Once established, tricuspid insufficiency adds another load on the right ventricle.

The occasionally transient nature of functional tricuspid insufficiency has been recognized clinically for many years and has been confirmed by Bloomfield and associates, who demonstrated in some patients reversion of the configuration of the right atrial pressure curve from one characteristic of tricuspid insufficiency during cardiac failure to normal after compensation was restored. However, there would appear to be a point in the course of a patient with mitral stenosis when dilatation of the right ventricle and tricuspid valve ring is so advanced that normal valvular function is not restored with compensation.

Atrial fibrillation, which was present in almost all of our patients, undoubtedly plays a role in the development or aggravation of functional tricuspid insufficiency. Little has demonstrated that atrial systole is essential to normal closure of the atriocentrical valves. Atrial systole creates an increase in pressure that is transmitted to the ventricle. Transmission of this pressure wave is so delayed that it is at its peak in the right ventricle when the atrium is relaxing. A gradient of pressure from ventricle to atrium is thus established and is sufficient to produce adequate closure of the valve just prior to the onset of ventricular systole. When the ventricle contracts without a preceding atrial systole, the valves swing close with a hinge-like movement during early systole. Because of this delay in closure, regurgitation through the incompletely approximated valve edges occurs readily.

Frequent organic deformities of the valve, dilatation of the valve ring, auricular fibrillation and severe modifications of the pulmonary circulation appear to contribute to the high incidence of tricuspid insufficiency in mitral stenosis. The respective importance of each of these factors in the individual patient cannot be readily assessed.

In 1868 Duroziez stated that involvement of the tricuspid valve is suggested when a cyanotic patient with distended neck veins, at times with edema and ascites, is able to maintain recumbency without discomfort. If to such a description is added auricular fibrillation, systolic pulsation of the liver and neck veins and a systolic murmur at the lower left sternal border, the unequivocally diagnostic picture is completed. The present evidence, however, clearly indicates that almost all of these signs occur with relative infrequency and that they are most apt to be present in patients with very high right atrial pressures and severer degrees of regurgitation. If the diagnosis of tricuspid insufficiency is dependent on recognition of these features, it will be made in only one fourth of the cases with definite physiologic manifestations of the lesion.

The high incidence of orthopnea among our patients deserves comment. All patients had mitral stenosis with the hemodynamic and roentgenographic alterations in the lungs and the pulmonary symptoms characteristic of this lesion. Why tricuspid insufficiency should cause disappearance of orthopnea when other clinical and physiologic evidences of severe mitral stenosis persist is not readily explained. Certainly the majority of the patients with severe tricuspid insufficiency manifested considerable orthopnea. Among those without this symptom only one had the other associated classic features of severe tricuspid insufficiency.

The transient nature of the murmur of tricuspid insufficiency, the difficulty in distinguishing it from the murmur of mitral insufficiency, its accentuation by inspiration have been noted in the literature. The murmur is not always accentuated by inspiration when mitral disease coexists. Although it is usually heard best along the left sternal border in the fourth and fifth intercostal spaces, it can be
heard above this point or close to the apex. The systolic murmur attributed to mitral insufficiency in a number of our patients in whom no evidence of mitral insufficiency was found at surgery could have been due to tricuspid insufficiency.

In contrast with the infrequency of the classic signs of tricuspid insufficiency, auricular fibrillation, persistent enlargement of the liver and increased size of the right atrium were the most constant findings. These signs in a patient with rheumatic mitral disease who has a history of previous right-sided failure that has required mercurial administration for control should suggest the diagnosis of tricuspid insufficiency. The sheer frequency of the lesion in patients with auricular fibrillation lends considerable support to such a diagnosis.

A QRS complex in V1 of small amplitude (i.e., less than 0.7 millivolt) and with a delayed onset of intrinsicoid deflection is the electrocardiographic feature that most consistently and with great statistical significance distinguishes patients with mitral disease and tricuspid insufficiency from those without insufficiency of the tricuspid valve. In a number of instances the small QRS in V1 was of rSR' configuration, which together with the delay in intrinsicoid deflection constituted the pattern of incomplete right bundle branch block. Because of the many factors influencing the electrocardiogram in this situation, it is not possible to state that tricuspid insufficiency alone is responsible for these alterations. However, in 62.6 per cent of 50 patients with autopsy-proved tricuspid insufficiency Aceves and Carral14 also found the onset of intrinsicoid deflection in V1 and V2 to be prolonged beyond 0.04 second. In other publications an incidence of 47 to 65 per cent of right bundle branch block has been cited.30, 31

According to Cabrera and Monroy,32 the characteristic electrocardiographic pattern associated with a lesion that produces diastolic overloading of the right ventricle (i.e., interatrial septal defect) in contrast to one producing systolic overloading (i.e., pulmonic stenosis) is complete or incomplete right bundle branch block. Tricuspid insufficiency is a diastolic overloading lesion. Electrocardiograms taken before and after the onset of tricuspid insufficiency in a patient with mitral stenosis and published by these authors demonstrate a decrease in the voltage of QRS in V1 from 0.9 to 0.4 millivolt after the tricuspid regurgitation had developed.33

Persistent hepatomegaly, enlargement of the right atrium and many of the other signs of tricuspid insufficiency are essentially the result of right atrial and systemic venous hypertension. Since right ventricular failure may produce these changes, their diagnostic usefulness may be questioned. The fact remains, however, that the right atrial and venous pressures are chronically and persistently higher (in tricuspid insufficiency, despite vigorous cardiac therapy) than when the valve is competent. Continued elevation of the venous pressure and signs thereof after full cardiac compensation is achieved can be regarded as presumptive evidence of a tricuspid lesion.

By its congestive effect on the venous system and consequent elevation of hydrostatic pressure in the venules and capillaries, tricuspid insufficiency is conducive to the development of edema even if the right ventricle is functioning normally. Aside from increasing filtration of fluid through the peripheral capillaries, venous hypertension promotes edema in other ways. There is evidence that elevation of the renal venous pressure above 150 mm. H2O is followed by a decrease in excretion of sodium and water because of augmented tubular reabsorption.31 More recently it has been demonstrated that congestion of any sizable segment of the peripheral venous bed induces similar changes in renal function.34-36 Objection has been raised to the application of these observations to the problem of edema in chronic cardiac failure because of the acute nature of the experiments. However, the marked and brisk rise in atrial and venous pressures that occurs with even mild exercise in tricuspid insufficiency represents an acute experiment that may be repeated many times in the course of the patient's day. These considerations are important in explaining the ease with which patients with tricuspid insufficiency accumulate
edema and the frequency of mercurial diuretic administration required for its control. We have therefore applied the term right-sided failure rather than right ventricular failure to patients with tricuspid insufficiency.

There is a need for the recognition of factors that may limit the beneficial effects of corrective surgical procedures on the mitral valve. Tricuspid insufficiency is such a factor. We have seen occasional patients with mitral stenosis and severe tricuspid insufficiency who had some relief of pulmonary symptoms after mitral valvuloplasty but who continued to manifest signs of tricuspid insufficiency and recurrent edema and ascites. Although it is not a contraindication to the operation, the impression is that patients with tricuspid insufficiency do not improve either hemodynamically or clinically after mitral valvular surgery as well as those without the lesion. An adequate and long term evaluation of many patients is needed before this impression can be substantiated. Such a study is thwarted from the start if the tricuspid insufficiency with its distinct hemodynamic effects is not recognized preoperatively.

**Summary**

Of 146 patients with rheumatic heart disease and predominant mitral valve involvement studied by cardiac catheterization, 60 were found to have a right atrial pressure curve characteristic of tricuspid insufficiency. The clinical diagnosis had previously been made in only 23.3 per cent of the cases. All the patients who had cardiac failure were under optimum medical control at the time of the study.

The most constant clinical features were auricular fibrillation, persistent liver enlargement, a history of right-sided failure and roentgenographic evidence of moderate to severe enlargement of the right atrium. A small QRS complex, frequently of the rSR' pattern, in lead V1 of the electrocardiogram was found in 60.4 per cent of the cases.

The pulmonary vascular resistance, right atrial mean and right ventricular pressures were distinctly greater as compared with the values in a group of patients with auricular fibrillation, a similar degree of mitral involvement but no tricuspid insufficiency.

The classical clinical features of tricuspid insufficiency were present relatively infrequently and were seen more often in patients with right atrial mean pressures greater than 10 mm. Hg.

Tricuspid insufficiency, functional or organic, is a frequent complicating lesion in mitral valvular disease and its presence should be suspected in any case with auricular fibrillation, persistent liver enlargement and definite increase in size of the right atrium.

**Summario in Interlingua**

In 60 inter 146 patientes de cardiopathia rheumatic e predominante affectation del valvula mitral, un studio a catheterisation cardiac revelava un curva de pression dexteroatrial del typo characteristic de insufficientia tricuspide. Iste diagnose habeava previemente essite establite per medios clinic in solo 23,3 pro cento del casos. Omne le patientes con insufficientia cardiac esseva sub optime surveilantia durante le studio.

Le plus constante tractos clinic esseva fibrillatio auricular, persistente allargamento hepatic, un historia de insufficientia del latere dexter, e evidentia roentgenographic de moderate o sever allargamento del atrio dexter. In 69,4 pro cento del casos nos constatava un parve complexo QRS—frequentemente del configuration rsR'—in le derivation V1 del electrocardiogramma.

Le resistentia pulmono-vascular, le pression median dexteroatrial, e le pression dexteroventricular esseva distinctemente plus grande que in un gruppo de patientes con fibrillation auricular, un simile grado de affection mitral, sed nulle insufficientia tricuspide.

Le classic caracteristicas clinic de insufficientia tricuspide esseva relativamente infrequente e se monstrava predominantemente in patientes con pressiones median dexteroatrial de plus que 10 mm Hg.

Insufficientia tricuspide—o functional o organic—es un frecuente complication in casos de morbo del valvula mitral. Su presentia debe esser suspectate in omne casos mostrante fibrillation auricular, persistente allargamento
hepatic, e definite aumento del dimension del atrio dextere.

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