Pathophysiology of Tricuspid Insufficiency
—Clinical and Experimental Study—

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Although clinical manifestations of tricuspid insufficiency (TI) have been interested since early nineteenth century, and its combination with other heart disease makes the treatment of patients difficult, and makes their prognosis worse, the methods for the diagnosis of this disease and its diagnostic criteria have not been settled firmly. And the mechanism of symptoms of TI have not been fully clarified.

We analysed symptoms of our clinical patients with TI, and performed animal experiments to investigate the mechanism concerning pathogenesis of TI.

Clinical Studies

Materials

One hundred and twenty three patients hospitalized to our department in the past 10 years were diagnosed to have TI by means of auscultation, PCG, cardiac catheterization and jugular phlebogram. Etiology of these 123 cases is shown in Table I. One hundred and two cases had rheumatic valvular disease, and 83 of them had mitral stenosis. Seven cases had TI complicating with congenital heart disease, of which 5 cases had ASD, one PS and one PS with VSD.

It seems interesting that 10 cases with primary myocardial disease (PMD) have TI, and it may suggest the role of myocardial factor on the pathogenesis of TI.

Key Words:
Functional tricuspid insufficiency
Organic tricuspid insufficiency
Vena caval flow velocity
"Ventricularization" of right atrial pressure
Ventricular function curve

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<th>TABLE I</th>
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<td>1) Rheumatic valvular dis.</td>
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<td>2) Congenital heart dis.</td>
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Of 6 cases of TI due to other etiologies, 2 had cor pulmonale, one myocardial infarction, one progressive muscle dystrophy, one hyperthyroidism and one isolated TI. It has been stated that cor pulmonale rarely produce tricuspid regurgitation and this reflects probably the shorter course of this disease, the normal valve leaflets, and the rarity of atrial arrhythmia1,2.

We have no cases with TI due to carcinoid, trauma, bacterial endocarditis and congenital lesion.

Symptoms

Of 102 with TI associated with rheumatic valvular disease, more than half cases had edema, but of 100 control cases with rheumatic valvular diseases, not accompanying TI, only 15% had edema, and ascites was observed in 20% of TI group but was not found in control group. As shown in Fig. 1, elevation in venous pressure, hepatic enlargement and cardiac enlargement were found in much higher percentages in TI.
group than the control group. But, on the other hand, it is noteworthy that there are many cases with normal venous pressure and normal cardiac size in TI group.

I) Cardiac output

It is generally recommended that the patients with TI of rheumatic valvular disease have low cardiac output, develop severe congestive heart failure and die earlier than patients without tricuspid diseases.

In our cases, 52% of patients with TI had low cardiac index (less than 2.5 l/min/M²) whereas only 25% of the control patients had low cardiac index. One of the causes of low cardiac output in cases with TI is that regurgitation from right ventricle into right atrium reduces the effective forward output into pulmonary artery. It was demonstrated that cardiac output correlated with the severity of tricuspid regurgitation and that the cardiac output was decreased by physical exercise, and at the same time the regurgitant volume was increased and venous pressure was elevated. This decrease in cardiac output by the backflow of the blood due to TI reduced the pulmonary congestion, and results would be less dyspnea in exertion than expected from the degree of associated mitral stenosis.

II) Venous pressure, right atrial pressure and jugular phlebogram

Well-known characteristic features of TI are raise of venous pressure, systolic pulsation of jugular vein and positive wave or "ventricularization" of right atrial pressure contour.

The cases with venous pressure exceeding 200 mm H₂O were observed in 31% of our TI group, and in only 2% of the control group. Thus a considerable number of cases with very high venous pressure (higher than 200 mm H₂O) have also TI, in accordance with McMitchel's finding.

Cardiac catheterization was performed in 58 cases of TI group, and ventricularization of right atrial pressure was observed in only 4 cases, and diagnosis of TI from atrial contour was difficult in most cases. The reason for right atrial pressure to show hardly the typical TI pattern even in the presence of regurgitant flow might be that right atrium is dilated in TI and pool size of venous side is enlarged, and the pressure pattern itself is not affected by regurgitation of blood by damping of pressure.

In contrast with right atrial pressure, jugular phlebogram is seemed useful for diagnosis of TI. Positive wave was observed in 25 cases of TI groups, but 22 cases of them had atrial fibrillation. In normal atrial pressure pattern, X descent is deeper than Y descent, and in TI cases X descent becomes shallower and finally shows positive pressure but in cases with atrial fibrillation, the absence of atrial contraction causes diminution of X descent or positive V wave, and makes the difference from the pressure pattern due to TI obscure. Therefore, it is

considered that the diagnosis of TI by jugular phlebogram has also some limitation.

III) Right ventricular and pulmonary arterial pressure

Pressure load to the right ventricle is an important factor as the underlying cause of functional TI. The elevation of right ventricular pressure causes right ventricular hypertrophy and later its dilatation. With this, the enlargement of atrioventricular annulus occurs and blood then regurgitates from the ventricle into the atrium. Coelho observed the relationship between pulmonary arterial systolic pressure and the severity of TI, but Weber did not find such a correlation, although he found a correlation between right ventricular enddiastolic pressure and the severity of TI.

In our patients, TI group had higher pulmonary systolic pressure than the control group as shown in Fig. 2, and pulmonary arterial pressure of the patients who had small cardiac size (C–T ratio less than 55%) and normal sinus rhythm were much higher than that of cases with atrial fibrillation. This finding suggests that atrial fibrillation may facilitate tricuspid regurgitation under relatively low pressure load, and this might
be explained by the reason that the lack of contraction of atroventricular ring by atrial systole leads to inadequate closure of tricuspid valve in ventricular systole, and causes tricuspid regurgitation. The relationship between pulmonary arterial pressure and venous pressure was investigated as shown in Fig. 3. in patients with TI. The cases with venous pressure less than 120 mm H₂O were 11 in 17 patients with sinus rhythm, and 3 in 14 patients with atrial fibrillation. Pulmonary arterial pressures were higher than 60 mmHg in 15 cases, of which 4 cases had atrial fibrillation and 22 cases had normal sinus rhythm, and venous pressure of the latter 11 cases were less than 120 mm H₂O. This is indicating that there are 2 extreme subgroups in the cases with TI. One is consisted of the cases with typical (or classical) symptoms of TI, such as a high venous pressure, edeme, cardiac enlargement and atrial fibrillation, and another subgroup is consisted of cases, who have only high pulmonary arterial pressure and did not have other symptoms of TI. This fact suggests that not only the pressure load on the right ventricle but also volume load, myocardial contractility, cardiac rhythm and other factors have important contribution to the appearance of symptoms of TI.

We performed animal experiments to clarify the pathogenesis of TI from this view point.

**ANIMAL EXPERIMENTS**

**Methods**

The experiments were carried out in mongrel dogs anesthetized with pentobarbital. They were thoracothomized using a positive pressure respirator. Aortic pressure and right atrium pressure were measured through catheters introduced from femoral artery and jugular vein respectively. Right ventricle was punctured by needle to register the pressure.

For measurement of vena caval flow velocity, a catheter-tip electromagnetic flowmeter was introduced into the vena cava from the femoral vein, and its tip was placed just below the junction of inferior vena cava and right atrium. Then catheter type microphone was inserted into the right atrium near tricuspid valve from jugular vein, and the detection of regurgitant murmur indicated the occurrence of TI.

In some dogs, organic TI was produced with scissors introduced through the right atrial appendage, with which either the valve, the chorda tendinae or the both were cut. Then the pressures, IVC flow velocity and PCG in right atrium were registered simultaneously under the condition of pressure load by pulmonary arterial constriction or of volume load by infusion of...
large dose of saline. Finally the effects of propranolol or isoproterenol on these hemodynamic changes were investigated.

Results


1) IVC flow velocity

The normal IVC flow velocity pattern is shown in Fig. 4. This pattern has an inverse relationship with the contour of right atrial pressure. At the time of atrial contraction ("a"
wave in right atrium pressure), the IVC flow velocity is almost 0 or sometimes negative, and this velocity has a peak during ventricular systole. Indicating the blood returns from vena cava to right atrium in the highest speed. In some dogs, the second peak of IVC flow velocity is
observed during ventricular diastole.
II) Effect of pulmonary artery constriction and volume load

Pulmonary artery was constricted by a tape surrounding its main trunc, then right ventricular pressure was elevated. (Fig. 5) By this maneuver systolic IVC flow velocity was reduced, and finally reversed to negative value, indicating the occurrence of TI, and intra-auricular PCG recorded regurgitant murmur. Although IVC

*Japanese Circulation Journal  Vol. 38, October 1974*
flow pattern and intracardiac PCG showed the production of TI, the right atrial pressure was elevated slightly, and its contour was not changed essentially from the control pattern, and did not show the typical TI pressure wave. When pulmonary artery was constricted after infusion of large volume of saline, the right auricular pressure showed typical ventricularization. (Fig. 6)

III) Organic TI

IVC flow velocity showed the negative phase in systole after the production of organic TI. (Fig. 7) But right atrial pressure pattern did not show the typical TI pressure wave. The infusion of large volume of saline caused again the typical pattern of TI of atrial pressure, "ventricularization". (Fig. 7)

The right atrial pressure pattern also showed the typical "ventricularization" by administration of propranolol to the dog with organic TI, and IVC flow velocity revealed markedly negative flow in systolic phase, showing the degree of tricuspid regurgitation was increased. (Fig. 8) On the contrary, the injection of isoproterenol to the dog with organic TI reduced the negative flow velocity in systolic phase, and deepened X descent in the right atrial pressure, and hemodynamic characterist TI were abolished. (Fig. 9)

These observations were indicating that tricuspid regurgitation was made easily by pulmonary constriction or tricuspid lesion, but typical right atrial pressure pattern of TI was not recorded constantly, and after the volume load by infusion or decrease in myocardial contractility by propranolol, the right atrial pressure revealed the typical "ventricularization", and that not only the pressure load but also volume load and decrease in myocardial contractility were important factors to the pathogenesis of tricuspid insufficiency.

IV) Ventricular function

Finally we investigated the effect of TI on ventricular function by the right and left ventricular function curves made during infusion, before and after the production of organic TI (Fig. 10). In the right ventricular function curve, the descending limb was demonstrated clearly after the production of TI, however no difference was observed in the left ventricular function curves between the control and TI. But the left ventricular enddiastolic pressure could not elevate to the maximal level of the control experiment, and this showed that the increase in cardiac output by infusion was limited after the production of organic TI.

CONCLUSION

From clinical studies, it was suggested that beside pressure load on the right ventricle, volume load, myocardial contractility, cardiac rhythm and other factors might have important role on the appearance of TI. And this possibility was clarified partly by animal experiments.

In this respects, a patient with TI, who has high pulmonary pressure level but lack another classic TI symptoms will by sufficiently treated only by the management of the original lesion, and then regurgitation of tricuspid valve will be disappeared.

But a patient with TI accompanied with typical symptoms of "right heart failure", that is, edema, ascites, cardiac enlargement, liver enlargement atrial fibrillation and others should be treated regarding not only the original lesion but also correlation of function of myocardium, kidney, liver and other organs.

Acknowledgement

The authors wish to acknowledge Drs. Hisakazu Yasuda and Tetsuji Ittaka especially for their contribution during performance of the animal experiments and also to thank Prof. Satoru Maruo for his valuable advise. We are further indebted to many other colleagues whose cooperations made it possible for us to study on clinical materials.

This animal experiments were supported by the aid of The Japan Research Promotion Society for Cardiovascular Disease.

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*Japanese Circulation Journal Vol. 38, October 1974*