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RHEUMATIC TRICUSPID STENOSIS

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Incompetence of the tricuspid valve is commonly noted in clinical practice, often in association with heart failure, and has been recently intensively studied (Müller and Shillingford, 1954, 1955 ; Korner and Shillingford, 1954, 1957 ; McMichael and Shillingford, 1957). The distinction between functional tricuspid insufficiency in heart failure and that due to organic disease of the valve presents considerable difficulty. The criteria for the diagnosis of tricuspid stenosis have been well documented by Gibson and Wood (1955), Whitaker (1955), and Yu *et al.* (1956), but, since many of the diagnostic signs are frequently obscured by other cardiac lesions, the diagnosis is often missed. We are therefore presenting a combined clinical, haemodynamic, and pathological study of 21 proved cases in order to draw attention to the wide spectrum of organic tricuspid valve disease and to clarify the diagnosis in cases complicated by other lesions.

Case Material and Methods

Twenty-one cases (15 female and 6 male) of organic tricuspid valve disease have been studied. Thirteen were observed clinically, and in these the diagnosis was confirmed by haemodynamic studies in seven (two of which also came to necropsy), and by necropsy alone in the remaining six. A further eight cases were studied retrospectively by examining the necropsy and case records of 190 consecutive cases of rheumatic heart disease over a period of 10 years. The total necropsy incidence was therefore 8.4%.

All the cases had associated mitral valve disease, and 12 had aortic disease also. The severity of the associated valve lesions and of pulmonary hypertension varied widely.

Criteria for Inclusion in the Series.—All the cases coming to necropsy had definite narrowing of the valve, with fusion of the cusps and thickening and shortening of the chordae. Minor thickening of the cusps without narrowing of the valve orifice was not accepted. In life the diagnosis was regarded as proved if there was a diastolic gradient of 5 mm. Hg or more across the tricuspid valve. The gradient was measured in presystole in cases with sinus rhythm, and early diastole if atrial fibrillation was present.

Radiological Criteria: Assessment of Right Atrial Size.—The maximum distance of the right atrial border from the midline in a 6-ft. (1.8-metre) postero-anterior radiograph was measured, and enlargement was graded as follows: grade 1 (slight), up to 50 mm. ; grade 2 (moderate), 51–70 mm. ; grade 3 (considerable), 71–90 mm. ; grade 4 (aneurysmal), over 90 mm. Attention was also paid to the size of the ventricles and left atrium and to signs of pulmonary hypertension (Davies *et al.*, 1953).

Cardiographic Criteria.—Right ventricular hypertrophy was graded as follows: Grade 0, no evidence of right ventricular hypertrophy. Grade 1, dominant R wave in Lead V4R only ; and/or dominant S in V5 with normal V1 ; and/or dominant R in VR with normal V1. Grade 2, dominant R (less than 5 mm.) in V1. Grade 3, dominant R of 5 mm. or more in V1. Grade 4, qR pattern in V4R and V1 with inverted T in V4R, V1, V2, and V3 ; dominant S in V5 and dominant R in VR. P waves which were pointed and 2–2.5 mm. or more in height were regarded as evidence of right atrial enlargement (right atrial P waves). The P–R interval was considered prolonged if it exceeded 0.2 sec. Attention was also paid to evidence of left ventricular hypertrophy and to atrial fibrillation.

History and Symptoms

Rheumatic Fever.—Histories of rheumatic fever were given by 15 patients, and six of these had suffered more than one attack. One patient had a history of rheumatoid arthritis.

Duration of Symptoms.—The time interval between the first attack and the onset of symptoms in the personally observed cases was very variable. In two cases symptoms followed immediately upon the attack, while the maximum period before the onset of symptoms was 33 years. Six patients developed symptoms less than 10 years after rheumatic fever. The duration of symptoms from onset to death was less than 10 years in all but two patients (aged 20 and 26 years). The range (apart from these two cases) was 6 months to 11 years.

Pulmonary Congestive Manifestations.—Dyspnoea on effort was present in all cases, and in 11 was severe (grade 3 or 4 according to the American Heart Association Classification, 1939). Paroxysmal dyspnoea or

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pulmonary oedema occurred in six patients and bronchitis in 13. Haemoptysis occurred in five and pulmonary infarction in seven. This infarction may have been embolic in origin in two cases, since thrombus was found in the right atrium. Three patients with infarction had thrombosis of a large pulmonary artery. When a history of swelling of the ankles was obtained it was always associated with dyspnoea. Substernal pain on effort was noted in six patients, and pain due to hepatic congestion simulated angina in some cases. In general, the history did not suggest tricuspid involvement.

Physical Examination

Many of the physical findings were attributable to associated valve lesions, and only those abnormalities which appear relevant to the diagnosis of the tricuspid lesions are described.

Mitral Facies.—Cyanotic malar flush (mitral facies) was observed in five cases.

Jugular Venous Pulse.—Only the personally observed group is discussed here. Six patients had sinus rhythm, and

FIG. 1.—Jugular phlebogram and cardiogram in Case A 10 during atrial fibrillation. The phlebogram shows the large positive wave (S), indicating regurgitation of blood back through the tricuspid valve in systole. The "y" descent is not sharp, suggesting some obstruction to right ventricular inflow.

normal. In one patient (Case A 10) a giant "a" wave was replaced by a large systolic pulsation when atrial fibrillation occurred (Fig. 1).

FIG. 2.—Six-foot (1.8-metre) postero-anterior radiograph of Case C 6, showing aneurysmal right atrium (measurement 105 mm.).

Arterial Pulse.—This was of small volume in all cases, and its quality was determined by the associated valve lesions.

Rhythm.—Of the total series, 13 had atrial fibrillation and eight were in sinus rhythm.

Pulsation of Right Parasternal Region.—In 2 of the 13 personally observed cases pulsation in the left parasternal region, conventionally attributable to right ventricular enlargement, extended to the right of the sternum and was even felt in the right mid-clavicular line (Case C 6). The right atrium was greatly enlarged radiologically (Fig. 2), and it was thought that the pulsation might represent systolic expansion of that chamber. In one case there was dullness but no pulsation to the right of the sternum. One patient had a normal left ventricular impulse, five had left parasternal lift, and seven had combined right and left ventricular enlargement due to associated valve lesions.

Auscultatory Signs.—Ten of the 13 personally observed cases had a presystolic murmur at the tricuspid area, which increased on inspiration in five. In eight cases there was a mid-diastolic murmur or presystolic murmur, which in six increased with inspiration (Fig. 3). A sharp sound in early diastole, thought to be the opening snap of the tricuspid valve (Kossmann, 1955) and increasing on inspiration, was noted in three. An atrial sound at the left sternal border was present in three of the cases with sinus rhythm. In those with pulmonary hypertension the pulmonary component of the second heart sound was accentuated.

Hepatic enlargement was present in 9 of the 13 personally observed cases and in 7 of the 8 retrospective cases. It was gross (6 cm. or more below the costal margin) in 12. In three of the cases with sinus rhythm there was presystolic pulsation, while systolic pulsation occurred in six of those with atrial fibrillation.

FIG. 3.—Phonocardiogram of Case C 3 showing increase in tricuspid presystolic and mid-diastolic murmurs on inspiration.

FIG. 4.—Six-foot (1.8-metre) postero-anterior radiograph of Case A 10, showing large right atrium (measurement 95 mm.). There is also pulmonary venous and arterial hypertension.

Splenic enlargement occurred in 5 of the 21 cases.

Peripheral oedema occurred in 11 of the 21 cases, and was gross in five.

Ascites was present in four cases, and jaundice (due to cardiac cirrhosis or pulmonary infarction) in six.

Radiology

Teleradiograms were available in 13 cases. The dominant feature was right atrial enlargement (Figs. 2 and 4) which was outside the normal range in nearly all, and

FIG. 5.—Size of right atrium in 25 normal adults compared with 14 patients with rheumatic heart disease without tricuspid stenosis, and in 13 with organic tricuspid valve disease.

greater than that found in rheumatic heart disease without tricuspid stenosis in half (Fig. 5). Right ventricular hypertrophy could not be reliably assessed in the presence of marked right atrial enlargement, which often prevented assessment of vascular changes also.

Accordingly we have tabulated the radiological grade of right atrial enlargement, the cardiographic grade of right ventricular hypertrophy, and the P-R interval (Table I). Although aneurysmal dilatation of the left atrium can interfere with measurement of the right atrium, it did not do so in this series.

It will be seen that right atrial enlargement was often considerable or gross when right ventricular hypertrophy was absent or slight. It was found that the cardiogram pro-

TABLE I

Case	X-ray Film R.A. (Grade)	Cardiogram R.V. (Grade)	E.C.G.
			P-R Interval (sec.)
C 1	1	0	0.30
C 2	2	1	0.26
C 3	3	2	0.24
C 5	1	0	0.20
C 6	4	3	—
C 7	2	0	—
A 6	3	0	0.26
A 8	4	0	0.20 A.F. later
A 9	3	0	—
A 10	4	1	0.20 A.F. lateq
A 13	3	R.B.B.B.	—
A 14	4	1	—
A 15	2	1	—

A.F.=Atrial fibrillation. R.B.B.B.=Right bundle-branch block.

vided the best index of right ventricular dominance and the x-ray film of right atrial size. The association of appreciable right atrial enlargement but little right ventricular hypertrophy on the cardiogram is particularly characteristic of tricuspid stenosis.

Cardiology

Atrial Fibrillation.—This was present or developed in 13 of the 21 cases. The presence of fibrillation appeared to be related directly to right atrial size (Table II).

The P Wave and P-R Interval.—Eleven cases showed sinus rhythm at some stage, and in five of them pointed P waves exceeding 2 mm. or more in height suggested right atrial enlargement (Fig. 6). In 6 of the 11 cases the P-R interval exceeded 0.20 sec. (Fig. 7), in four it was

TABLE II.—Right Atrial Size and Rhythm in 13 Personally Observed Cases

Size of Right Atrium on X-ray Film					Total No. of Patients	Cases with Atrial Fibrillation
Grade 1	..	..	..	..	2	0
" 2	..	..	..	..	3	2
" 3	..	..	..	..	4	2
" 4	..	..	..	..	4	4

FIG. 6.—Cardiogram of Case C1. There is sinus rhythm, P waves are 3 mm. in height in lead V5, and the P-R interval is 0.36 second. There is normal ventricular balance.

0.20 sec., and in one it was 0.18 sec. A comparison between the P-R interval in tricuspid disease and rheumatic heart disease without tricuspid disease is shown in Fig. 7. It will be seen that a significantly greater proportion of cases of tricuspid disease have a prolonged P-R interval (see also Table I). Active rheumatic carditis which might prolong the P-R interval was absent in all cases. Thus a prolonged P-R interval supports the diagnosis of tricuspid stenosis, and one of less than 0.16 sec. is against it.

Right Ventricular Hypertrophy.

This was usually either absent or slight and never gross (Table III). Table I shows the discrepancy between the degree of right atrial and right ventricular hypertrophy. The association of a

FIG. 7.—The P-R interval in 11 cases of organic tricuspid valve disease compared with 25 cases of rheumatic heart disease without tricuspid stenosis.

TABLE III.—Cardiographic Signs of Ventricular Hypertrophy

	R.V.H. Grade					L.V.H.	L.B.B.B.	R.B.B.B.	R & L.V.H.
	0	1	2	3	4				
No. of patients	5	9	1	2	0	1	1	1	1

R.V.H.=Right ventricular hypertrophy. L.V.H.=Left ventricular hypertrophy. R.B.B.B.=Right bundle-branch block. L.B.B.B.=Left bundle-branch block.

FIG. 8.—Cardiogram of Case C3. There is sinus rhythm. The P waves are 3-4 mm. in height in V1, and the P-R interval is 0.26-0.28 second. There is evidence of modest grade 2 right ventricular dominance, V4R and V1 showing a dominant R wave.

long P-R interval (in the absence of active carditis), right atrial P wave, and normal ventricular balance in a patient with rheumatic heart disease should at once suggest the diagnosis of tricuspid stenosis (Fig. 6). The presence of only slight or moderate right ventricular dominance, with a right atrial P wave and long P-R interval, should also arouse suspicion (Fig. 8).

Haemodynamic Data

Cardiac catheterization was performed in eight cases. In two (C 6 and A 10) the catheter could not be advanced into the right ventricle because of the enormous right atrium and the obstruction offered by the stenosed tricuspid valve. In one of these cases a gradient across the tricuspid valve was recorded at operation for mitral valvotomy, and in the other the diagnosis was confirmed at necropsy.

TABLE IV.—Haemodynamic Studies in 8 Cases

Case	Right Atrial Pressure (mm. Hg)			Right Ventricular Pressure (mm. Hg)		R.A./R.V. Diastolic Gradient (mm. Hg)	Pulmonary Arterial Pressure (mm. Hg)		Pulmonary "Wedge" Pressure (mm. Hg)	Cardiac Output (l./min.)
	a	v	y	Systolic	Diastolic		Systolic	Diastolic		
C 1	20-30	4	3	25	-7	25	24	6	11	3.1
C 2	14.5	42	1	50	2.5	12	55	24	27	2.8
C 3	5	2	1.5	47	-1.5	8	51	25	16	2.0
C 5	24	—	14	40	10	10	36	18	—	5.2
C 6	A.F.	30	—	53	6	11	56	30	18	—
C 7	A.F.	22	12	55	6	6	55	20	30	3.2
A 9	A.F.	12	5	35	0	5-10 (variable)	35	13	16	2.4
A 10	A.F.	—	—	—	—	—	—	—	—	—

a=Height of peak "a" wave above baseline (5 mm. below sternal angle)
v=Height of peak "v" wave above baseline. y=Height of trough of "y" wave above baseline.

Table IV gives the relevant data, and it will be seen that a gradient of from 5 to 25 mm. Hg was recorded in diastole across the tricuspid valve in the other six cases (Figs. 9 and 10). The pulmonary artery "wedge pressure" was

FIG. 9.—Cardiac catheterization record during withdrawal from right ventricle to right atrium (Case C 1). The "a" wave is considerable (25 mm. Hg), and the end-diastolic gradient across the tricuspid valve is 25-30 mm. Hg. In two cycles when the catheter tip is at the tricuspid valve, the "a" wave and ventricular pulse can be seen consecutively. The "y" descent of the atrial pulse is not precipitous.

FIG. 10. — Cardiac catheterization record showing right ventricular, followed by right atrial, pressure pulses (Case C 2). The "a" wave is 14 mm. Hg and the presystolic gradient across the tricuspid valve 12 mm. Hg. Note that there is no gradient in early diastole.

considerably raised in two patients, moderately raised in three, and normal in one. Pulmonary arterial hypertension was appreciable in four cases and was absent in one. The cardiac output was low in all except one case.

Clinico-pathological Correlation

The normal tricuspid valve orifice is usually stated to be 130 mm. in circumference. We have taken a circumference of 70 mm. or less as indicating "severe" stenosis, and 71-120 mm. as "slight" stenosis. It should be emphasized that this measurement indicates the effective valve orifice and not the valve ring, which is often enlarged owing to

FIG. 11.—Necropsy specimen of Case C 2, showing tricuspid valve from the ventricular aspect. The valve orifice is greatly narrowed (circumference 60 mm.). On examination it appeared rigid and grossly distorted.

dilatation of the right ventricle. Where exact measurements were not recorded in necropsy records, we have regarded an orifice which would not admit two fingers as being less than 70 mm. in circumference.

Only four cases had marked stenosis, and only two were personally observed (C 2 and A 14, Figs. 11 and 12). Case C 2 had definite clinical signs suggesting tricuspid stenosis, with giant "a" wave in the jugular venous pulse, long P-R interval, and enlarged right atrium. She had moderate pulmonary arterial hypertension (moderately increased pulmonary resistance) and considerable pulmonary venous hypertension. Case A 14 had gross and unremitting tricuspid incompetence, prolonged congestive heart failure, a tricuspid systolic (but no diastolic) murmur, and clinical signs of considerable pulmonary hypertension. The signs of obstruction to the tricuspid valve were masked by atrial fibrillation, pulmonary hypertension, and critical mitral stenosis.

The valve cusps of the personally observed cases

FIG. 12.—Necropsy specimen of Case A 14, showing tricuspid valve from the atrial aspect. The right atrium is greatly enlarged and the tricuspid valve distorted, rigid, and narrowed (circumference 70 mm.).

were thickened, irregular, and rigid (Figs. 11, 12, and 13). Their appearance suggested that incompetence had been present in life, and clinical evidence of this had been found

FIG. 13.—Necropsy specimen of Case A 9, showing tricuspid valve from the atrial aspect. Right atrium considerably enlarged, tricuspid valve moderately narrowed, and cusps fused (circumference 100 mm.).

in five of these patients. Incompetence had been judged slight in one and severe in four. In one of these, Case C 10, the venous pulse showed a good X descent while sinus rhythm was present, but developed a large systolic wave when atrial fibrillation supervened (Fig. 1).

Spectrum of Tricuspid Valve Disease.—The clinical picture resulting from tricuspid disease varies considerably according to the severity of the tricuspid stenosis, the presence of atrial fibrillation, and the pres-

ence and severity of associated valve lesions. This is illustrated by reference to five cases in the present series.

The Varying Clinical Picture

Case C 1 : Moderately Severe Tricuspid Stenosis with Mild Mitral Stenosis and Normal Pulmonary Pressures

Woman aged 51 with tiredness and moderate dyspnoea (grade 1-2) on exertion for 10 years. Three attacks of rheumatic fever. **Physical signs.**—Arterial pulse small; regular. Jugular venous pulse; giant "a" wave. Cardiac impulse normal. Tricuspid presystolic murmur. B.P. 140/85. P2 normal. Mitral presystolic murmur and opening snap. Mitral first sound accentuated. Liver enlarged and pulsating in presystole. Fourth heart sound. **Cardiography.**—Right atrial P wave. Long P-R interval. Normal ventricular balance (Fig. 6). **Radiology.**—Slight but definite enlargement of right atrium. Slight enlargement of left atrium. Right ventricle and pulmonary vascular pattern normal. **Clinical diagnosis.**—Tricuspid stenosis; mitral stenosis. **Haemodynamic studies.**—Gradient, maximum in presystole, across tricuspid valve. Minimal pulmonary hypertension (Fig. 9).

Conclusion.—Tricuspid and mitral stenosis. Clear clinical evidence of tricuspid disease not obscured by other valve lesions or atrial fibrillation. Mitral valve lesion not severe enough to warrant valvotomy. Tricuspid stenosis moderately severe, but tricuspid valvotomy not advised as symptoms not urgent.

Case C 3 : Tricuspid Stenosis Associated with Critical Mitral Stenosis and Moderate Pulmonary Arterial Hypertension

Woman aged 48. Six years' increasing dyspnoea (grade 2-3). No history of rheumatic fever. **Physical signs.**—Mitral facies. Arterial pulse small and regular. Jugular venous pulse: giant "a" wave. B.P. 120/90. Cardiac impulse; moderate right ventricular hypertrophy. Tricuspid systolic and diastolic murmur. Mitral presystolic murmur and opening snap. Fourth heart sound. P2 accentuated, normal split (Fig. 3). **Cardiography.**—Right atrial P waves. Moderate right ventricular dominance. Long P-R interval (Fig. 8). **Radiology.**—Right atrium considerably enlarged. Left atrium moderately enlarged. Right ventricle slightly enlarged. Pulmonary vascular pattern suggests slight pul-

monary hypertension. **Clinical diagnosis.**—Mitral stenosis. Moderate pulmonary hypertension. ? Tricuspid stenosis. **Haemodynamic studies.**—Diastolic gradient, maximum in presystole, across tricuspid valve. Moderate pulmonary hypertension.

Conclusion.—Tricuspid stenosis, moderately severe. Mitral stenosis, severe. Tricuspid disease suspected clinically because of tricuspid murmur and "a" wave and right atrial size out of proportion to signs of pulmonary hypertension.

Treatment.—Mitral valvotomy (severe mitral stenosis; valve less than 1 cm. diameter). Considerable improvement in symptoms maintained until present time. Signs of mitral stenosis have virtually disappeared.

Case C 2 : Tricuspid Stenosis Associated with Severe Mitral Stenosis and Incompetence; Moderate Pulmonary Arterial Hypertension

Woman aged 51. Progressive dyspnoea on exertion for 20 years. Paroxysmal nocturnal dyspnoea. Ankle-swelling. Pulmonary infarction. **Physical signs.**—Mitral facies. Arterial pulse small and collapsing; regular. B.P. 130/70. Cardiac impulse; right and left ventricular enlargement. Jugular venous pulse: giant "a" wave. Variable systolic wave. Tricuspid presystolic and systolic murmurs. P2 accentuated, normal split. Mitral presystolic and systolic murmurs. Faint aortic diastolic murmur. Poor mitral first sound; no opening snap. Fourth heart sound. Liver enlarged and pulsating in presystole. Lungs: crepitations. **Cardiography.**—Combined ventricular hypertrophy. Long P-R interval; right atrial P waves. **Radiology.**—Right atrium considerably enlarged. Left atrium moderately enlarged. Right ventricle moderately enlarged. Heavy calcification of mitral valve. Pulmonary vascular pattern suggests moderate pulmonary hypertension. **Clinical diagnosis.**—Mitral stenosis and incompetence. Slight aortic incompetence. ? Tricuspid stenosis. **Haemodynamic studies.**—Presystolic gradient across tricuspid valve. No gradient in early diastole. Moderate pulmonary hypertension (Fig. 10).

Conclusion.—Severe mitral stenosis and incompetence. Moderate tricuspid stenosis and incompetence. Aortic incompetence. Tricuspid stenosis suspected clinically, but signs complicated by other valve lesions.

Treatment.—Mitral valvotomy not performed owing to rigid, incompetent, and calcified valve. Tricuspid valvotomy not performed because of incompetence and presence of severe inoperable mitral lesion.

Necropsy.—Severe tricuspid stenosis (circumference 60 mm.) (Fig. 11). Mitral valve stenosed, distorted, grossly calcified, and probably incompetent.

Case A 10 : Tricuspid Stenosis Associated with Severe Mitral Stenosis, Pulmonary Hypertension, Atrial Fibrillation, Pulmonary Artery Thrombosis, and Chronic Congestive Heart Failure

Man aged 43. Increasing dyspnoea on exertion for two years (grade 2-3). Congestive heart failure six months. **Physical signs.**—Arterial pulse normal; regular. B.P. 150/110. Cardiac impulse, marked right ventricular hypertrophy. P2 accentuated; narrow split. Jugular venous pulse; large "a" wave and systolic wave (subsequently atrial fibrillation and systolic wave only). Tricuspid presystolic and systolic murmurs. Faint mitral diastolic murmur. Mitral systolic murmur. Mitral first sound only slightly accentuated. Opening snap poor. Early diastolic murmur at left sternal edge (Fig. 1). **Cardiography.**—Atrial fibrillation. Right ventricular hypertrophy (grade 1). **Radiology.**—Right atrium grossly enlarged. Right ventricle enlarged. Heavy calcification of mitral valve (Fig. 4). **Clinical diagnosis.**—Mitral stenosis and incompetence. ? Tricuspid stenosis. Pulmonary hypertension. **Haemodynamic studies.**—Systolic wave in right atrial pulse. Right ventricle not entered with catheter. **Conclusion.**—Tricuspid stenosis and incompetence. Mitral stenosis and incompetence. Pulmonary hypertension. Tricuspid disease suspected because of tricuspid murmurs and very large right atrium.

Treatment and Progress.—Atrial fibrillation occurred while awaiting admission to hospital. Repeated pulmonary thromboembolism treated with anticoagulants. Mitral valvotomy not advised in view of heavy calcification of mitral valve and mitral incompetence.

Necropsy.—Severe mitral stenosis and incompetence. Moderate tricuspid stenosis (circumference 110 mm.) and incompetence. Thrombosis of pulmonary artery to right lower lobe.

Case A 9: Tricuspid Stenosis Associated with Severe Aortic Stenosis, Critical Mitral Stenosis, Atrial Fibrillation, and Slight Pulmonary Arterial Hypertension

Woman aged 42. Dyspnoea on exertion and paroxysmal nocturnal dyspnoea for many years. **Physical signs.**—Cyanosed. Arterial pulse small. Atrial fibrillation. B.P. 120/90. Jugular venous pulse: initially 2–3 cm. systolic wave—subsequently normal. Signs of severe aortic stenosis,

mitral stenosis, and ? incompetence.

No tricuspid murmurs. **Cardiography.**—Left ventricular dominance. Atrial fibrillation.

Radio-logy.—Great enlargement of right atrium (80 mm.); enlargement of both ventricles and left atrium. No evidence of pulmonary hypertension (Fig. 14).

Clinical diagnosis.—Aortic stenosis; mitral stenosis and ? incompetence. Tricuspid stenosis suspected because of size of right atrium in absence

FIG. 14.—Six-foot (1.8-metre) postero-anterior radiograph of Case A 9. Right atrium grossly enlarged (80 mm.) and strikingly at variance with the normal pulmonary arterial pattern and minimal pulmonary hypertension.

of signs of pulmonary hypertension. **Haemodynamic studies.**—Small gradient across tricuspid valve in diastole (5 mm.). Normal right ventricular pressure.

Conclusion.—Severe aortic stenosis. Mitral stenosis and incompetence. Mild tricuspid stenosis. Signs of tricuspid stenosis totally obscured by atrial fibrillation and other valve lesions. Diagnosis suspected on radiological grounds alone.

Treatment.—Aortic and mitral valvotomy. Death from cerebral embolism immediately after operation.

Necropsy.—Slight tricuspid stenosis (circumference 100 mm.) (Fig. 13). Severe aortic and mitral stenosis.

Discussion and Conclusions

The incidence of appreciable tricuspid valve disease found at necropsy in this series approximates closely to that described by Smith and Levine (1942), who found an incidence of 9.4% in a series of 340 cases of rheumatic heart disease coming to necropsy, and of Cooke and White (1941), who noted 14% in 217 cases of rheumatic heart disease. Associated valve disease was present in all the cases, and was severe in most. Mitral valve disease was present in all, and aortic disease in 75%.

Diagnosis

In one of our cases (C 1) mitral valve disease was clinically insignificant, and in this type of case the presence of a large presystolic wave in the jugular venous pulse, a tricuspid valve murmur, and an enlargement of the right atrium, without evidence of right ventricular hypertrophy, makes clinical diagnosis easy. Usually, however, the signs of tricuspid valve disease are obscured by those of associated

mitral valve disease. Tricuspid murmurs may become louder during inspiration (Gibson and Wood, 1955; Yu *et al.*, 1956; Hollman, 1956), and this sometimes enables them to be distinguished from mitral murmurs which do not. Even in cases where this distinction cannot be made with certainty, tricuspid disease can nevertheless be strongly suspected if the degree of right atrial enlargement is disproportionate to the degree of pulmonary hypertension and right ventricular hypertrophy. The present study also suggests that when the right atrium is considerably enlarged (over 70 mm. from the midline) in cases of rheumatic heart disease, the diagnosis of tricuspid involvement can be made with confidence, particularly if the P–R interval exceeds 0.20 sec. in the absence of active carditis.

The importance of prolongation of the P–R interval in the diagnosis of tricuspid disease has not been mentioned in previous studies (Ferrer *et al.*, 1953; Whitaker, 1955; Gibson and Wood, 1955; Yu *et al.*, 1956; Hollman, 1956; Pantridge and Marshall, 1957), although Trace *et al.* (1954) described it in their own case. Dalla Volta and Forattini (1957) described three cases of tricuspid stenosis in which the P–R interval appeared to exceed 0.20 sec. in the published cardiograms, but the timing of the cardiograph paper was not mentioned.

The most difficult differential diagnosis is from functional tricuspid incompetence, which may produce identical signs. Such incompetence is usually due to pulmonary hypertension from mitral stenosis, and differentiation depends on the proportion between the hypertension and right atrial size, as already mentioned. When, however, mitral incompetence is present there may be a raised venous pressure without much pulmonary hypertension (Gibson and Wood, 1955). However, prolongation of the P–R interval does not seem to occur in these cases, and, if present, may suggest the correct diagnosis. When mitral incompetence is severe and the left atrium aneurysmal, atrial fibrillation is always present and tricuspid incompetence frequently so. Under these circumstances the differentiation becomes clinically impossible. Final proof depends on the demonstration of a considerable pressure gradient across the tricuspid valve, for a slight gradient may be present in functional incompetence (Yu *et al.*, 1956). This may be due to a very high stroke volume.

Effect of Tricuspid Disease on Symptoms of Rheumatic Heart Disease

It has been suggested that tricuspid stenosis modifies the symptoms of pulmonary congestion due to left-sided valvular lesions (Thompson and Levine, 1937), but the findings in our series do not support this; for all patients were dyspnoeic, and pulmonary oedema or paroxysmal dyspnoea occurred in nearly one-third of them. Orthopnoea and paroxysmal dyspnoea occurred in three of Gibson and Wood's 14 rheumatic cases, and orthopnoea in 7 of the 13 cases of Reale *et al.* (1956). The most severe case of tricuspid stenosis in our series (Case C 2) had marked dyspnoea on effort and repeated attacks of paroxysmal dyspnoea and orthopnoea, attributable to gross mitral disease. Thus even gross tricuspid disease will not modify the symptoms of mitral disease if the latter is severe enough, but may do so if the mitral disease is slighter.

Haemodynamic Data

Our haemodynamic studies are similar to those published by Ferrer *et al.* (1953), Gibson and Wood (1955), Whitaker (1955), Hollman (1956), Yu *et al.* (1956), and Pantridge and Marshall (1957), and demonstrate the gradient in diastole across the tricuspid valve. In the cases with sinus rhythm, the atrial systolic pressure is not transmitted to the ventricle, and the height of the "a" wave in the atrial pulse as compared with the right ventricular systolic pressure is striking. The slow "y" descent is also noteworthy. It is of interest that patient C 10, who had a large "a" wave in the jugular venous pulse when in sinus rhythm, developed gross tricuspid insufficiency at the onset of atrial fibrillation

(Fig. 1), and at necropsy his tricuspid valve was thought to be incompetent as well as stenosed. A similar case, but without proof at necropsy, is reported by Ferrer *et al.* (1953).

Association of Incompetence with Stenosis

The development of signs of tricuspid insufficiency in all patients with tricuspid stenosis who developed atrial fibrillation led us to suppose that the organically stenosed and obstructed valve is probably always incompetent to some degree. The findings of Ferrer *et al.* (1953), McCord *et al.* (1954), Whitaker (1955), and Hollman (1956, 1957), in general support this contention. However, Reale *et al.* (1956) doubt whether a marked systolic wave in the right atrial pulse necessarily indicates tricuspid incompetence, and they suggest that if the atrium is filled and stretched at the time of ventricular systole the regurgitation of even a very small amount of blood might produce a considerable systolic wave. Ten of their 13 cases came to operation, and "significant" regurgitation was detected in only one. A low cardiac output at the time of palpation of the valve, however, may obscure a regurgitant jet. Pantridge and Marshall (1957) reported incompetence in one of two cases of tricuspid stenosis at operation for tricuspid valvotomy.

However, the work of Müller and Shillingford (1954) and Korner and Shillingford (1957) suggests that a large positive systolic wave is in fact associated with a considerable volume of reflux estimated by the dye-dilution technique. Such a wave was regularly seen in our patients with atrial fibrillation, and at necropsy it seemed probable that the valves had been incompetent. The absence of this wave in sinus rhythm may have been partly due to better emptying of the atrium, which is then able to accept a regurgitant flow without conspicuous rise of pressure.

We believe, therefore, that incompetence is a very common accompaniment of stenosis, but is not necessarily invariable. This point has an important bearing on the surgical treatment of tricuspid stenosis, since valvotomy is unlikely to be wholly successful if appreciable incompetence is present, for it may be increased by valvotomy (Hollman, 1956), to the detriment of the patient. At the present time, tricuspid valvotomy is a well-recognized though uncommon operation (Brofman, 1953; Chesterman and Whitaker, 1954; Krook *et al.*, 1955; O'Neill *et al.*, 1954; Trace *et al.*, 1954; Bailey, 1955; Swan *et al.*, 1955; Gibson and Wood, 1955; Hollman, 1956; Reale *et al.*, 1956; Yu *et al.*, 1956; Pantridge and Marshall, 1957; Dalla Volta and Forattini, 1957). But, as Hollman (1956) points out, the risk of increasing incompetence is appreciable. We have no personal experience of tricuspid valvotomy, but we believe that the general applicability of the operation is small, and that the difficulties of selecting the suitable case are considerable. The operation should probably be reserved for patients whose symptoms are urgent and unrelieved by medical means.

Summary

Twenty-one proved cases of organic disease of the tricuspid valve have been presented. The diagnosis is discussed, with special reference to the modification in the clinical picture produced by other valve lesions. Clinical, haemodynamic, and pathological data are discussed. Special emphasis is laid upon enlargement of the right atrium, and a method of radiological measurement is presented. The importance of right atrial P waves in the absence of evidence of right ventricular hypertrophy on the cardiogram is stressed. Prolongation of the P-R interval is underlined as a valuable diagnostic pointer.

Reasons are given for concluding that tricuspid incompetence usually accompanies tricuspid stenosis, and the surgical implications of this are mentioned.

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EFFECT OF TEMPERATURE ON SURVIVAL OF BACTERIA IN BLOOD FOR TRANSFUSION

WITH A NOTE ON CONTAMINATION BY COLD-GROWING ORGANISMS

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From 1946 to 1949 the practice in the North London Blood Transfusion Centre was to transport blood collected at the donor sessions to the centre in vans without refrigeration. Thus, at the most outlying sessions, the first donor's blood was unrefrigerated for about eight hours before reaching the cold room at the centre; approximately 210,000 bottles of blood were so treated. In 1949 transport by refrigerator vehicles was considered.

When fresh blood is kept for four days at room temperature the red cells do not survive well on subsequent storage (Gibson *et al.*, 1947), but if it is allowed to cool slowly for a few hours before refrigeration it is unlikely to suffer, and, since the phagocytic and bacteriolytic processes in the blood are most active at body temperature, such treatment might even have definite advantages (James, 1954).

It was decided that a complete change to refrigerator transport should be delayed until evidence of its value could be obtained. In the meantime two refrigerator vans were employed in conjunction with five non-refrigerators. In fact, owing to the inconvenient size of the new vans when negotiating the narrow streets of London and some provincial towns, the approximate ratio in which they were used was 2:11.

During the period when both types of van were in use about 700,000 bottles of blood were collected and one death from infected blood was recorded, this bottle having been carried in a non-refrigerator van, but the