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MORPHOLOGIC STUDIES

Etiology of Pure Tricuspid Regurgitation Based on Anular Circumference and Leaflet Area: Analysis of 45 Necropsy Patients With Clinical and Morphologic Evidence of Pure Tricuspid Regurgitation

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Despite recent renewed interest in the detection of tricuspid valve regurgitation by echocardiographic and Doppler techniques, little morphologic information is available on dysfunctioning tricuspid valves. This report describes 45 necropsy patients with clinical and morphologic evidence of *pure* (no element of stenosis) tricuspid regurgitation and provides morphometric observations (anular circumference, leaflet area) of the tricuspid valve useful in determining the etiology of pure tricuspid regurgitation. Of 45 patients, 24 (53%) had pure tricuspid regurgitation resulting from an anatomically abnormal valve (prolapse in 7, papillary muscle dysfunction in 6, rheumatic disease in 5, Ebstein's anomaly in 3, infective endocarditis in 2, carcinoid tumor in 1), and 21 (47%) had an anatomically normal valve with systolic pulmonary artery hypertension (cor pulmonale in 12, mitral stenosis in 9). Anular circumference was dilated (> 12 cm) in patients with various causes of pulmonary hypertension, floppy valve and Ebstein's tricuspid anomaly. Leaflet area was increased in floppy valve and Ebstein's anomaly.

Of the 45 patients, 24 had pulmonary systolic artery pressure measurements available for correlation with tricuspid valve morphology. Pulmonary artery pressures accurately predicted morphologically normal from abnormal valves in 16 patients (89%). Morphologic overlap occurred in six patients with pulmonary pressures of 41 to 54 mm Hg. Of these six, the additional knowledge of normal or dilated anular circumference correctly separated valves with normal and abnormal leaflets.

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Various clinical manifestations of tricuspid regurgitation have interested physicians since the early 19th century (1). Despite this early interest, only five studies (1–5) in the last 157 years have provided detailed description or classification, or both, of human tricuspid valve morphology-histology pertaining to structure-function relations of the various elements of the tricuspid valve apparatus. Recent developments in echocardiographic techniques for the measurement of anular circumference and detection of tricuspid regurgitation by contrast (6) and pulsed Doppler (7) echocardiography have stimulated new interest in morphologic aspects of purely regurgitant tricuspid valves. The following report of 45 necropsy patients with clinical evidence of tricuspid regurgitation provides morphometric observations of the tricuspid valve useful in determining the etiology of pure tricuspid regurgitation and predicting tricuspid valve morphology in patients with combined pure tricuspid regurgitation and mitral valve stenosis.

Methods

Morphologic criteria and definitions. *Pure regurgitation.* Because the diagnosis of tricuspid regurgitation as established by clinical examination and hemodynamic and angiographic studies is not consistently adequate, certain morphologic variables were used as primary criteria in defining dysfunctioning tricuspid valves as purely regurgitant (no element of stenosis) or stenotic (8). Purely regurgitant tricuspid valves have one or more of the following morphologic features: focal to extensive leaflet fibrous thick-

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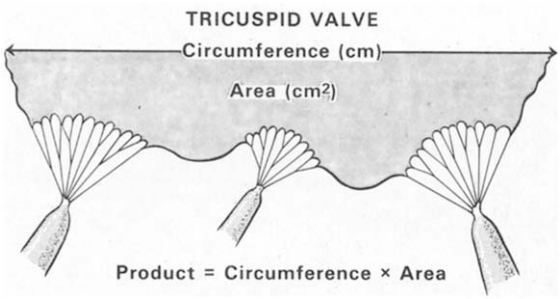


Figure 1. Diagram showing three morphometric variables recorded for each study and control tricuspid valve.

ening, absent calcific deposits, absent commissural fusion and nonfused chordae tendineae. In contrast, stenotic or combined stenotic and regurgitant (so-called predominantly regurgitant) tricuspid valves have extensive or diffuse leaflet fibrous thickening, fusion of commissures with or without mild fusion of chordae tendineae and, occasionally, calcific deposits. The following morphologic criteria were used for etiologic subgrouping of purely regurgitant tricuspid valves.

Rheumatic. Tricuspid valves with extensive or diffuse leaflet fibrosis without commissural fusion or chordae tendineae fusion in the presence of mitral valve stenosis were classified under rheumatic.

Table 1. Certain Clinical Observations in 45 Necropsy Patients With Pure Tricuspid Regurgitation

Age at death (yr)		17 to 72 (49)
Sex (M:F)		29 (64%):16 (36%)
Evidence of tricuspid regurgitation (no. of patients)		
Jugular venous dilation/pulsation	36	
Hepatic pulsation	19	
Right ventricular impulse	21	
Systolic murmur	43	
Contrast echocardiogram	2	
Right ventricular angiogram	6	
Elevated mean right atrial pressure	24	
Range (average) systolic pulmonary artery pressures in 24 patients (53%)		
Cor pulmonale, 12 patients (SLE = 1, 1° PH = 1, COPD = 9, chronic PE = 1)		40 to 90 (55) mm Hg
Mitral stenosis, 10 patients		30 to 70 (55) mm Hg
Other, 2 patients (IE = 1, Ebstein's anomaly = 1)		45, 55 (50) mm Hg
Number of patients with systolic pulmonary artery hypertension > 35 mm Hg		23 (96%)
COPD, 12 of 12 patients		
Mitral stenosis, 9 of 10 patients (3 of 4 rheumatic TR, 6 of 6 functional TR)		
Other, 2 of 2 patients		

COPD = chronic obstructive pulmonary disease; F = female; IE = infective endocarditis; M = male; PE = pulmonary embolism; PH = pulmonary hypertension; SLE = systemic lupus erythematosus; TR = tricuspid regurgitation.

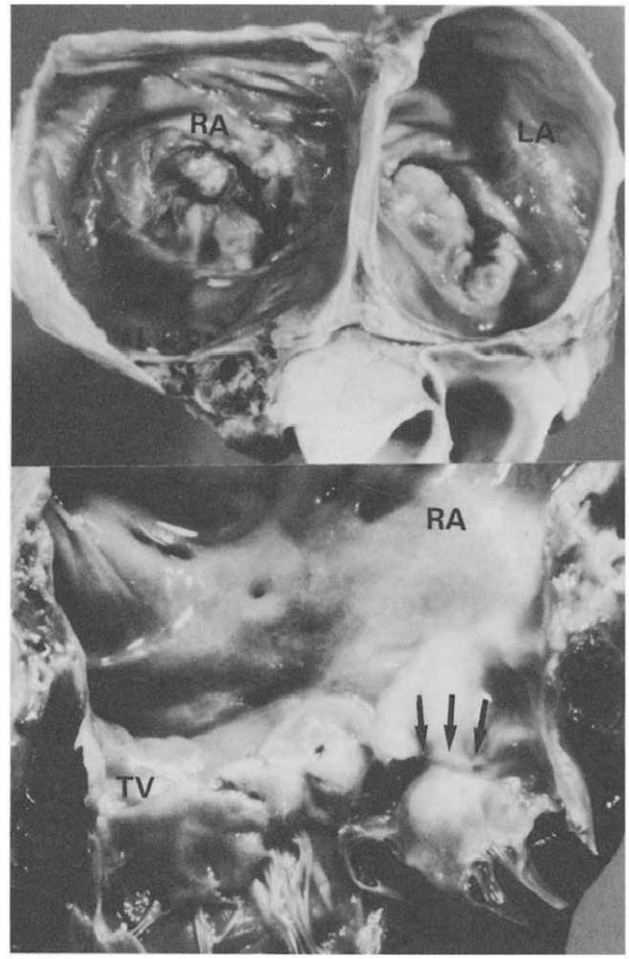


Figure 2. Pure tricuspid regurgitation due to floppy tricuspid valve (TV). **Top,** Note dilated anular circumference and tricuspid valve leaflets protruding into the right atrium (RA). **Bottom,** Prolapsing tricuspid leaflet and "buckling" hinge point (**arrows**). LA = left atrium.

Floppy (prolapsed). Tricuspid valves with focal elongation of at least one leaflet segment (increased distance between anular insertion and margin of closure) and dilated anulus (> 12 cm) were classified under floppy valve (prolapse).

Infective endocarditis. Tricuspid valves with leaflet vegetations, indentations or perforations with or without ruptured chordae tendineae were classified under active or healed infective endocarditis.

Atherosclerotic coronary heart disease with papillary muscle dysfunction. Tricuspid valves with normal or focally thickened leaflets and chordae tendineae without leaflet indentation or perforation and associated with at least one necrotic or scarred and atrophied papillary muscle were classified under papillary muscle dysfunction. At least one major epicardial coronary artery was narrowed more than 75% in cross-sectional area by atherosclerotic plaque.

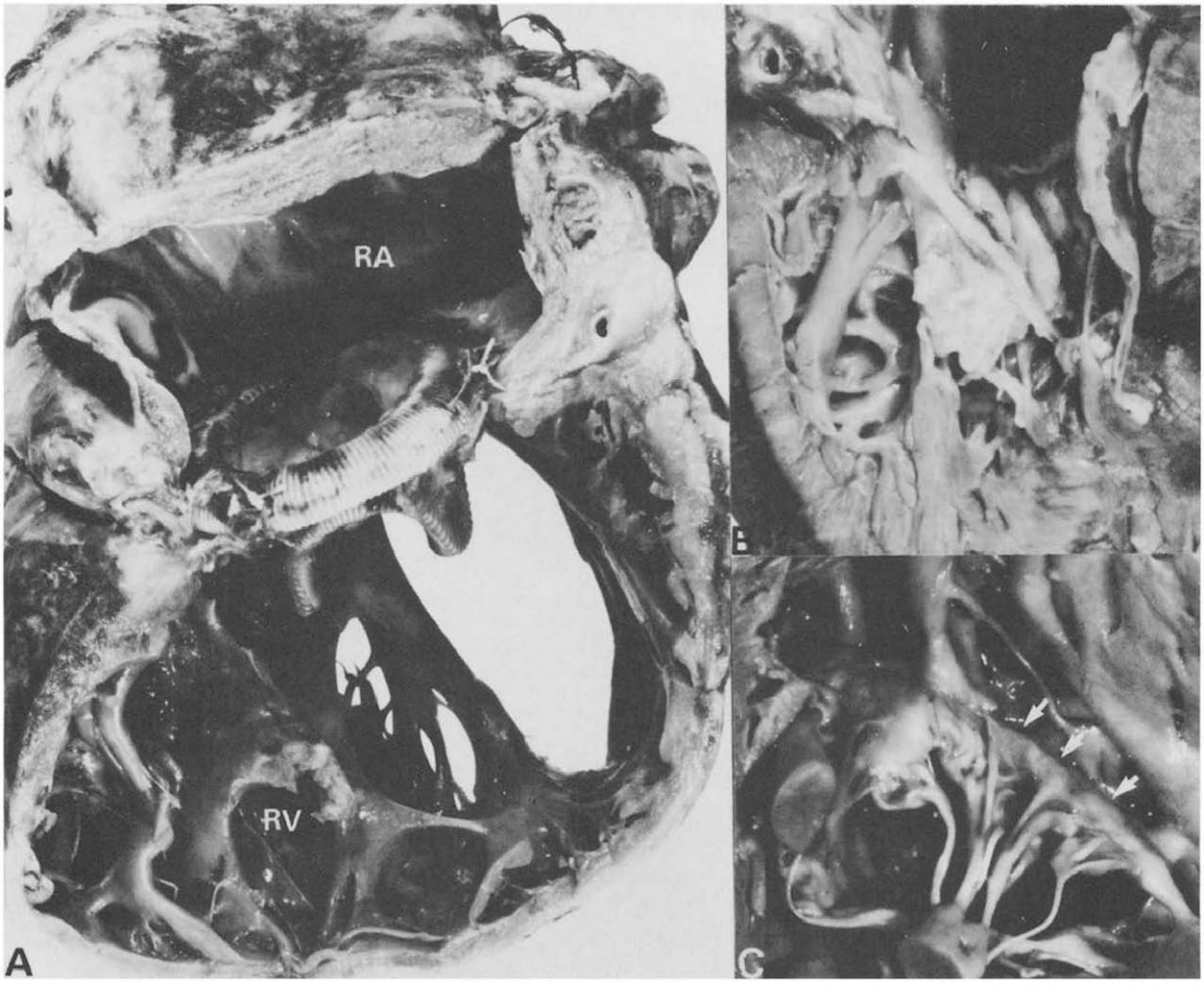
Patients studied. Hearts of 52 necropsy patients greater than 15 years of age with clinical evidence of tricuspid

regurgitation were identified in the Indiana University Cardiovascular Pathology Registry. Of these 52 hearts, 7 had a triscuspid valve with fusion of commissures and diffuse leaflet fibrous thickening indicating some element of tricuspid valve stenosis. These seven patients were excluded from further study. Medical records including results of cardiac catheterization and angiographic and echocardiographic procedures were reviewed in the remaining 45 patients and the anatomic diagnosis of a purely regurgitant tricuspid valve was substantiated. These 45 necropsy patients with clinical and morphologic evidence of pure tricuspid regurgitation constitute the study group. None of the 45 study patients had an intracardiac shunt.

Morphometric variables. The tricuspid valve in each of the 45 hearts was excised along its anular insertion site and removed intact. Three morphometric variables (Fig. 1) were examined in each valve: 1) anular circumference (in centimeters), 2) leaflet area (in square centimeters), and 3) product of circumference times area (in cubic centimeters). The circumference was determined by measuring the length of the anulus of the excised leaflets, and the area was determined by outlining the basal and distal margins of the leaflet on paper and measuring the resulting area by videoplanimetry. The calculation of the product of anular circumference times leaflet area may be viewed as an index of volume of the tricuspid valve orifice.

Clinical data. Table 1 summarizes certain clinical information in the 45 necropsy patients with pure tricuspid regurgitation. Clinical evidence of tricuspid regurgitation was variable and incomplete but at least one of seven clinical or laboratory variables was present in each of the 45 patients, and a precordial systolic murmur characteristic of tricuspid regurgitation was noted in 43 (96%). Additional physical findings indicative of tricuspid regurgitation included: jugular venous distension and pulsation (36 patients), right ventricular systolic impulse (21 patients) and hepatic systolic pulsations (19 patients). Of the 45 patients, 24 (53%) underwent cardiac catheterization or had Swan-Ganz catheter pressure measurements available within 60 days of death.

Figure 3. Pure tricuspid regurgitation due to primary disease of the valve complex. **A**, Tricuspid valve replacement for Ebstein's anomaly. Note dilated right atrium (RA) and ventricle (RV). **B**, Coronary atherosclerosis. Papillary muscle and adjacent ventricular septal necrosis from acute inferior myocardial infarction with an attempt at surgical repair. **C**, Carcinoid heart disease. Carcinoid plaques (**arrows**) causing tricuspid leaflet thickening.



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Of the 24 patients with pressure recordings, the systolic pulmonary artery pressure ranged from 30 to 70 mm Hg (average 53). Of these 24 patients, 23 (96%) had systolic pulmonary artery hypertension (defined as a pressure > 35 mm Hg).

Necropsy data. At necropsy, the right atrial and right ventricular cavities were variably dilated in all 45 patients. Of the 45 tricuspid valves, 7 had leaflet "hooding" or redundancy, 5 had diffuse leaflet fibrous thickening without commissural fusion, 3 had "displaced" anular insertion sites, 2 had leaflet vegetations without ruptured chordae tendineae and 1 had extensive leaflet adherence to mural endocardium by carcinoid fibrous plaques. The remaining 27 tricuspid valves had anatomically normal leaflets except for focal areas of mild fibrous thickening. One of these 27 valves had necrosis of the papillary muscles associated with acute right ventricular infarction. None of the 45 valves had ruptured chordae tendineae.

Control subjects. The tricuspid valves from the hearts of 25 necropsy patients who had no cardiac abnormalities were excised and measured in a manner similar to that in the study group. The patients' ages at death ranged from 19 to 80 years (average 47), and all died from noncardiovascular events. The prevalence of men and women was similar in the control and study groups.

Results

Etiology of pure tricuspid regurgitation. Gross examination of the 45 necropsy-excised tricuspid valves dis-

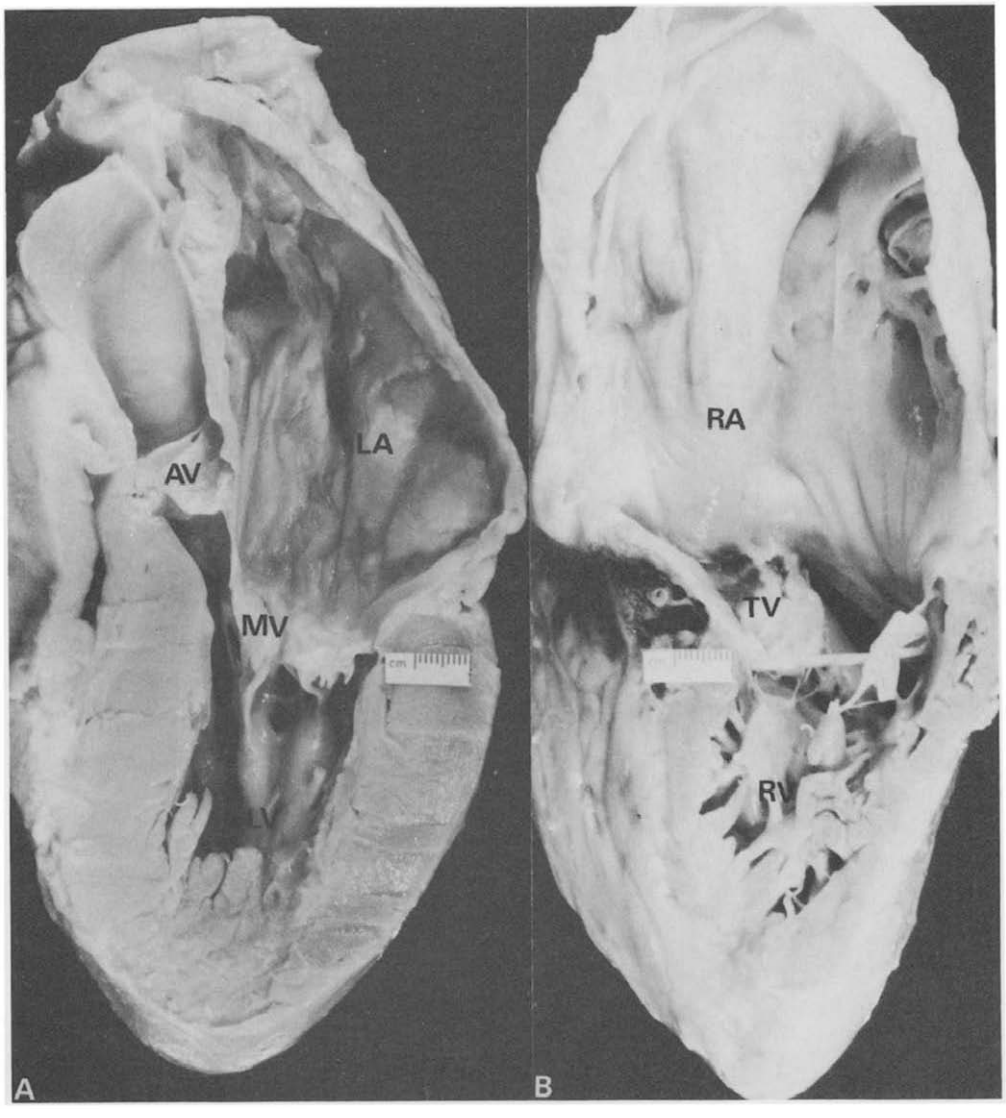
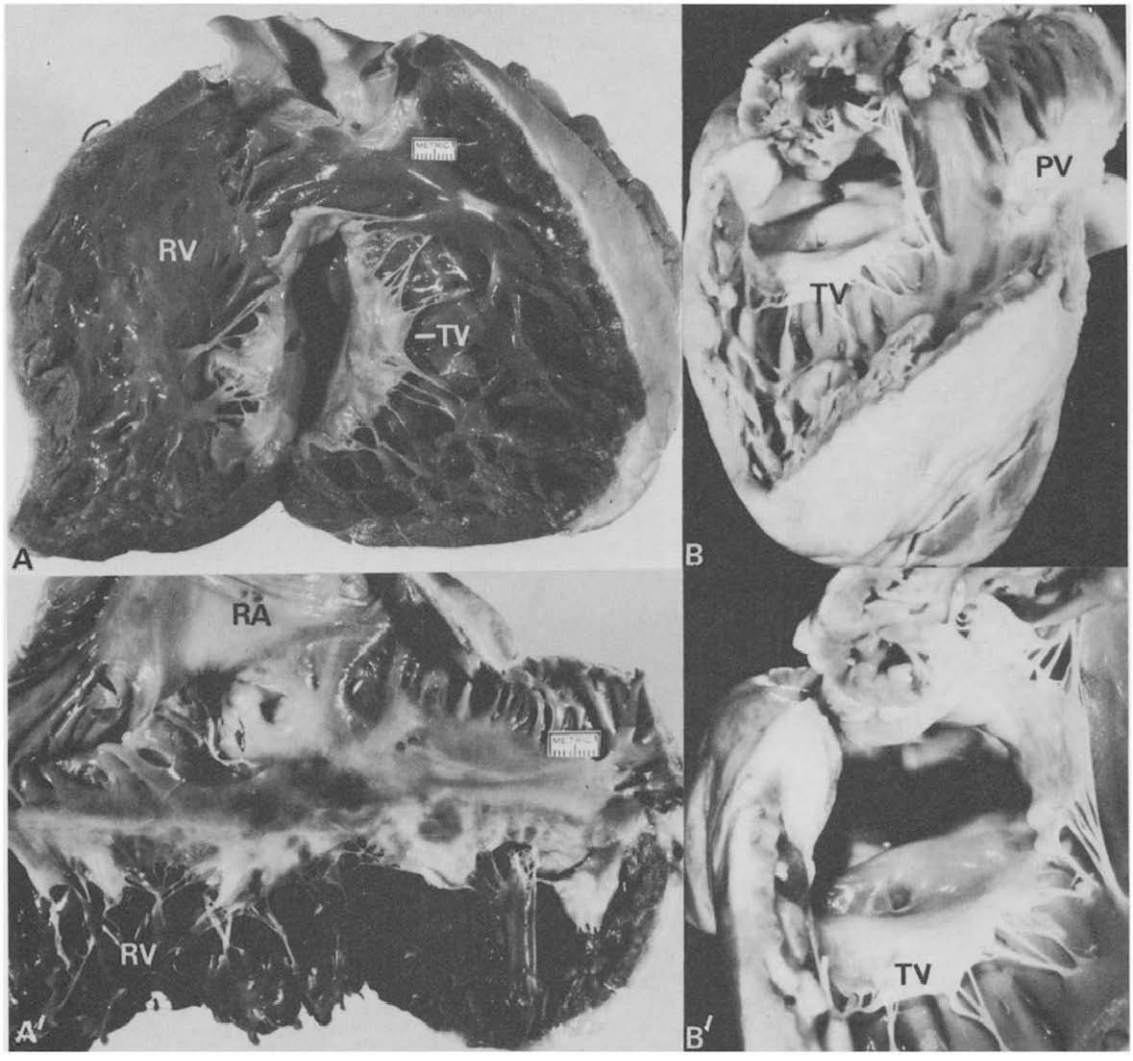


Figure 4. Pure tricuspid regurgitation due to primary rheumatic disease of the tricuspid valve (TV). **A**, Left-sided view showing stenosis of the mitral valve (MV) with a dilated left atrium (LA). **B**, Diffuse fibrous thickening of the tricuspid valve leaflets, absent commissural fusion and normal tricuspid valve anulus. The right atrium (RA) is markedly dilated. AV = aortic valve; LV = left ventricle; RV = right ventricle.



closed that the causes of pure regurgitation could be classified into conditions associated with anatomically *abnormal* valves (''organic,'' ''primary'') (Fig. 2 to 4) and conditions associated with anatomically *normal* tricuspid valves (''functional,'' ''secondary'') (Fig. 5 and 6) (Table 2, Fig. 7). Of the 45 patients, 24 (53%) had pure tricuspid regurgitation resulting from an anatomically abnormal valve: 7 (16%) with leaflet prolapse (floppy) (Fig. 2); 6 (13%) with papillary muscle dysfunction (idiopathic dilated cardiomyopathy in 4, atherosclerotic coronary heart disease in 2) (Fig. 3B); 5 (11%) with rheumatic disease (Fig. 4); 3 (7%) with Ebstein's anomaly (Fig. 3A); 2 (4%) with active infective endocarditis; and 1 (2%) with carcinoid tumor (Fig. 3C). The remaining 21 patients (47%) had pure tricuspid regurgitation associated with an anatomically normal valve and pulmonary artery systolic hypertension: 12 (27%) had cor pulmonale (chronic obstructive pulmonary disease in 9 [Fig. 5A], systemic lupus erythematosus in 1, chronic pulmonary emboli in 1, primary pulmonary hypertension in 1 [Fig. 5B]) and 9 (20%) had mitral valve stenosis (Fig. 6).

Figure 5. Pure tricuspid regurgitation due to cor pulmonale. **A**, Chronic obstructive pulmonary disease. Right ventricular (RV) view of the tricuspid valve (TV) showing a dilated tricuspid valve anulus. **A'**, Opened tricuspid valve showing a dilated anular circumference with anatomically normal leaflets and chordae tendineae. RA = right atrium. **B**, Chronic pulmonary emboli. Opened right ventricle showing a dilated outflow tract and tricuspid valve anulus. PV = pulmonary valve. **B'**, Close-up of tricuspid valve showing a dilated anular circumference with anatomically normal leaflets and chordae tendineae.

In this necropsy series of 45 patients with clinical and morphologic evidence of pure tricuspid regurgitation, nonrheumatic conditions accounted for 40 patients (89%) and rheumatic tricuspid disease the remaining 5 patients (11%). Isolated (confined to the tricuspid valve) pure tricuspid regurgitation was found in 18 patients (40%): cor pulmonale in 12, Ebstein's anomaly in 2, coronary papillary muscle dysfunction in 2 and infective endocarditis in 2. The remaining 27 patients (60%) had pure tricuspid regurgitation associated with another dysfunctioning (stenotic or purely

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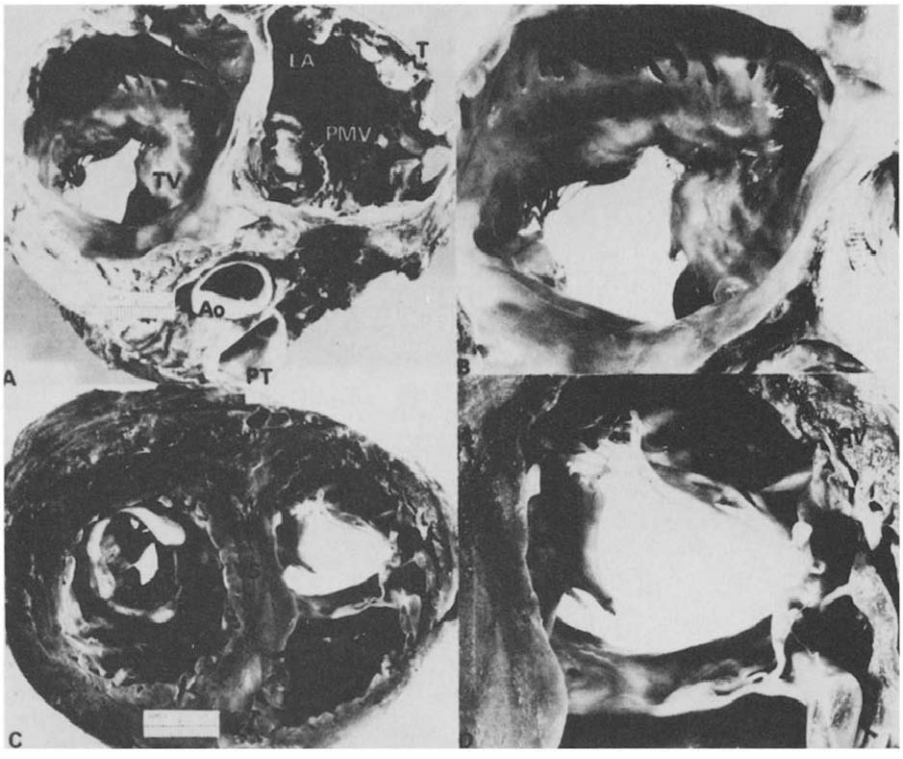


Figure 6. Pure tricuspid regurgitation due to pulmonary hypertension secondary to mitral stenosis. **A**, View of heart showing prosthetic mitral valve (PMV) previously inserted for mitral valve stenosis, and tricuspid valve (TV) with dilated right atrium (RA). Ao = aorta; LA = left atrium. **B**, Close-up (atrial view) of the tricuspid valve showing anatomically normal leaflets with a dilated anulus. **C**, View of right and left ventricular (LV) cavities showing a dilated and hypertrophied right ventricle. VS = ventricular septum. **D**, Close-up (ventricular view) of the regurgitant tricuspid valve showing normal leaflets. Compare this tricuspid valve with that shown in Figure 5. PT = pulmonary trunk; RV = right ventricle; T = thrombus.

regurgitant) valve: 15 stenotic mitral valves, 7 purely regurgitant floppy mitral valves, 4 purely regurgitant mitral valves in dilated cardiomyopathy and 1 stenotic pulmonary valve in carcinoid heart disease. One patient with Ebstein's anomaly also had a stenotic mitral valve (probably rheumatic in origin) with pulmonary hypertension, and of the remaining 14 patients with a rheumatic stenotic mitral valve, 8 had previous mitral valve replacement or commissurotomy. All seven patients with a floppy tricuspid valve had an associated floppy mitral valve.

Table 2. Etiology of Pure Tricuspid Regurgitation in 45 Patients

	No. (%)	
Anatomically abnormal valves	24 (53)	
Floppy valve (prolapse)	7 (16)	
Papillary muscle dysfunction	6 (13)	
Idiopathic dilated cardiomyopathy		4
Atherosclerotic coronary heart disease		2
Rheumatic disease	5 (11)	
Ebstein's anomaly	3 (7)	
Infective endocarditis	2 (4)	
Carcinoid tumor	1 (2)	
Anatomically normal valves	21 (47)	
Pulmonary hypertension		
Cor pulmonale	12 (27)	
Chronic obstructive pulmonary disease		9
Systemic lupus erythematosus		1
Chronic pulmonary emboli		1
Primary pulmonary hypertension		1
Mitral stenosis	9 (20)	

Anular circumference. The circumference of the tricuspid valve anulus measured in the 25 necropsy control patients without heart disease ranged from 8.5 to 12.5 cm (average 11.0) (Table 3, Fig. 8 and 9). The anular circumference was not significantly different in the 24 necropsy tricuspid valves rendered regurgitant by rheumatic fever, carcinoid tumor, atherosclerotic coronary artery disease and active infective endocarditis (10.5 to 12.0 cm, average 10.9) (Table 3) (Fig. 3B, 3C and 4). The anular circumferences of the tricuspid valves in the 24 patients with various causes of cor pulmonale, dilated cardiomyopathy and nonrheumatic tricuspid valves associated with systolic pulmonary artery hypertension from mitral stenosis were significantly larger than control measurements and those in the previously described causes (13.0 to 15.5 cm, average 14.1) ($p < 0.05$) (Fig. 2, 3A, 5 and 6). Furthermore, the anular circumference of floppy and Ebstein's tricuspid valves was significantly larger than in all other study groups and control valves (14.0 to 19.0 cm, average 16.8) ($p < 0.01$) (Table 3, Fig. 2 and 3A). Average anular circumference of the three patients with Ebstein's anomaly was larger than that of the seven patients with a floppy tricuspid valve (18.2 and 15.4 cm, respectively).

Leaflet area. In the 25 control patients, the area of necropsy-excised tricuspid valve leaflets ranged from 9.0 to 18.8 cm (average 13.9) (Table 3, Fig. 9). In seven of the nine conditions producing pure tricuspid regurgitation (cor pulmonale, pulmonary hypertension from mitral stenosis, dilated cardiomyopathy, rheumatic disease, carcinoid tumor, coronary disease and infective endocarditis), the tri-

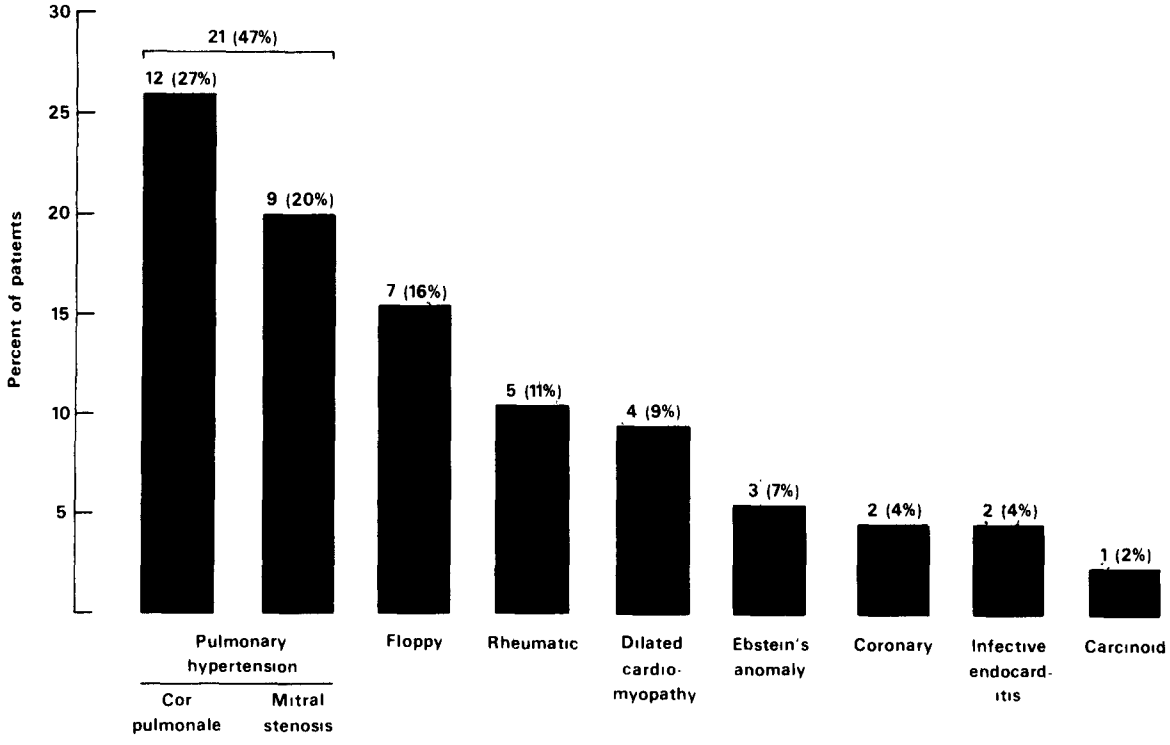


Figure 7. Etiology of pure regurgitation in 45 necropsy patients with clinical evidence of tricuspid regurgitation.

cuspid leaflet areas were not significantly different from those of the control valves (11.4 to 21.3 cm^2, average 13.7 and 9.0 to 18.8 cm^2, average 13.9, respectively). In necropsy patients with a floppy tricuspid valve or Ebstein's anomaly, the leaflet areas were significantly larger than those in the remaining study groups or control subjects (23.4 to 34.4 cm^2, average 29.4) ($p < 0.01$) (Table 3). Furthermore, tricuspid valves associated with Ebstein's anomaly had a larger leaflet area than that of floppy tricuspid valves (average 32.2 and 26.5 cm^2, respectively) ($p < 0.05$).

Product of valve circumference and area. The product of the circumference (in centimeters) times the area (in square centimeters) calculated for normal tricuspid valves

Table 3. Pure Tricuspid Regurgitation (TR): Morphometric Observations in 45 Necropsy Patients and 25 Control Subjects

Etiology of TR	Anular Circumference (cm)	Leaflet Area (cm^2)	Circumference × Area Product (cm^3)
Floppy valve (prolapse)	14.0 to 17.4 (15.4)	23.4 to 29.0 (26.5)	339 to 467 (415)
Ebstein's anomaly	17.5, 18.0, 19.0 (18.2)	29, 33.1, 34.4 (32.2)	508, 596, 654 (586)
Right ventricular cavity dilation			
Cor pulmonale	13.0 to 15.5 (14 1)	11.4 to 21.3 (14.9)	151 to 302 (211)
Chronic obstructive pulmonary disease	13.0 to 15.5 (14.3)	11.4 to 21.3 (14.4)	151 to 302 (191)
Systemic lupus erythematosus	13.0	14.1	187
Primary pulmonary hypertension	15.0	14.7	228
Chronic pulmonary emboli	14.5	16.4	237
Dilated cardiomyopathy	13.0 to 15.0 (13.9)	11.7 to 17.1 (14.7)	175 to 255 (196)
Mitral stenosis	12.0 to 14.5 (13.6)	11.9 to 18.8 (14.6)	149 to 273 (201)
Rheumatic disease	11 0 to 12.0 (11.5)	12.6 to 16.6 (14.2)	142 to 185 (164)
Carcinoid tumor	10.5	11.5	121
Coronary disease	10.5, 10.0 (10.8)	12.4, 13.5 (13.0)	130, 149 (139)
Infective endocarditis	10.5, 10 0 (10.8)	11.6, 14.1 (12.8)	122, 155 (138)
Control subjects	8.5 to 12.5 (11.0)	9.0 to 18.8 (13.9)	94 to 216 (155)

Values in parentheses indicate the mean

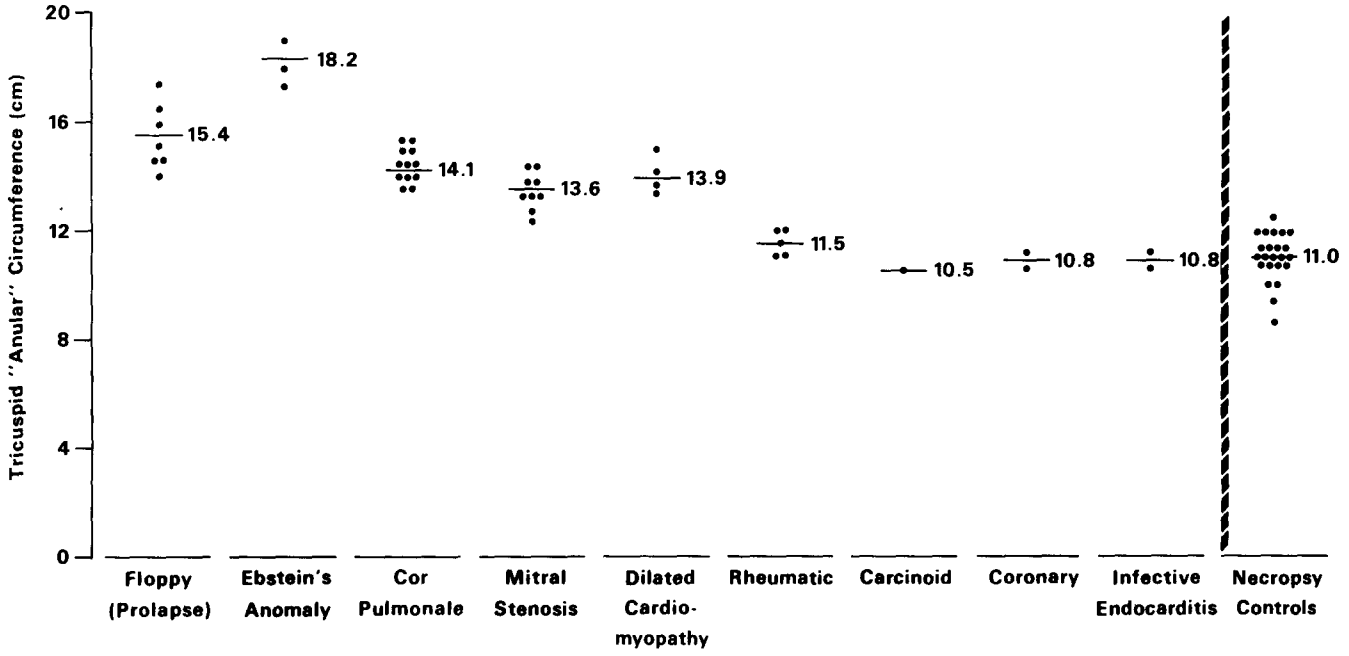
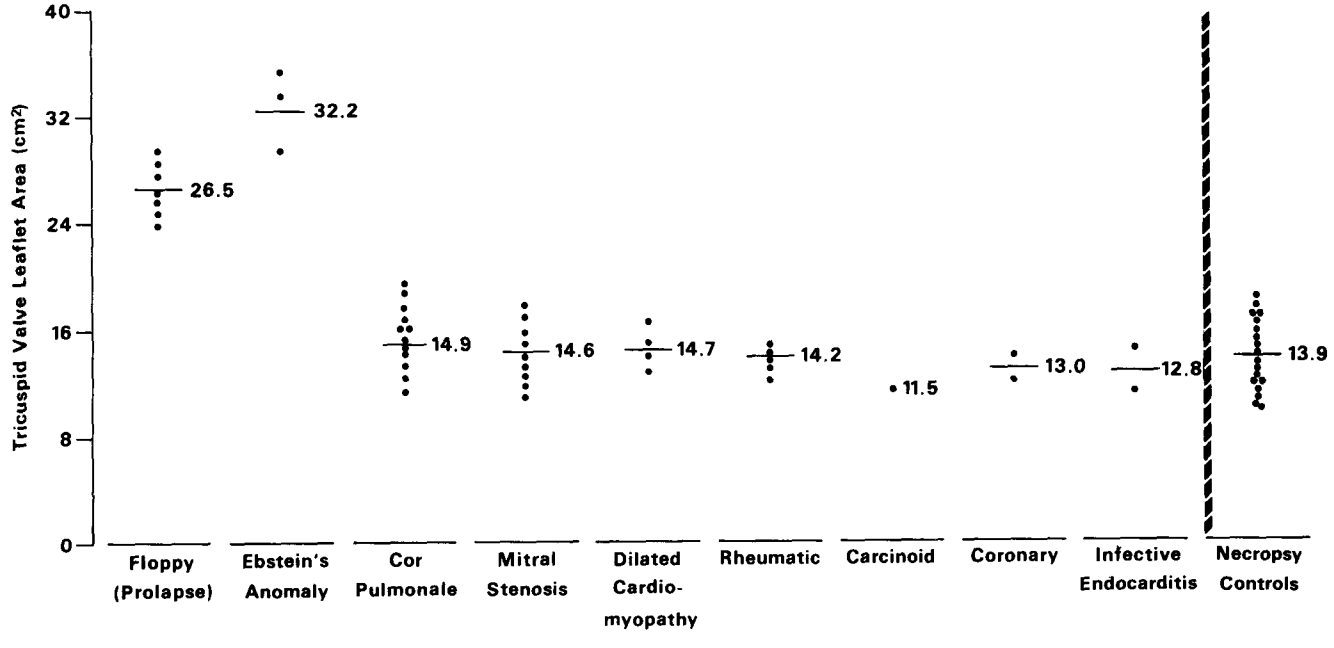


Figure 8. Tricuspid valve anular circumference (in centimeters) in nine conditions causing pure tricuspid regurgitation in 45 necropsy patients and 25 necropsy control subjects.

ranged from 94 to 216 cm^3 (average 155) (Table 3, Fig. 10). The average product was 586 cm^3 for the Ebstein anomaly valves, which was significantly larger than the average product of 415 cm^3 for the floppy tricuspid valves ($p < 0.01$). Both Ebstein's anomaly and floppy tricuspid valves had average products significantly larger than those of the remaining groups of purely regurgitant valves (167 cm^3) and control valves (155 cm^3) ($p < 0.001$) (Table 3).

Figure 9. Tricuspid valve leaflet area (in square centimeters) in nine conditions causing pure tricuspid regurgitation in 45 necropsy patients and 25 necropsy control subjects.

Tricuspid valve morphology and systolic pulmonary artery pressures. Of the 45 necropsy patients, 24 had systolic pulmonary artery pressures available for correlation with tricuspid valve morphology (Table 1). Of 14 patients with a pressure of 55 mm Hg or more, 13 (93%) had anatomically normal leaflets and a dilated anular circumference. The single patient in this subgroup with anatomically abnormal leaflets was a patient with Ebstein's anomaly who also had pulmonary hypertension secondary to rheumatic



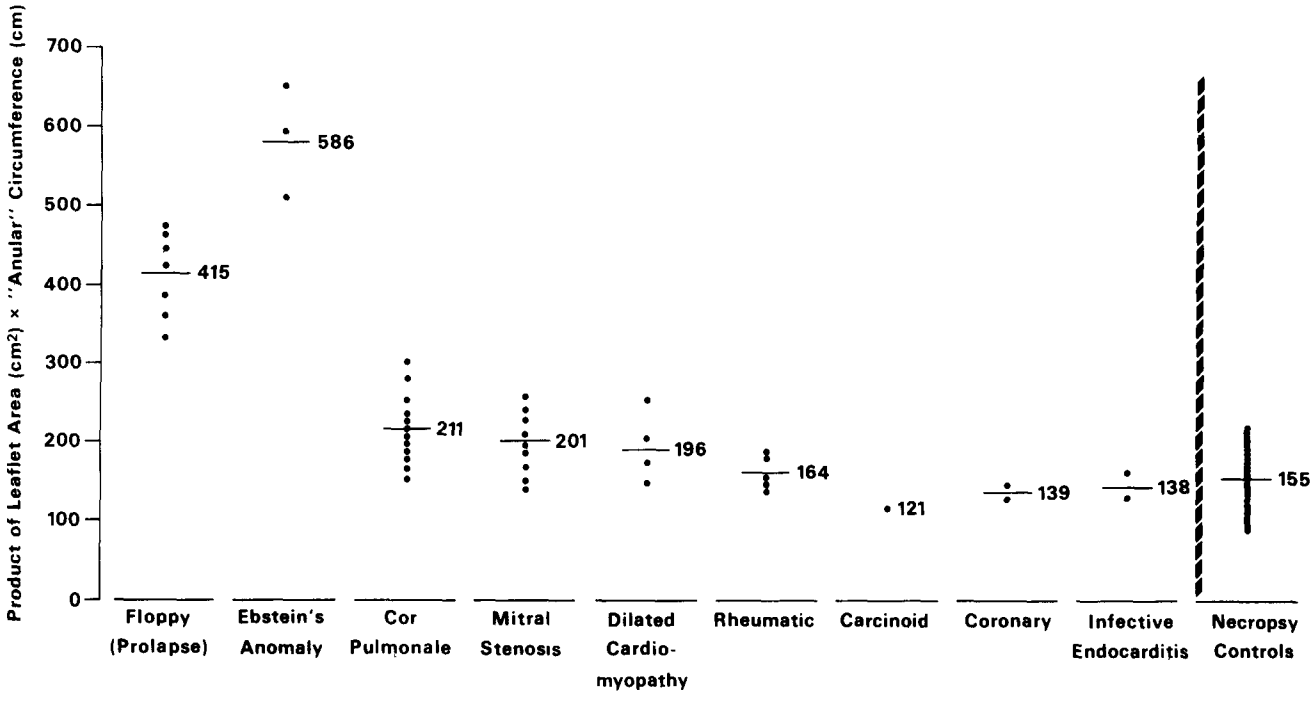


Figure 10. Product of tricuspid anular circumference (in centimeters) times leaflet area (in square centimeters) in nine conditions causing pure tricuspid regurgitation in 45 necropsy patients and 25 necropsy control subjects.

mitral stenosis. In contrast, of four patients with a systolic pulmonary artery pressure of 40 mm Hg or less, three had abnormal leaflets and normal anular circumference. The remaining patient had anatomically normal leaflets and a dilated anulus with previous mitral valve replacement for mitral stenosis. Thus, systolic pulmonary artery pressures accurately separated morphologically normal from abnormal tricuspid valves in 16 (89%) of 18 patients. Morphologic overlap occurred in six patients with a pulmonary systolic pressure of 41 to 54 mm Hg. Of these six patients, four had anatomically normal leaflets and two had abnormal leaflets. Each of the four patients with normal leaflets had a dilated anular circumference (13.5 to 15.5 cm, average 14.3) and the two patients with abnormal leaflets had a normal anular circumference (11.0 and 12.0 cm). Thus, knowledge of a normal or dilated anular circumference accurately separated the six overlap patients.

Purely regurgitant tricuspid valves with primary ("organic") and secondary ("functional") changes from rheumatic disease. A subgroup of patients with pure tricuspid regurgitation comprises those patients with associated rheumatic mitral stenosis. In the present study group of 45 patients with pure tricuspid regurgitation, 14 had mitral stenosis (with or without previous surgical repair or replacement) and pure tricuspid regurgitation. Of the 14 tricuspid valves, 5 (36%) were anatomically abnormal valves ("organic," "primary") with extensive to diffuse leaflet fibrous thickening, and 9 (64%) had anatomically normal leaflets ("functional," "secondary").

Anular circumferences of these 14 purely regurgitant tricuspid valves separated the valves into two subgroups. Of the five anatomically abnormal tricuspid valves (that is, primary rheumatic involvement), the anular circumference ranged from 11.0 to 12.0 cm (average 11.5). In contrast, anular circumference measurements for the anatomically normal tricuspid valves (that is, secondary effects of rheumatic mitral stenosis) were significantly larger, ranging from 12.5 to 14.5 cm (average 13.7) ($p < 0.05$). Leaflet areas of the anatomically abnormal valves ranged from 12.9 to 15.4 cm^2 (average 13.9) and were not significantly different from the leaflet areas of the anatomically normal purely regurgitant tricuspid valves (11.9 to 18.8 cm^2, average 14.8). The products of circumference times area in the anatomically abnormal tricuspid valves ranged from 142 to 185 cm^3 (average 168), and were significantly smaller than the products of anatomically normal tricuspid valves (149 to 273 cm^3, average 208) ($p < 0.01$). *Thus, in patients with pure tricuspid regurgitation associated with rheumatic mitral stenosis, those with an anatomically abnormal tricuspid valve ("organic" disease) have a significantly smaller anular circumference and product of circumference times area than those with an anatomically normal tricuspid valve ("functional" disease).*

Comparison of pulmonary artery systolic pressures and tricuspid valve morphology in these 14 patients disclosed that in 4 of the 5 patients with a purely regurgitant anatom-

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ically abnormal tricuspid valve (rheumatic involvement), this pressure was 40 mm Hg or less. In eight of the nine with a purely regurgitant anatomically normal tricuspid valve, it was 55 mm Hg or more. Thus, in 12 (86%) of 14 patients tricuspid valve morphology was correctly predicted by use of systolic pulmonary artery pressure. In the two remaining patients with a systolic pulmonary artery pressure of 41 to 54 mm Hg, one tricuspid valve had abnormal leaflets (rheumatic) with normal anular circumference (11.0 cm) and the remaining valve had anatomically normal leaflets with a dilated anular circumference (14.0 cm). *Thus, the combination of systolic pulmonary artery pressure and anular circumference was useful in separating all 14 purely regurgitant tricuspid valves into anatomically normal or abnormal groups.*

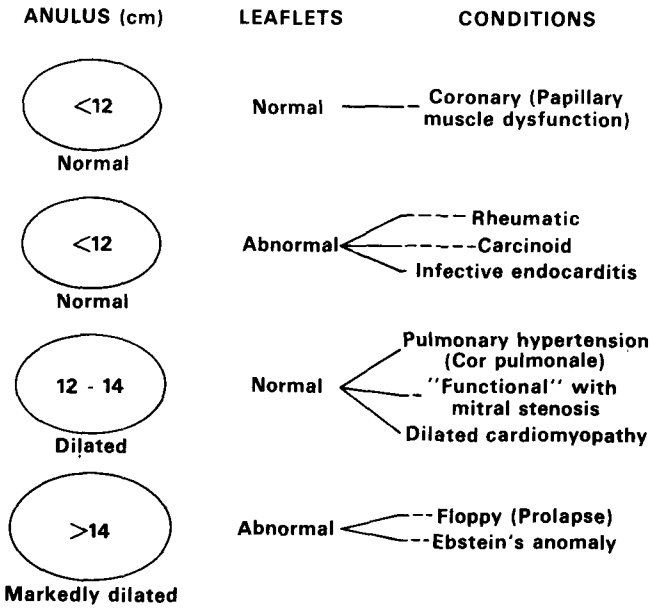

Figure 11. Diagram showing usefulness of anular circumference in determining etiology of pure tricuspid regurgitation.

Discussion

Pure tricuspid valve regurgitation may result from structural alterations of one or all of the components of the tricuspid valve apparatus (leaflets, chordae tendineae, papillary muscles, adjacent right ventricular myocardium) or from abnormal function of a structurally normal valve (dilated anulus). Pure tricuspid regurgitation can be classified as primary ("organic") when it is due to intrinsic abnormality of the valve apparatus, or secondary ("functional") when it is due to right ventricular pressure or volume overload. Although some authors (4,9) have indicated that from a clinical viewpoint the "organic-functional" classification of tricuspid regurgitation is untenable, from a morphologic and etiologic standpoint it is still very useful (8). In the present report of 45 necropsy patients with clinical and morphologic evidence of pure tricuspid regurgitation, 24 (53%) had an anatomically abnormal tricuspid valve from various causes and the remaining 21 (47%) patients had an anatomically normal tricuspid valve with a dilated anulus secondary to various causes of systolic pulmonary artery hypertension.

Although it has been generally accepted that functional tricuspid regurgitation from pulmonary hypertension and right ventricular failure is caused by tricuspid valve anular dilation, few necropsy data are available to support this belief or compare anular sizes in pure tricuspid regurgitation of various causes. Several echocardiographic studies are available that provide data in experimental models (10) and humans (11–14) for the role of the tricuspid anulus in the production of functional tricuspid regurgitation, but only one study (11) makes reference to anular measurements at necropsy.

Morphometric analysis. In the present study, anular circumference was a useful measurement to separate purely regurgitant tricuspid valves into three etiologic groups: 1) normal anulus (< 12 cm) (rheumatic disease, coronary papillary muscle dysfunction, carcinoid tumor or infective endocarditis); 2) slightly dilated anulus (12 to 14 cm) (various causes of pulmonary hypertension and dilated cardiomyopathy); and 3) markedly dilated anulus (> 14 cm) (Ebstein's anomaly or floppy valve) (Fig. 11). These morphometric data support the concept that tricuspid valve anular dilation is the major mechanism for the development of functional tricuspid regurgitation with right ventricular cavity dilation from pulmonary hypertension. It has been suggested (13,14) that anterior displacement of the anatomically normal tricuspid leaflets (malaligned coaptation) may also play a role in the development of functional tricuspid regurgitation.

Tei et al. (11) reported the first echocardiographic measurements of tricuspid valve anulus by using a new two-dimensional anular reconstruction method in 18 patients with clinical tricuspid regurgitation and 28 control subjects. The cause of tricuspid regurgitation was pulmonary hypertension (five patients), dilated cardiomyopathy (five patients), rheumatic valve disease (four patients) and previous myocardial infarction (four patients). In addition, tricuspid anular measurements of 18 necropsy control hearts in fresh and formalin-fixed states were obtained. Comparison of echocardiographic measurements in control subjects corresponded most closely with the necropsy formalin-fixed anular measurements. Echocardiographic measurements of the tricuspid anulus were increased in all patients with tricuspid regurgitation, but breakdown of anular measurements into etiologic subgroups was not provided.

Come and Riley (14) evaluated tricuspid "anular diameter" by two-dimensional echocardiography in 11 pa-

tients with clinical tricuspid regurgitation and 15 control subjects. Anular diameter was increased in patients with tricuspid regurgitation compared with that in control subjects. Their report provided evidence that patients with a purely regurgitant tricuspid valve due to floppy valve disease and Marfan's syndrome have a dilated "anular diameter" compared with that of control subjects. In our necropsy study, seven patients with a floppy tricuspid valve (without Marfan's disease) had a markedly dilated anular circumference ranging from 14 to 17.5 cm.

Measurement of tricuspid leaflet areas was also useful in separating etiologic subgroups of pure tricuspid regurgitation. The leaflet area was significantly greater in patients with a floppy tricuspid valve and Ebstein's anomaly than in the remaining patients with other conditions causing pure tricuspid regurgitation and the necropsy control subjects. Furthermore, the leaflet area helped to separate floppy from Ebstein's tricuspid valves. Valves with Ebstein's anomaly had an average leaflet area 1.2 times larger than that of floppy valves. Although our study is the first to provide data on leaflet areas in purely regurgitant floppy tricuspid valves, similar increases in leaflet areas of purely regurgitant floppy mitral valves compared with other conditions producing pure mitral regurgitation were reported by Waller et al. (15). The increased leaflet area in both tricuspid and mitral valves corresponds to leaflet "hooding" and "redundancy" described in the floppy valve syndrome.

Pulmonary artery pressure and valve morphology. Controversy surrounds the use of pulmonary artery pressure measurements in predicting the morphologic status (normal or abnormal) of purely regurgitant tricuspid valves, particularly in the setting of rheumatic mitral stenosis (4,9,16–22). Hemodynamic-morphologic correlations in the present study indicate that systolic pulmonary artery pressure generally predicts the morphologic status of the tricuspid leaflets. Of 14 necropsy patients with a systolic pulmonary artery pressure of 55 mm Hg or more, 13 had anatomically normal leaflets with a dilated anular circumference ("functional" regurgitation). Six patients had mild elevation of systolic pulmonary artery pressure (41 to 54 mm Hg); of these six, four had anatomically normal and two had abnormal leaflets. Each of the four normal tricuspid valves had an anular circumference greater than 12 cm and the two abnormal valves had an anular circumference of 12 cm or less. Thus, the combination of elevated systolic pulmonary artery pressure (> 40 mm Hg) and dilated tricuspid valve anular circumference (> 12 cm) accurately predicted normal tricuspid leaflets in 19 (95%) of these 20 patients with functional tricuspid regurgitation.

In the subgroup of 10 necropsy patients with pure tricuspid regurgitation and mitral stenosis, systolic pulmonary artery pressure of 55 mm Hg or greater was always associated with anatomically normal tricuspid leaflets and a pressure of 40 mm Hg or less was always associated with anatomically abnormal (rheumatic involvement) leaflets. Two patients had a pulmonary pressure of 41 to 54 mm Hg; one valve was normal and one abnormal. The anatomically normal valve had an anular circumference of 14 cm (dilated) and the abnormal valve had a circumference of 11 cm (normal).

Previous studies. Previous clinical reports (16–22) have suggested a correlation between functional versus organic tricuspid regurgitation and various hemodynamic measurements of the right side of the heart. Sepulveda and Lukas (16) found higher pulmonary vascular resistance, mean right atrial pressure and right ventricular pressure in patients with rheumatic mitral valve disease and secondary ("functional") tricuspid regurgitation compared with patients with rheumatic mitral disease and no tricuspid regurgitation. Salazar and Levine (17) suggested that when tricuspid regurgitation was associated with a moderate elevation (that is, < 60 mm Hg) of pulmonary artery pressure it was likely that the tricuspid lesion was organic, whereas all instances of functional tricuspid regurgitation were associated with severe (that is, > 60 mm Hg) pulmonary artery hypertension. Hansing and Rowe (18) correlated hemodynamic data from 28 patients with tricuspid regurgitation and found that certain right-sided measurements (pulmonary arteriolar and total pulmonary resistance, pulmonary artery mean, right ventricular systolic and end-diastolic pressures) were related to the production of tricuspid regurgitation. Lichtlen and Simon (19) and Schwarz et al. (20) also found higher right atrial and systolic and end-systolic right ventricular pressures in patients with secondary ("functional") forms of rheumatic tricuspid regurgitation. Braunwald (21) suggested that in patients with rheumatic tricuspid regurgitation a pulmonary artery systolic pressure greater than 60 mm Hg favored a secondary or "functional" cause of tricuspid regurgitation, whereas a pulmonary artery systolic pressure less than 40 mm Hg favored primary or "organic" tricuspid valve disease. Although these previous studies support our observations correlating systolic pulmonary artery pressures with tricuspid valve morphology, each lacks morphologic (necropsy or surgical) confirmation and morphometric analysis.

Recently, an echocardiographic-hemodynamic study by Daniels et al. (22) compared patients with rheumatic tricuspid regurgitation with patients having functional tricuspid regurgitation. Systolic pulmonary artery pressure, mean pulmonary artery pressure and pulmonary vascular resistance did not distinguish organic from functional lesions. Their study, however, also lacked surgical or necropsy confirmation of tricuspid valve morphology.

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