Rheumatic tricuspid stenosis

A haemodynamic correlation

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Studies were made on 21 cases with haemodynamically significant tricuspid stenosis, including 12 in sinus rhythm and 9 with atrial fibrillation. A remarkable degree of correlation was found between the haemodynamic changes and the physical signs, particularly auscultation and left parasternal pulsations represented by the right ventricular apex cardiogram. Inspiratory augmentation of the tricuspid diastolic murmur was always evident, while tricuspid systolic murmur either decreased appreciably or showed little change. This sign was considered essential for the diagnosis of dominant stenosis, especially in the presence of atrial fibrillation – where a systolic murmur is nearly always associated – and it reflected a characteristic haemodynamic response to inspiration showing a concomitant increase of right atrial/right ventricular diastolic gradient and decrease of right ventricular/right atrial systolic gradient. A tricuspid opening snap was recorded in half the cases. This was mainly facilitated by the simultaneous recording of both right and left apex cardiograms separating a frequently earlier tricuspid snap from a mitral snap. The II-OS (T) interval showed considerable variation with the heart rate and cycle length. The right ventricular apex cardiogram was introduced as a valuable aid in the diagnosis of dominant stenosis, especially in the presence of atrial fibrillation where it showed absence of a rapid filling wave. In patients with sinus rhythm, the right ventricular apex cardiogram showed a prominent a wave which represented direct right atrial tracing. This wave correlated well with the degree of stenosis and with the height of P wave in V₂. Early diagnosis and interference were stressed in cases with atrial fibrillation where the diagnosis is often missed. These cases were associated with a higher degree of venous hypertension leading to retention of salt and water and chronic congestion of the viscera, which can adversely affect the results of operation.

Tricuspid stenosis is now recognized as a not uncommon sequela of rheumatic affection of the heart. However, the surprisingly high incidence both in pathological reports (30% in seven series collected by Kitchin and Turner, 1964) and in some surgical reports (16% in a report by Bailey and Bolton, 1956) contrasts with the modest incidence in most clinical reports. This can only be partially explained by the relatively lax criteria utilized in necropsy studies (probably including haemodynamically insignificant lesions) on one hand, and the large element of selectivity in some surgical reports on the other hand. The discrepancy can be more reasonably attributed to the deficiency of a high initial clinical index of suspicion, the established difficulty of the diagnosis in the presence of atrial fibrillation (Sanders *et al.*, 1966), and the inadequate appreciation of the whole spectrum of organic tricuspid valve disease from pure stenosis to

dominant insufficiency and from mild to severe lesions.

During our work on rheumatic tricuspid stenosis we were impressed by the remarkable degree of correlation between the haemodynamic characteristics of the disease and the physical signs. This correlation forms the main purpose of the present report which is an endeavour to cover, to some extent, the gap between the actual incidence of the disease and its clinical recognition by both better use of traditional physical signs, e.g. auscultation, and the search for new ones, e.g. the study of the left parasternal pulsations as represented by the right ventricular apex cardiogram which is introduced as a valuable aid in diagnosis.

Materials and methods

Twenty-one patients with haemodynamically significant tricuspid stenosis were studied in the Cardiac Department, Kasr El-Aini Faculty of

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TABLE 1 *Clinical and electrocardiographic data*

Case No.	Age (yr.)	Sex	Tricuspid stenosis		Mitral stenosis		Aortic valve disease		Auscultation of tricuspid area				Electrocardiogram			
			Pure	With TI	Pure	With MI	AS	AI	PSM	MDM	SM	TOS	Vent. hyper.	Height of P LII (mm.)	Height of P V ₂ (mm.)	PR interval (1/100 sec.)
1	18	F	+	—	+	—	—	Mild	+	—	—	—	—	3	3.5	18
2	24	F	+	—	+	—	—	—	+	+	—	+	—	4	4.5	16
3	25	F	+	—	+	—	—	—	+	+	—	+	—	5	7	19
4	30	F	+	—	+	—	—	Moderate	+	—	—	—	—	2.5	3	20
5	23	M	—	—	+	—	—	Moderate	+	—	—	—	LVH	3	3	18
6	25	M	—	Moderate	+	—	—	—	+	+	+	—	RVH	4	4.5	19
7	22	F	+	—	+	—	—	Mild	+	—	—	—	—	3	3.5	17
8	26	F	+	—	+	—	—	—	+	—	—	—	—	3.5	3.5	19
9	19	F	+	—	+	—	—	—	+	+	—	+	—	4	5	21
10	20	M	+	—	+	—	—	—	+	+	—	—	—	4	4.5	19
11	26	F	—	Mild	+	—	—	—	+	—	+	+	RVH	3.5	2.5	22
12	20	F	—	Mild	+	—	—	—	+	—	+	+	—	2.5	3	18
13	38	F	—	Mild	+	—	—	—	—	+	+	+	Qr (V ₁)	AF		
14	22	F	—	Moderate	—	Moderate	—	Mild	—	+	+	+	—	AF		
15	35	M	—	Moderate	+	—	—	—	—	+	+	—	RVH	AF		
16	19	F	—	Mild	—	Mild	—	—	—	+	+	—	RVH	AF		
17	35	F	—	Mild	+	—	—	—	—	+	+	+	—	AF		
18	30	F	—	Mild	—	Mild	Mild	Moderate	—	+	+	—	Combined	AF		
19	24	F	—	Moderate	+	—	—	—	—	+	+	+	—	AF		
20	29	F	—	Moderate	—	Mild	Mild	Mild	—	+	+	—	Qr (V ₁)	AF		
21	34	F	—	Moderate	+	—	—	—	—	+	+	+	—	AF		

Abbreviations: TI, tricuspid insufficiency; PSM, presystolic murmur; LVH, left ventricular hypertrophy; MI, mitral insufficiency; MDM, mid-diastolic murmur; RVH, right ventricular hypertrophy; AS, aortic stenosis; SM, systolic murmur; AI, aortic insufficiency; TOS, tricuspid opening snap.

Medicine, between January 1963 and December 1968. Of these, 12 patients were in sinus rhythm and 9 had atrial fibrillation. The diagnosis was confirmed in 13 by surgical exploration of the tricuspid valve during mitral valvotomy. In 9 of these patients tricuspid valvotomy was also performed. In the remaining 4 patients significant tricuspid insufficiency was found, and these were considered to be in need of valve replacement which was not available at the time of the study. Four patients came to necropsy, one died after operation for mitral valvotomy. In the remaining 4, there was either significant aortic incompetence and no operation was attempted (2) or the operation was refused (2). These 4 patients were all in sinus rhythm and showed significant tricuspid valve gradient. Thus all patients with atrial fibrillation had either surgical or postmortem proof.

All patients had a complete cardiological examination, with particular attention to the jugular venous pulsations, praecordial pulsations, and auscultation. Investigations included a 12-lead conventional electrocardiogram and chest radiograph. Phonocardiograms were recorded on an Elema-Schönander Mingograph type 42 B at paper speed of 50 mm. per second. Indirect jugular venous pressure pulses were recorded with a pulse wave amplifier. Right ventricular apex cardiograms were recorded at the left sternal border in the third to fifth intercostal spaces by a pick-up bell either secured in place by a rubber strap or supported by the hand. Left ventricular apex cardiograms were usually recorded with the patient

in the left lateral position. Simultaneous recordings of both the right ventricular apex cardiogram and left ventricular apex cardiogram were obtained in 19 patients. The recordings were usually obtained in mid-expiratory position. Right heart catheterization was performed in the conventional manner. Simultaneous recording of right atrial and right ventricular pressure pulses was performed in 15 patients. The pressures were obtained through two equisensitive Elema-Schönander transducer-electromanometer sets, and recorded on a 4-channel Mingograph type 42 B. The tracings were inscribed from the same baseline. In every case, however, a pull-back record across the tricuspid valve was also obtained while the patient was holding his breath at deep inspiration. The effect of inspiration was observed in every patient, and the effects of exercise and amyl nitrite inhalation were recorded in 11 and 9 patients, respectively. The cardiac output was measured by the direct Fick principle. Mitral and tricuspid valve areas and pulmonary arteriolar resistance were calculated according to Gorlin formulae (Gorlin and Gorlin, 1951; Gorlin *et al.*, 1951).

Results

Pertinent data in 21 cases with tricuspid stenosis are included in Tables 1 and 2.

Clinical history Apart from the paucity of symptoms of acute pulmonary congestion,

TABLE 2 Haemodynamic data

Case No.	PCVP or LA (mm. Hg)	PA pressure (mm. Hg)	Cardiac index (l./min./m. ² BSA)	PAR (dynes. sec. cm. ⁻⁵)	RA pressure (mean) (mm. Hg)	Tricuspid gradient (mm. Hg)			AZ interval (1/100 sec.)	Calculated tricuspid valve area (cm. ²)	Calculated mitral valve area (cm. ²)	Operation	
						Exp.	Insp.	Average				Mitral valvotomy	Tricuspid valvotomy
1	8	20/11 (16)*	1.9	195	5	3	6	5	7	2.0	—	+	+
2	14	35/15 (22)	1.7	224	8	7	13	10	12	1.5	1.6	+	+
	9†	27/12 (18)†	2.65†	164†	2†	0†	3†	1.5†	—	—	—	+	+
3	8	21/11 (15)	1.5	255	11	8	14	11	12	0.6	—	+	+
4	15 LA	22/15 (19)	2.6	76	7	5	14	9	8	1.6	1.4	Not attempted	+
5	9	21/12 (16)	2.2	126	6	4	12	8	8	1.7	—	Not attempted	+
6	20 LA	40/20 (32)	1.8	305	8	6	12	9	10	1.2	0.7	+	Explored
7	14	30/12 (19)	2.1	137	6	5	9	7	8	1.5	1.6	Refused	+
8	14 LA	40/21 (28)	2.3	301	7	5	11	8	9	1.6	1.5	+	+
9	20	35/21 (28)	1.6	237	10	9	15	12	11	0.8	0.6	+	+
	11†	24/11 (18)†	2.7†	122†	3†	1†	3†	2†	—	—	—	+	+
10	23	41/28 (32)	1.8	235	8	7	13	10	10	0.8	0.5	+	+
	12†	22/12 (17)†	2.4†	97†	3†	1.5†	4†	3†	—	—	—	+	+
11	22	50/28 (35)	2.7	275	6	5	12	9	8	1.7	0.8	+	+
12	20	45/22 (31)	2.4	215	6	4	10	7	7	1.6	0.8	Refused	+
13	35 LA	50/36 (45)	1.6	290	16	12	18	15	—	0.6	0.4	+	+
	15†	30/16 (22)†	2.2†	148†	5†	2†	5†	4†	—	—	—	+	+
14	15	26/15 (19)	2.4	87	9	6	13	9	—	1.5	1.4	Died before operation	+
15	26	60/40 (52)	2.3	554	23	15	25	20	—	1.1	1.1	+	Explored
16	29	48/35 (41)	1.9	375	18	12	20	16	—	0.8	0.5	Died (after mitral valvotomy)	+
17	22	40/26 (33)	2.1	248	10	8	16	12	—	1.3	0.9	+	+
18	20	42/20 (29)	1.8	235	11	8	17	13	—	1.4	1.2	Died before operation	+
19	17	31/18 (24)	2.2	158	9	6	13	10	—	1.2	1.0	+	Explored
20	34 LA	48/35 (44)	1.6	290	13	8	15	12	—	0.7	0.4	Died before operation	+
21	23	38/26 (33)	2.4	190	8	6	14	10	—	1.2	0.8	+	Explored

Abbreviations: PCVP, pulmonary capillary venous pressure; RA, right atrium; LA, left atrium; Exp., expiratory; PA, pulmonary artery; Insp., inspiratory; PAR, pulmonary arteriolar resistance.

* Mean pressure is included between parentheses.

† Postoperative haemodynamic study.

there was no striking difference in symptomatology between these patients and patients with pure mitral stenosis, especially if individual cases are considered. Oedema of the lower limbs was not reported in the 12 patients in sinus rhythm (apart from slight oedema in last months of pregnancy). On the other hand, a history of recurrent oedema and treatment for congestive failure was obtained in 8 out of 9 patients with atrial fibrillation.

Jugular venous pulse Flickering presystolic pulsations (A wave) were observed in every case with sinus rhythm. Though the degree of prominence of the A wave varied from one patient to another, yet in all cases it could be detected while the patient was sitting. The jugular phlebogram showed a tall spiky A wave and a diminutive V wave with imperceptible Y descent. On the other hand, patients with atrial fibrillation showed a positive CV systolic wave with a relatively slow Y descent.

Right ventricular apex cardiogram

The right ventricular apex cardiogram could

be always recorded even in those cases showing minimal pulsations at the left sternal border. However, usually a careful search for a suitable point for recording and sufficient amplification are necessary. In the cases with sinus rhythm the right ventricular apex cardiogram was bifid due to the presence of a prominent presystolic a wave (Fig. 1 A, B, C and Fig. 2 A). It was sometimes difficult to distinguish the beginning of the a wave which appeared as a continuation of the slow filling wave of the previous cycle. The slow filling wave was the only wave recorded due to the absence of a rapid filling wave. The a wave percentage amplitude was calculated according to the formula:

$$\frac{\text{'a' wave amplitude}}{\text{OE amplitude}} \times 100$$

where O and E are points of the apex cardiogram (Ginn *et al.*, 1967) and varied from 16 to 86 per cent (Table 3). It showed a fair degree of correlation with the calculated tricuspid valve area (Fig. 3).

On the other hand, in cases with atrial fibrillation the a wave was absent, which

FIG. 1 Records (A) and (B) represent the right ventricular apex cardiogram (RV ACG) of the same patient (Case 10) before and after tricuspid valvotomy. Note the decrease in size of the *a* wave after operation. The post-operative phonocardiogram still shows a short pre-systolic murmur (PSM) and a tricuspid opening snap (TOS) that coincides with the *O* point of the RV ACG. (C) The RV ACG of Case 3 with severe tricuspid stenosis. It shows the *a* wave nearly as prominent as the systolic component of the right ventricle. (D) The RV ACG in a case of mitral stenosis with pulmonary hypertension and functional tricuspid insufficiency. Note the small *a* wave compared with cases of tricuspid stenosis.

FIG. 2 Right and left ventricular apex cardiograms (RV ACG and LV ACG) of the same patient with combined mitral and tricuspid stenosis taken on two different occasions. Note the prominent *a* wave in the RV ACG and the absent *a* wave in the LV ACG. Note also the earlier *O* point of the RV ACG (OT) coinciding with an earlier tricuspid opening snap (TOS) and the later *O* point of the LV ACG (OM) coinciding with a later mitral snap (MOS).

TABLE 3 Right ventricular apex cardiogram in tricuspid valve disease

Tricuspid stenosis (sinus rhythm)		Tricuspid insufficiency (sinus rhythm)		Tricuspid stenosis (atrial fibrillation)				Tricuspid insufficiency (atrial fibrillation)			
Case No.	<i>a</i> WPA %	Case No.	<i>a</i> WPA %	Case No.	<i>S</i> angle (degrees)	<i>T</i> angle (degrees)	<i>T</i> / <i>S</i> ratio	Case No.	<i>R</i> angle (degrees)	<i>T</i> angle (degrees)	<i>T</i> / <i>R</i> ratio
1	17	1	7	1	37	48	1.30	1	70	64	0.91
2	37	2	15	2	38	62	1.63	2	75	69	0.92
3	86	3	17	3	37	46	1.24	3	78	65	0.86
4	24	4	21	4	40	55	1.38	4	54	49	0.91
5	22	5	9	5	32	54	1.67	5	65	55	0.85
6	39	6	14	6	28	57	2.00	6	63	59	0.94
7	25	7	28	7	41	64	1.56	7	74	67	0.91
8	21	8	10	8	31	49	1.58	8	71	54	0.76
9	58	9	9	9	35	59	1.69	9	68	42	0.62
10	32	10	26					10	52	44	0.85
11	16			Average	35.4		1.51				
12	19	Average	15.6	SD	± 1.7		± 0.22	Average	67		0.85
		SD	± 6.9					SD	± 8.1		± 0.09
Average	32.2										
SD	± 19.7										

Abbreviations: *a* WPA = (*a*) wave percentage amplitude; *R* = rapid filling angle; *S* = slow filling angle; *T* = total angle.

FIG. 3 The figure shows the fair degree of correlation between the calculated tricuspid valve area and the a wave percentage amplitude of the right ventricular apex cardiogram and the height of the P wave in V₂.

made the analysis of the diastolic component of the right ventricular apex cardiogram easier. No case showed convincing evidence of a rapid filling wave; only a slow filling wave was recorded (Fig. 4, Fig. 5 A, and Fig. 6). The S and T angles and the T/S ratio were

calculated according to the procedure of Benchimol *et al.* (1960) (Table 3).

The right ventricular apex cardiogram in 20 patients with mitral stenosis and functional tricuspid insufficiency was studied and compared with that in the present series (Table 3). These patients included 10 patients with sinus rhythm and 10 patients with atrial fibrillation. In patients with sinus rhythm signs of severe pulmonary hypertension were usually present. As a group, the wave was less prominent than in patients with tricuspid stenosis, and a rapid filling wave was usually seen (Fig. 1 D). The 'a' wave percentage amplitude ranged from 7 to 28 per cent (average 15.6%). Patients with atrial fibrillation always showed a definite rapid filling wave (Fig. 5B), sometimes ending with a small positive wave coinciding with a loud third heart sound. The R and T angles and the T/R ratio in these cases are shown in Table 3.

From the simultaneous records of both right and left apex cardiograms (Fig. 7) the O point of the right ventricular apex cardiogram was analysed and the interval between the first high-pitched components of the second sound and the O point was calculated

FIG. 4 (A) Right ventricular apex cardiogram (RV ACG) in a case of tricuspid stenosis with atrial fibrillation, showing a slow filling wave (SFW). The O point coincides with a tricuspid opening snap (TOS). (B) Simultaneous RV ACG and right ventricular pressure pulse (RV) showing smooth rise of the RV diastolic pressure reflected in the SFW of the RV ACG. (C) Post-operative RV ACG of the same patient showing a rapid filling wave (RFW). S₁, first heart sound; S₂, second heart sound.

FIG. 5 (A) Right ventricular apex cardiogram (RV ACG) in a case of tricuspid stenosis with atrial fibrillation showing a slow filling wave (SFW). The jugular phlebogram reveals a slow Y descent. (B) RV ACG in a case of tricuspid insufficiency with atrial fibrillation showing a rapid filling wave (RFW) ending with a small positive wave. The jugular phlebogram reveals a rapid Y descent. S₁, first heart sound; S₂, second heart sound; SM, systolic murmur; MDM, mid-diastolic murmur.

FIG. 6 Right ventricular apex cardiogram in a case of tricuspid stenosis with atrial fibrillation, showing slow filling waves and variable intervals between the second heart sound (S₂) and both the O point of the RV ACG and the tricuspid opening snap (TOS). The intervals are prolonged following long cardiac cycles. S₁, first heart sound; SM, systolic murmur; MDM, mid-diastolic murmur.

FIG. 7 Simultaneous recording of both right and left apex cardiograms (RV ACG and LV ACG, respectively), together with the phonocardiogram at the tricuspid area. The O point of the RV ACG, which coincides with a tricuspid opening snap (TOS) precedes the O point of the LV ACG by 0.04 sec. S_1 , first heart sound; S_2 , second heart sound; SM, systolic murmur; MDM, mid-diastolic murmur.

FIG. 8 Phonocardiograms in two cases of tricuspid stenosis with sinus rhythm showing the crescendo decrescendo presystolic murmur (PSM). Note in (B) that inspiratory augmentation of the PSM did not change its crescendo decrescendo character. S_1 , first heart sound; S_2 , second heart sound; TOS, tricuspid opening snap.

(II - OT interval) and compared with the interval between the second sound and the O point of the left ventricular apex cardiogram (II - OM interval). This interval was shorter by 0.01 to 0.06 sec. in 16 patients, equal in 2, and only longer in one case. The II-OT interval ranged widely from 0.03 to 0.13 sec., and showed considerable variation with the heart rate and the length of the preceding cycle especially in atrial fibrillation (Fig. 6).

Auscultatory findings Patients with sinus rhythm always showed a tricuspid presystolic murmur, and in 5 patients a mid-diastolic component was also present (Fig. 8). All 9 patients with atrial fibrillation had a tricuspid mid-diastolic murmur that began early in mid-diastole and occasionally continued to the next cycle (Fig. 9 and 10). A clear tricuspid diastolic thrill was felt in 7 cases (4 with sinus rhythm). The tricuspid diastolic murmur was usually located at the left sternal border in

FIG. 9 Phonocardiogram in a case of tricuspid stenosis with atrial fibrillation showing the effect of inspiration leading to an appreciable decrease in the intensity of the systolic murmur (SM), while the mid-diastolic murmur (MDM) showed obvious augmentation; TOS, tricuspid opening snap.

FIG. 10 Phonocardiograms in two cases of tricuspid stenosis with atrial fibrillation showing the effect of inspiration resulting in obvious augmentation of the mid-diastolic murmur (MDM) with slight decrease in the intensity of the systolic murmur (SM).

the fourth intercostal space, but occasionally in the third or fifth spaces. A tricuspid systolic murmur was present in all cases with atrial fibrillation and in 3 cases with sinus rhythm. Characteristic changes in both the diastolic and systolic murmurs were observed with inspiration, usually in the first one or few cycles at the beginning of inspiration. In all cases the diastolic murmur increased conspicuously in intensity, while the systolic murmur either decreased appreciably (Fig. 9), or showed little change (Fig. 10). Only in one case was there a convincing inspiratory augmentation of the tricuspid systolic murmur.

An opening snap of tricuspid origin was recorded in 10 cases in the present series. This sound was convincingly appreciated by auscultation in a smaller number of cases, since it was usually difficult to separate from a mitral opening snap. Identification was based on analysis of records of simultaneous right and left apex cardiograms with the phonocardiogram at the tricuspid area (Fig. 7) where a sound coinciding with the O point of the right ventricular apex cardiogram was recorded. This sound frequently occurred

earlier than the mitral opening snap which could be separately shown to coincide with the O point of the left ventricular apex cardiogram (9 cases). In one case no difference in timing could be seen, but the medial snap showed appreciable inspiratory augmentation and was considered of tricuspid origin. In 2 patients, both snaps could be recorded from the same area (Fig. 2).

Electrocardiographic findings Electrocardiographic evidence of right ventricular hypertrophy was present in 4 patients. Evidence of either left ventricular hypertrophy or combined hypertrophy was present in one patient each. Two patients with atrial fibrillation showed a Qr pattern in V₁. Peaked P waves characteristic of right atrial hypertrophy were present in all patients with sinus rhythm and were best seen in leads II, V₁, and V₂. In fact in 9 patients (75%), the P waves were higher in V₂ than in V₁. The height of the P waves in V₂ correlated reasonably well with the calculated tricuspid valve area (Fig. 3 and 11). The PR interval ranged from 0.16 to 0.22 sec. (average 0.19 sec.).

FIG. 11 The electrocardiogram in three cases of tricuspid stenosis with sinus rhythm showing tall peaked P waves highest in leads II and V₂. Note the correlation between the height of the P wave in V₂ and the degree of tricuspid stenosis judged by the diastolic transvalvular gradient shown on the left.

We were not impressed by the presence of a prolonged PR interval in our cases.

Radiographic findings The postero-anterior view was usually analysed. It showed prominence of the lower right cardiac border occupied by the right atrium. There was no correlation between the degree of enlargement as judged by measurement of the maximum distance of the right heart border from the middle line and the calculated tricuspid valve area. Patients with severe tricuspid stenosis were seen with unimpressive right atrial enlargement. As a rule the enlargement was severe in cases with atrial fibrillation. Pulmonary artery dilatation was absent or minimal in 8, mild in 8, and moderate in 5 patients. Pulmonary venous hypertension as revealed by hilar congestion, middle fissure effusion, and transverse septal lines in the costophrenic angles was seen in 6 patients, all with tight tricuspid stenosis. These signs seem to be governed by a pre-existing and/or dominating mitral stenosis. Other positive findings in the x-rays were consistent with the associated valve lesions.

Haemodynamic findings Though simultaneous right atrial and right ventricular pressure pulses were recorded in 15 patients, yet

frequently excellent pull-back tracings could be obtained (Fig. 12), provided that the patient held his breath at deep inspiration and the catheter was withdrawn rapidly. The first records however had the advantage of study of the simultaneous changes in the right atrial and right ventricular pressure pulses by inspiration, exercise, and drugs.

Analysis of right atrial pressure pulse showed the same characteristics described in the jugular phlebogram (a prominent sometimes spiky A wave, a diminutive V wave with imperceptible Y descent in sinus rhythm, and a positive CV systolic wave, with relatively slow Y descent in atrial fibrillation). The mean diastolic gradient was always used for the calculation of the tricuspid valve area. However, in cases with sinus rhythm the maximum gradient occurred in presystole and corresponded to the giant A wave in the atrial pulse tracings. The interval from the summit of the A wave to the point of intersection of the right atrial and right ventricular pressure pulses (AZ interval) was measured and ranged from 0.07 to 0.12 sec. (average 0.085 sec.) (Table 1).

In cases with atrial fibrillation the gradient was maximum in early diastole. It varied little in short diastolic cycles, while in long cycles the diastolic pressure of the right ventricle

FIG. 12 *Excellent pull-back records in patients with tricuspid stenosis obtained while patient was holding his breath at full inspiration. (A) Tricuspid stenosis with sinus rhythm. (B) Tricuspid stenosis with atrial fibrillation. RV, right ventricular pressure pulse; RA, right atrial pressure pulse.*

FIG. 13 Simultaneous recording of right ventricular and right atrial pressure pulses in a case of tricuspid stenosis with atrial fibrillation, showing in (A) the effect of long diastolic cycles on the diastolic transvalvular gradient which may be nearly abolished; (B) effect of inspiration leading to considerable drop of both right ventricular (RV) systolic and diastolic pressures, with little effect on right atrial (RA) pressure resulting in a concomitant increase of the RA-RV diastolic gradient with decrease of the RV-RA systolic gradient.

gradually rose to approach the right atrial pressure so that the gradient might approach zero in long diastolic intervals in atrial fibrillation (or in the compensatory pause following a ventricular premature systole) (Fig. 13A).

The effect of inspiration was analysed for both the diastolic and systolic components of the simultaneous right atrial and right ventricular pressure pulses. The diastolic gradient always showed an obvious increase which was primarily due to the decrease in right ventricular diastolic pressure (sometimes to -19

FIG. 14 The effect of inspiration leading to slight drop of the right atrial pressure (A) and conspicuous drop of the right ventricular diastolic pressure (B). (C) The combined effect of inspiration on the simultaneous tracings of right ventricular and right atrial pressure pulses resulting in a conspicuous increase in the diastolic transvalvular gradient. The sinus tachycardia present is secondary to amyl nitrite inhalation given to augment the pressure changes.

mm. Hg, Fig. 14C), the right atrial pressure showing either no or minimal changes in either direction (Fig. 13B and Fig. 14A). The maximum diastolic gradient still kept its timing characteristics in sinus rhythm and atrial fibrillation. On the other hand, the systolic pressure of the right ventricle was found to drop appreciably in more than half the cases. This, together with the minimal changes in right atrial pressure, resulted in an obvious decrease in the systolic gradient between the right ventricle and right atrium (Fig. 13B). Exercise and amyl nitrite were found to augment the diastolic gradient, their changes being additive when they were applied together.

Post-operative findings. The post-operative course was usually less smooth in patients with atrial fibrillation. No significant traumatic tricuspid insufficiency was observed, but mild incompetence occurred in 3 cases. The physical signs of tricuspid stenosis usually regressed considerably after the operation. Jugular venous pulsations became less prominent and the diastolic murmur disappeared or became less intense. The right ventricular apex cardiogram showed decrease in the size of the a wave in cases with sinus rhythm (Fig. 1A and B) and the appearance of a rapid filling wave best seen in patients with atrial fibrillation (Fig. 4C). The tall peaked P waves were found to regress rapidly in the postoperative period (Fig. 15). Changes in the x-ray were less impressive. Postoperative haemodynamic studies were available in 4 patients and showed always an appreciable decrease in the transvalvular gradient (Table 2).

Discussion

There is a general impression that tricuspid stenosis occurs much more frequently than it is clinically diagnosed. This means in other words that refinement in the physical signs of the disease will still be needed. A specific symptomatology is not expected to offer much help in an individual case; therefore careful clinical examination will usually give the first clue to the diagnosis.

Though the jugular venous pulsations are of value in sinus rhythm, yet in the presence of atrial fibrillation analysis of the jugular venous pulse becomes more difficult. Classically a positive CV systolic wave is present with a slow Y descent. However, the rate of Y descent is not an easy sign to assess even in the jugular phlebogram which can be easily distorted. As a rule, cases with atrial fibrilla-

FIG. 15 The electrocardiogram in a case of tricuspid stenosis with sinus rhythm showing an appreciable decrease in the height of the P wave in the immediate postoperative period. (B) Electrocardiogram obtained one week after operation.

tion showed more congestion in the venous pulse which usually persisted unchanged despite active antifailure regimen. This point may be more valuable in suspecting organic tricuspid obstruction than the rate of Y descent and should stimulate further study. Meanwhile, we find it difficult to explain the report of Sanders *et al.* (1966) which describes minimal abnormality of the jugular venous pulse in half their cases of tricuspid stenosis with atrial fibrillation, while the lowest mean right atrial pressure in 9 out of 10 patients was 6 mm. Hg, which usually corresponds to a CV wave of 8–10 mm. Hg in height.

As a group, patients with atrial fibrillation showed a mean right atrial pressure nearly double the pressure in the group with sinus rhythm (average 13 and 7.5 mm. Hg in cases with atrial fibrillation and sinus rhythm, respectively), though the degree of tricuspid stenosis was comparable in both groups. This speaks of the importance of right atrial contraction, as reflected in the giant A wave, in maintaining the flow across the stenosed valve. Similar observations were previously reported (Killip and Lukas, 1957; Kitchin and Turner, 1964). The higher degree of chronic venous hypertension in cases with atrial fibrillation will explain the frequent occurrence of retention of salt and water and

ankle oedema in these patients (8 out of 9 cases in the present series). A similar mechanism was noted in patients with tricuspid insufficiency and a mean right atrial pressure greater than 10 mm. Hg (Sepulveda and Lukas, 1955). This chronic venous hypertension in patients with atrial fibrillation will adversely affect the function of the liver, and probably underlies the frequent postoperative complications seen in this group. This point represents in our opinion the single most important argument for early diagnosis and intervention for tricuspid stenosis in the presence of atrial fibrillation.

The purpose in studying the right ventricular apex cardiogram was initially the analysis of its diastolic component in a similar manner to the analysis of the left ventricular apex cardiogram in mitral valve disease (Benchimol *et al.*, 1960). Both systolic and diastolic components of the right ventricular apex cardiogram are similar to those described for the left ventricular apex cardiogram (Benchimol and Dimond, 1963; Tafur, Cohen, and Levine, 1964), reflecting identical haemodynamic events; however, minor differences in timing should be expected. As with the case in the left ventricular apex cardiogram, the rate of emptying of the right atrium and the rate of filling of the right ventricle should reflect the same haemodynamic events, and are in part related to the size of the orifice of the tricuspid valve. With a normal tricuspid valve opening, there is usually rapid emptying of the V wave of the right atrial pressure pulse and a small early diastolic dip followed by a smooth rise in the right ventricular diastolic pressure pulse, ending with a small presystolic rise coinciding with right atrial contraction. These findings are reflected in the jugular phlebogram by a rapid Y descent and in the right ventricular apex cardiogram by an initial early filling component followed by a delayed component. On the other hand, obstruction at the tricuspid valve orifice will be reflected in both the right atrial pressure pulse and the jugular phlebogram beside the raised pressure by a slow descent of the V wave, while it will be reflected in the right ventricular diastolic pressure pulse by the absence of an early diastolic dip and a smooth rise in the pressure to the next cycle, with absence of a presystolic rise in cases with sinus rhythm. The right ventricular apex cardiogram similarly will show absence of the rapid filling wave; the whole diastolic component will consist of a slow filling wave with no evidence of rapid filling (Fig. 4B). Analysis of the rate of Y descent in the jugular phlebogram is not always easy, and the tracing may

be subject to artefacts. Though the right atrial pressure pulse could be analysed, no working formulae are available similar to those of left atrial and wedge pressure tracings in mitral stenosis. This is probably related to the relatively low pressure in the right atrium in tricuspid stenosis. On the other hand, the right ventricular apex cardiogram will offer a simpler method for the study of the same phenomenon and will be in no way less reliable.

The present study indicates that analysis of the diastolic component of the right ventricular apex cardiogram is of particular help in the presence of atrial fibrillation for differentiating cases with dominant tricuspid stenosis – where practically always a certain degree of insufficiency is present – from cases with pure or dominant tricuspid insufficiency. The first group revealed a slow filling wave with no convincing evidence of rapid filling. The right ventricular apex cardiogram will become indispensable in the absence of the characteristic auscultatory findings. A tricuspid diastolic murmur was absent in 8 out of 10 cases of tricuspid stenosis with atrial fibrillation in one series (Sanders *et al.*, 1966). In these cases the apex cardiogram will help to stimulate further study.

On the other hand, the right ventricular apex cardiogram in tricuspid stenosis with sinus rhythm showed surprisingly enough a prominent presystolic a wave giving the tracing a bifid appearance. The left ventricular apex cardiogram in cases of mitral stenosis is characterized by absence of the a wave, which is explained simply by failure of transmission of the powerful left atrial contraction to the left ventricle because of the obstruction at the mitral valve (Fig. 2B). A similar phenomenon was expected to occur in the right ventricular apex cardiogram in cases with tricuspid stenosis, especially that the right ventricular diastolic pressure pulse showed absence of transmission of the giant A wave in the right atrial pressure pulse. The size of the a wave in the apex cardiogram was found to correlate fairly well with the calculated tricuspid valve area. The wave decreased after successful valvotomy and was absent in atrial fibrillation. There could be no doubt that it represented powerful right atrial contraction. The a wave percentage amplitude was distinctly higher compared with cases of mitral stenosis and pulmonary hypertension (Table 3). Since it could not be explained as a result of the atrial contraction causing the blood to enter the ventricle leading to a change in right ventricular end-diastolic volume – as in the case of pulmonary hypertension – it was feasible to consider it

as a representative of a direct tracing as sensed by the recording piece applied to the left sternal border. Revision of the anatomical characteristics of tricuspid stenosis and the findings during operation where the right atrium is appreciably enlarged in comparison to the right ventricle, and forms a wider area of contract with the chest wall, makes this explanation likely. External recording of right atrial pulse tracings is less surprising in view of a recent recording of external left atrial pulse tracings from the right anterior chest wall in cases with extreme left atriomegaly (unpublished observations).

As a result of using the right ventricular apex cardiogram as an investigating method in tricuspid valve disease, it becomes easier to detect by simple palpation at the left sternal borders the existence of either dominant tricuspid insufficiency or stenosis especially in cases with atrial fibrillation. In the presence of functional tricuspid insufficiency, there was usually an active parasternal impulse showing a prominent descent and a rebound wave ending with a palpable third sound, while in the presence of dominant tricuspid stenosis a more gentle impulse was seen. These findings represent the clinical reflection of the specific characteristics of the diastolic component of the right ventricular apex cardiogram in both conditions, and are at the same time the clinical counterpart of the rate of Y descent in the jugular venous pulse (see Fig. 5).

Compared with the jugular venous tracing, the right ventricular apex cardiogram represented a better tool for timing the events from the right side of the heart. Similar experiences were previously reported (Benchimol, Diamond, and Carson, 1961). We analysed with particular interest the O point of the right ventricular apex cardiogram which was used to time the tricuspid valve opening. With the help of simultaneous recording of both right and left apex cardiograms, a tricuspid opening snap could be identified in half the cases where it could be separated from a mitral snap that frequently occurred later, coinciding with the O point of the left ventricular apex cardiogram. The present work found precedence of the O point of the right ventricular apex cardiogram over the O point of the left ventricular record in 84 per cent of cases. This agrees with Perloff and Harvey (1960) but is at variance with other reports (McKusick, 1958).

Auscultation in tricuspid stenosis reflected to a great extent the haemodynamic characteristics of the disease. With sinus rhythm the murmur was mainly atriosystolic correspond-

ing to the maximum transvalvular gradient created by the atrial systole. However, a mid-diastolic component was not uncommonly registered in severe cases, which is again explained by the presence of a substantial gradient in mid-diastole in these cases. The presystolic murmur showed a characteristic crescendo-decrescendo character and lacked the apparent crescendo character of the mitral presystolic murmur. This has been previously ascribed to the prolonged PR interval which was commonly observed in patients with tricuspid stenosis (Perloff and Harvey, 1960; Goodwin *et al.*, 1957). We failed to show abnormal prolongation of the PR interval in our cases. On the other hand, a definitely prolonged PR interval will usually lead to a decrescendo character not only of tricuspid but also of mitral presystolic murmurs (Bousvaros and Stubington, 1964). A more adequate explanation for the decrescendo character of the tricuspid presystolic murmur is offered by our haemodynamic observation of the early summit of right atrial systole (AZ interval). Earlier onset of right compared with left atrial systole was previously reported (Braunwald, Fishman, and Cournand, 1956), and was found to explain reasonably the character of the tricuspid presystolic murmur (Bousvaros and Stubington, 1964). It is interesting to observe that inspiratory augmentation of the presystolic murmur did not affect its crescendo-decrescendo character (Fig. 8B), again reflecting the haemodynamic change.

With atrial fibrillation, the characteristic murmur is early mid-diastolic, with a distinct decrescendo character especially in long diastolic intervals where the murmur may end long before the next cardiac cycle. In short diastolic cycles and in the presence of severe obstruction the murmur may occupy the whole diastole. This reflects the characteristics of the diastolic gradient in the presence of atrial fibrillation. In short diastolic cycles the gradient may remain relatively constant, while in long diastolic pauses the gradient gradually diminishes and may be even abolished late in the cycle (Fig. 13A).

Tricuspid diastolic murmurs were usually considered to be of higher pitch than mitral diastolic murmurs (Killip and Lukas, 1958; Bousvaros and Stubington, 1964). We occasionally met with tricuspid mid-diastolic or presystolic murmurs that sounded superficial and high-pitched, sometimes with a scratchy or musical character (5 cases). In fact in 2 of these cases the murmur was first diagnosed as a pericardial rub. However, in the majority of cases (16), there was no appreciable difference between the tricuspid and mitral diastolic

murmurs, and analysis of their respective frequency response in the phonocardiogram confirmed this observation. Contrary to previous reports (Bousvaros and Stubington, 1964), we usually found no difficulty in differentiating a tricuspid diastolic murmur from an accompanying early diastolic murmur of aortic incompetence; the distinctly higher pitch of the latter murmur was usually unmistakable.

The effect of inspiration on the tricuspid murmurs represents the one single most valuable point in favour of the intimate correlation between the auscultatory and haemodynamic characteristics in tricuspid stenosis. All cases showed a substantial increase in the intensity of the diastolic murmur, which correlated well with the observed increase in the diastolic gradient. More interesting, however, was the effect of inspiration on the systolic murmur which either appreciably diminished or showed little change. This again correlated with the disproportionate drop in the right ventricular systolic pressure leading to an obvious decrease in the right ventricular-right atrial systolic gradient. The paradoxical inspiratory augmentation of the tricuspid diastolic murmur with the decrease in intensity of the tricuspid systolic murmur, especially in cases with atrial fibrillation – where nearly always an associated systolic murmur is present – is considered a crucial point for the diagnosis of dominant tricuspid stenosis. The explanation of the above haemodynamic and auscultatory findings has been previously suggested (Kitchin and Turner, 1964), though no previous reports were found. During inspiration in a normal heart, the obvious increase in the negative intrapleural pressure which is normally transmitted to all intrathoracic structures does not show to any appreciable extent in the right atrial and right ventricular pressure pulses, because it is well negated by the augmented inflow of blood into both chambers. In the presence of tricuspid stenosis, the right atrial pressure will still not show any appreciable change. On the other hand, the right ventricular pressure which suffers from the effect of severe restriction to its filling will reflect the full swings in intrapleural pressure. This will affect both its systolic and diastolic pressures, resulting in a concomitant increase in right atrial/right ventricular diastolic gradient, and hence the increase in the diastolic murmur, and a decrease in right ventricular/right atrial systolic gradient, and hence decrease of the systolic murmur.

In the present series the tricuspid opening snap constituted an important auscultatory finding, being recorded in nearly half the

cases. In a recent report (Luisada, Slodki, and Krol, 1965), the tricuspid opening snap was frequently observed when simultaneous phonocardiograms were recorded from both the mitral and tricuspid areas. In fact in this report the tricuspid snap was rarely associated with anatomical abnormality of the valve itself. The interval between the first high-pitched components of the second sound and the tricuspid opening snap was measured and was found to range from 0.03 to 0.11 sec. It was designated II-OS T interval, and corresponded exactly with the II-O T interval of the right ventricular apex cardiogram. Though classically this interval has to be measured from the pulmonary component of the second sound, yet frequently we failed to show splitting of the second sound in our cases with tricuspid stenosis. This interval is practically considered equivalent to the isometric phase of relaxation of the right ventricle and similar to the mitral II-OS interval (Messer *et al.*, 1951; Mounsey, 1953); it showed considerable variation in duration with the heart rate and in the presence of atrial fibrillation attributed to variations in ventricular filling and atrio-ventricular pressure gradient with changes in the cycle length.

The effect of tricuspid stenosis on the haemodynamics of the accompanying mitral stenosis was again reflected on auscultation. Three patients in the present series showed absence of a diastolic murmur of mitral stenosis; in one of them a short presystolic murmur appeared on exercise. This reflected the finding of a normal resting wedge pressure in these cases which showed a moderate increase with exercise in only one case. Thus, the failure to show a separate mitral diastolic murmur in the presence of severe tricuspid stenosis and/or the finding of a normal resting wedge pressure in these cases cannot exclude the presence of an associated obstruction at the mitral orifice which can only be surely confirmed at operation. Though the parameters to judge 'silence' of the mitral stenosis varied widely according to different authors, yet severe tricuspid stenosis can be safely added to the other mechanisms for silent mitral stenosis (El-Sherif *et al.*, 1968). A more interesting observation was recorded in two other cases with atrial fibrillation where a systolic and mid-diastolic murmur was heard over and inside the apex, while a clear sharp first sound and an opening snap were heard outside the apex. Inspiration revealed augmentation of the diastolic murmur and a decrease in the intensity of the systolic murmur denoting their tricuspid origin. The mitral stenosis is silent in so far as the diastolic mur-

mur is concerned. These two cases showed a Qr pattern in V₁, and right atrial injection of dye in one case showed the tricuspid valve considerably displaced to the left. These two cases simulated the more common syndrome of tricuspid insufficiency masquerading as mitral insufficiency in patients with pure mitral stenosis (Uricchio *et al.*, 1958), and can be called 'dominant tricuspid stenosis masquerading as mitral stenosis and insufficiency in patients with pure mitral stenosis'.

The genesis of P wave changes in tricuspid stenosis merits a separate comment. Our frequent observation after successful tricuspid valvotomy, where appreciable decrease in the height of P waves occurred in the immediate post-operative period (Fig. 15), cannot be simply ascribed to regression of a hypertrophied right atrium, which is a non-transient condition. It can be more reasonably attributed to changes in the anatomical heart position and rotation after successful operation. The relative importance of these changes in the genesis of the P pulmonale pattern, apart from the degree of hypertrophy and dilatation of the right atrium, finds its support in the fleeting occurrence of P pulmonale pattern during acute episodes of pulmonary embolism, status asthmaticus, and pulmonary oedema (Massie and Walsh, 1960). More recently, an increase in the height of P waves was observed in patients with emphysema cor pulmonale after exposure to acute thermal stress for two hours (El-Sherif, Shahwan, and Sorour, 1970).

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