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KAKU, KOHZU

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CLINICAL SEVERITY OF TRICUSPID VALVE REGURGITATION AS GRADED BY CONTRAST ECHOCARDIOGRAPHY

KOHZU KAKU

1st Department of Internal Medicine
Kobe University School of Medicine

INDEXING WORDS

tricuspid valve regurgitation; contrast echocardiography; tricuspid annular diameter

SYNOPSIS

In order to evaluate the severity of tricuspid regurgitation (TR) by a convenient and non-invasive method, contrast echocardiograms utilizing peripheral vein injection of saline were performed in 75 patients consisting of 59 with mitral valvular disease, 12 with atrial septal defects (ASD-II), and 4 with primary pulmonary hypertension (PPH). Since previous classifications, evaluating only the flow patterns of M-mode contrast echocardiograms at the tricuspid orifice, could not provide sufficient information about TR severity, a new criterion evaluating both the regurgitant flow patterns and flow amount across the tricuspid valve, has been used to classify the severity of TR by M-mode contrast echocardiography. With this method, we were able to obtain a favorable result which not only proved to be closely related to angiocardiographic grading ($\rho=0.91$, $p<0.01$), but also superior to the other contrast echocardiographic methods and Doppler sonography. Based on this contrast grading, the maximum and minimum diameters of tricuspid annulus in each grade were measured from two dimensional echocardiograms. These annular measurements showed a

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progressive dilatation of tricuspid annulus and a progressive reduction of annular fractional shortening as TR deteriorated from mild to severe. Hemodynamic changes associated with TR included a higher pulmonary arterial pressure (PAP) in the TR group (48.2 mmHg) than in the non-TR group (32.6 mmHg). The PAPs in cases with sinus rhythm were significantly higher than those with atrial fibrillation (AF) in each corresponding grade of TR. Significant elevation of right atrial pressure (RAP) was observed only in severe TR (TR-III & TR-IV).

In conclusion, the above results elucidate that contrast echocardiography based on the new criterion is a convenient and reliable method for evaluating the severity of TR, and that the mechanisms of TR are multifarious and associated with various factors such as PAP, cardiac rhythm, annular diameter and annular fractional shortening.

INTRODUCTION

The majority of patients with tricuspid valve problems are overlooked unless careful physical and laboratory examinations are carried out. A number of clinical techniques have been employed to evaluate tricuspid valve regurgitation such as the dye indicator method,[7, 30] intracardiac phonocardiography,[6, 35] angiocardiography,[2, 11] contrast echocardiography,[1, 19, 31] and Doppler sonography,[33, 34] etc. Among them, angiocardiography is generally accepted as one of the most reliable methods for evaluating the severity of TR. However, the use of angiocardiography in clinical cases remains limited because it is an invasive method available only in fully-equipped hospitals. It can not be performed on out-patients and in addition, repeated follow-up studies are almost impossible. On the contrary, contrast echocardiography and Doppler sonography are non-invasive techniques and repeated follow-up examinations can be carried out easily even on out-patients. In terms of these techniques, there are problems which remain unsolved, i.e., the difficulty in evaluating the severity of TR with reliable criteria. In the present study, we attempted to characterize the clinical features of various degrees of TR and to establish convenient and useful criteria for TR grading by contrast echocardiography.

MATERIALS AND METHODS

The subjects studied were 75 patients (45 females and 30 males) who were admitted to the First Department of Internal Medicine of Kobe University Hospital for detailed cardiac examinations during the period between January 1981 and November 1982. Of the 75 cases studied, 59 had mitral valvular disease, 12 had atrial septal defects, and 4 had primary pulmonary hypertension. The ages of patients ranged from 17 to 78 year with a mean of 45.6 years.

Echocardiography was performed using a Toshiba phased-array sector scanner SSH-11A with a 2.4 MHz probe. The M-mode output was recorded by a Honeywell line scan recorder Model FR-06A. A Sony video-display system was employed in recording B-mode echocardiograms which allowed repeated examinations of the original real time images. Hard copy was obtained by still-frame photography using a Polaroid camera attachment.

A standard echocardiographic procedure was used throughout the study. The heart was visualized from both the precordial and subcostal echocardiographic windows and the following standard echocardiographic planes were scanned and recorded (Fig. 1), i.e., apical four chamber view (4CV), long axis view of right ventricular inflow (RIF), subcostal four chamber view, and left ventricular long axis and short axis views. In order to identify the tricuspid annulus exactly, special attention was paid to obtaining a clear-cut image of the attachment points of the tricuspid valves to the annulus when the apical 4 chamber and RIF views were taken. The distance between the attachment points of the septal leaflet and the anterior leaflet in apical 4 chamber view, or the anterior leaflet and the posterior leaflet in RIF view, serve as the echocardiographic tricuspid annular diameter (TAD). The diastolic maximum annular diameter was measured at the onset of the Q wave and the systolic minimum annular diameter at mid-systole.[29] Each measurement represented a mean value from 4 to 5 cardiac cycles. The percent fractional shortening of annular diameter (%FS of TAD) was calculated by the following formula: (maximum diameter - minimum diameter) ÷ maximum diameter x 100. Doppler sonographic mapping of right atrium (RA), using a Toshiba Echo Doppler Unit SDS-10A, was performed during the standard echocardiographic examination. The results of Doppler mapping

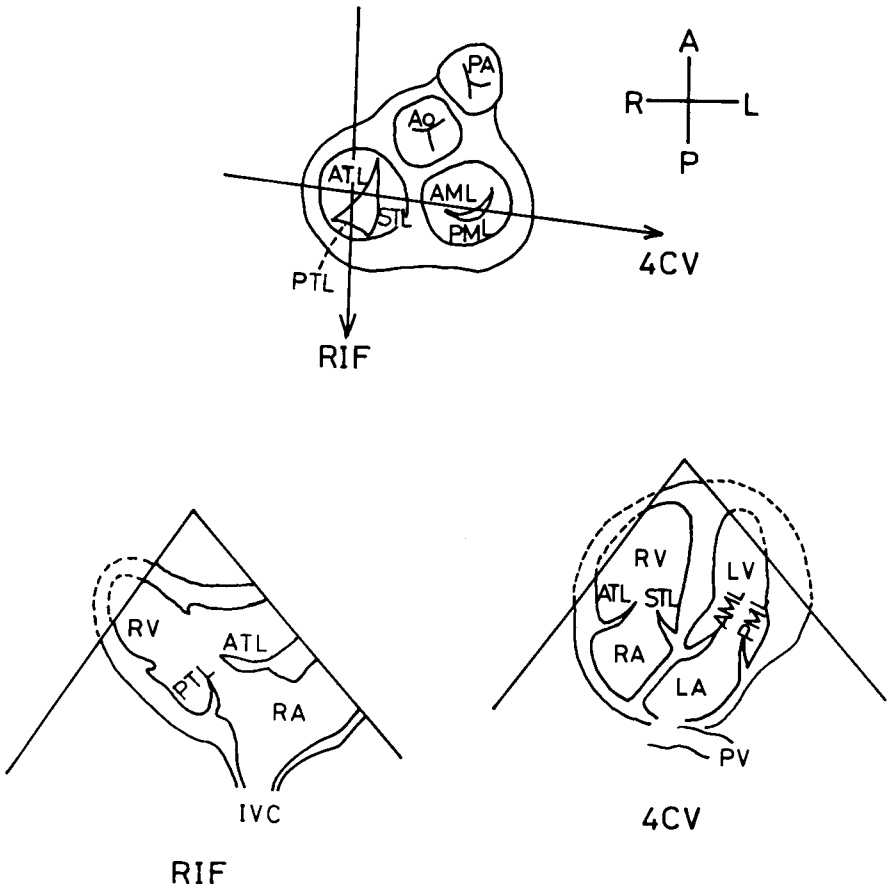

Fig. 1 Schematic drawing of echocardiographic planes utilized for the recording of the tricuspid annulus. Upper panel shows the section of the heart at the level of the tricuspid and mitral ring seen from the apex. Arrows indicate the two-dimensional echo beams, by which apical four chamber view (4CV) and long axis view of RV inflow (RIF) can be recorded.
PA : Pulmonary artery. Ao : Aorta. ATL : Anterior tricuspid leaflet. PTL : Posterior tricuspid leaflet. STL : Septal tricuspid leaflet. RV : Right ventricle. LV : Left ventricle. RA : Right atrium. LA : Left atrium. IVC : Inferior vena cava. PV : Pulmonary vein. AML : Anterior mitral leaflet. PML : Posterior mitral leaflet.

were classified into 5 grades, no systolic turbulent signal is detected in Gr-0; systolic turbulent signals (STS) are detected within 1.4 cm from the tricuspid valve in Gr-I; STS are detected within 2.9 cm from tricuspid valve in Gr-II; STS are detected within 4.4 cm from tricuspid valve in Gr-III; and STS are detected in whole right atrium in Gr-IV.[18] Contrast echocardiography was performed after a rapid injection of 6 ml saline into the patient's precubital vein. The injection was repeated several times in order to get satisfactory contrast echocardiograms both at tricuspid orifice and inferior vena cava (IVC). The results of M-mode tricuspid contrast echocardiography were divided into 5 grades, no regurgitation contrast flow (RCF) is observed during systole in TR-0; non-pansystolic RCF is observed in RA in TR-I; pansystolic RCF is observed in RA but is not larger than the forward contrast flow (FCF) in TR-II; pan-systolic RCF in RA is larger than the FCF in TR-III (A); pan-systolic RCF is not larger than FCF but is present in both RA and right ventricle (RV) in TR-III (B); and pan-systolic RCF is larger than FCF and is observed both in RA and RV in TR-IV (Fig. 2).

Cardiac catheterization and angiocardiography were performed in all patients within 7 days before or after the contrast echocardiographic examination. The degree of TR was classified by the grading proposed by Grossman[11]: 1+, regurgitation contrast material essentially clears with each beat and never opacifies the entire atrium; 2+, regurgitation contrast does not clear with 1 beat and generally opacifies the entire atrium after several beats, but the degree of opacification does not equal that of the ventricle; 3+, regurgitant dye completely opacifies the atrium and equals opacification with the ventricle; and 4+, opacification of the atrium occurs in 1 beat and becomes progressively denser with each beat.

Statistical analysis was performed using Student's unpaired t test unless otherwise stated.

RESULTS

Comparison between TR gradings by angiocardiography and contrast echocardiography using the new criteria for M-mode contrast echocardiograms

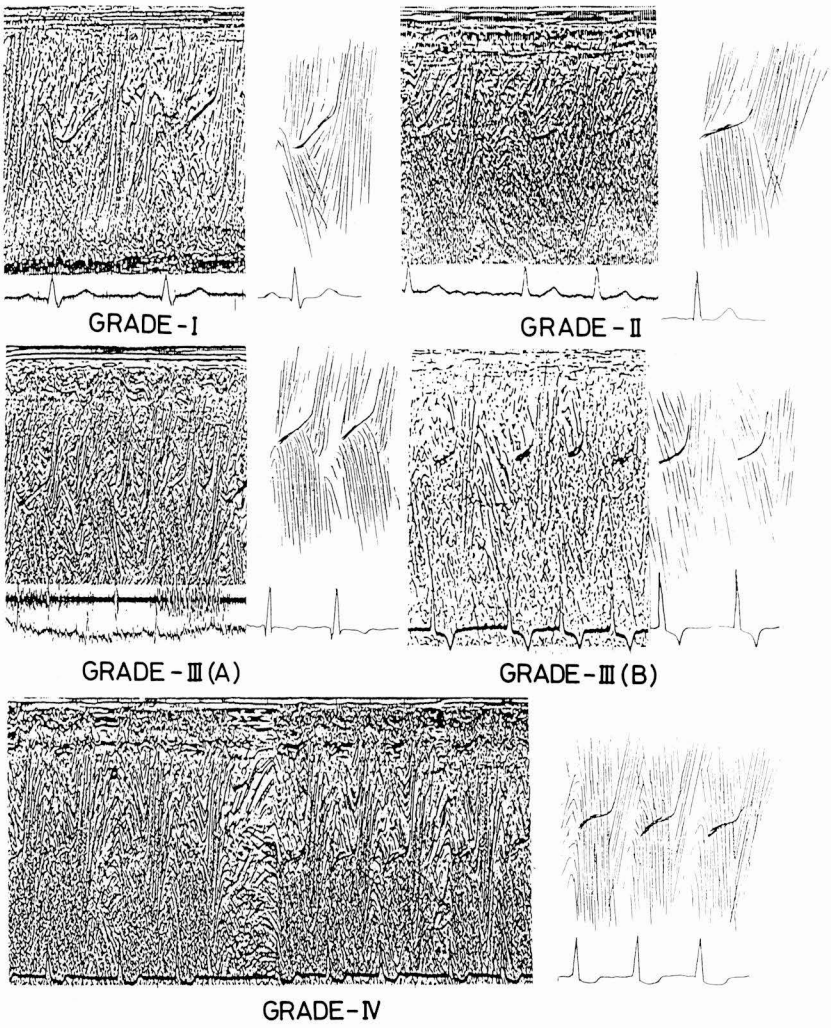

Fig. 2 The M-mode echocardiograms and illustrative drawings of each TR grade based on the new contrast classification. Gr-I : Non-pansystolic regurgitation contrast flow (RCF) is observed during systole. Gr-II : Pansystolic RCF is observed in RA but is not larger than the forward contrast flow (FCF). Gr-III (A) : Pansystolic RCF is larger than the FCF. Gr-III (B) : Pansystolic RCF is present in both RA and RV. Gr-IV : Pansystolic RCF is observed both in RA and RV and is larger than the FCF.

The numbers of patients in each TR-grade for echocardiography were as follows: TR-0, 16 cases; TR-I, 13 cases; TR-II, 25 cases; TR-III, 13 cases; and TR-IV, 8 cases. The numbers of patients in each TR grade for angiocardiography were as follows: 0, 14 cases; 1+, 17 cases; 2+, 21 cases; 3+, 16 cases; and 4+, 7 cases. The relation between these two methods is shown in Fig. 3 ($\rho = 0.91$, $p < 0.01$, Spearman rank correlation). Fifty-two patients (69%) had the same TR grades by contrast echocardiography and by angiocardiography. Twenty-three patients (31%) had a one grade difference between the two methods. Fig. 4 depicts the relation between Doppler TR grading and angiographic TR grading with $\rho = 0.79$ ($p < 0.01$, Spearman rank correlation). Thirty-six patients (48%) had the same grades both by Doppler and angiocardiographic methods, 34 patients (45%) had a one grade difference, and 5 patients (7%) had a two grade difference. Both contrast echocardiographic and Doppler sonographic TR gradings showed a significant relationship to the angiocardiographic TR grading, but contrast echocardiography had a better relation to angiocardiography than Doppler sonography to angiocardiography. Another way of classifying the M-mode contrast echocardiograms, which evaluated only the contrast flow patterns by the presence of regurgitant contrast echo in both RV and RA (AB type) or in RA only (B type), was also made in the 75 cases (N indicated no regurgitation). The relationship of this classification and the angiocardiographic grading is shown in Fig. 5. Seven cases of grade 4+ and six of grade 3+ belonged to the AB type. Ten cases of grade 3+, 21 of grade 2+ and 13 of grade 1+ belonged to B type. It appeared that the AB type was associated with moderate to severe TR and the B type with mild to moderate TR. But considerable overlapping of the same type in different grades was also observed.

Tricuspid annular diameters by echocardiogram (Fig. 6)

The diastolic maximum diameters and the systolic minimum diameters of tricuspid annulus in each grade from TR-0 to TR-IV were: 29.3 ± 2.2 mm & 22.0 ± 1.7 mm; 30.9 ± 2.0 mm & 24.8 ± 1.7 mm; 32.1 ± 2.0 mm & 27.1 ± 2.1 mm; 35.7 ± 2.8 mm & 30.7 ± 3.0 mm; and 37.5 ± 4.7 mm & 34.5 ± 5.7 mm, respectively. Positive increments of TAD from TR-0 to TR-IV were recognized both on the maximum

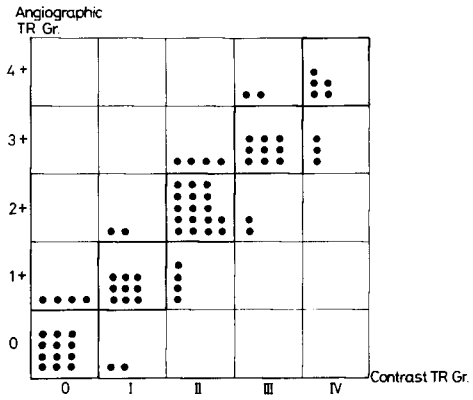

Fig. 3
Relationship between contrast TR grades and angiographic TR grades in 75 patients.

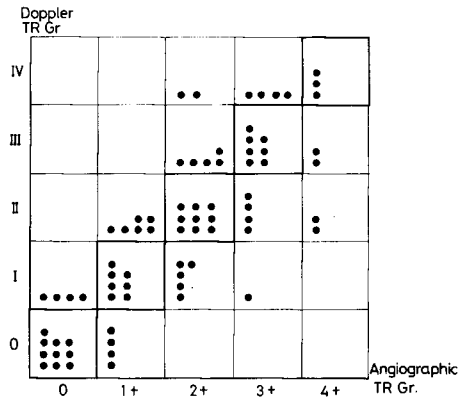

Fig. 4
Relationship between Doppler TR grades and angiographic TR grades in 75 cases.

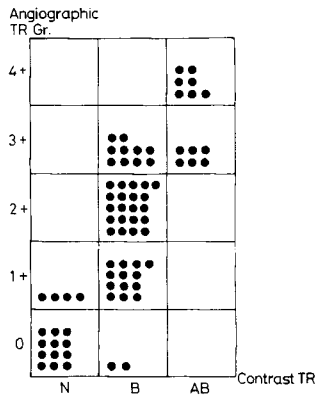

Fig. 5
Relationship between angiocardiographic TR grading and ABN contrast TR grading (For details see text).

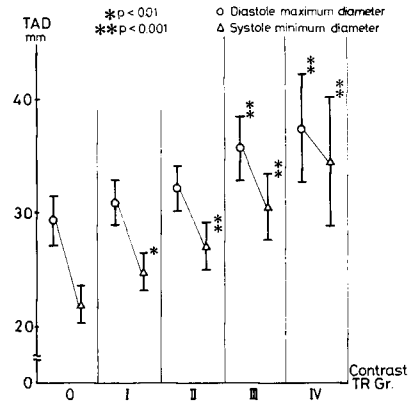

Fig. 6.
The diastolic maximum and systolic minimum diameters (expressed by mean ± SD) of tricuspid annulus in each grade of contrast TR.
TAD : Tricuspid annulus diameter.

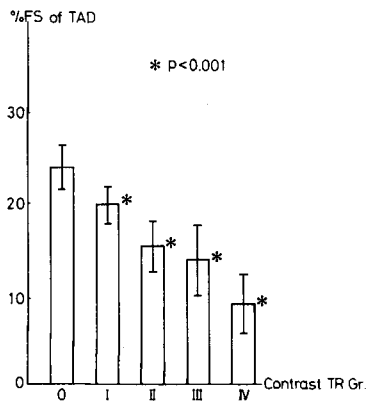

Fig. 7
The percent fraction shortening of tricuspid annulus (%FS of TAD) in each grade of contrast echocardiographic TR (expressed by mean ± SD).

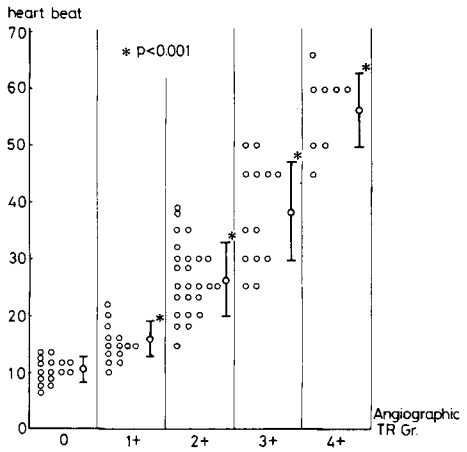

Fig. 8
Relationship between each grade of angiocardiographic TR and the contrast echo disappearance time, expressed by number of heart beats.

diameters and the minimum diameters, but were much more prominent on the minimum diameters. The maximum diameters of TR-I and TR-II were not significantly larger than that of TR-0. The minimum diameters of TR-III and TR-IV were greater than the maximum diameters of TR-0. The mean %FS of TAD in each grade from TR-0 to TR-IV were: 23.8 ± 2.4%; 19.8 ± 2.0%; 15.7 ± 2.8%; 14.0 ± 3.9%; and 8.0 ± 3.5%, respectively (fig. 7). These values indicated a progressive reduction of annular contraction from TR-I to TR-IV. The disappearance time of contrast echo at tricuspid orifice was expressed by the number of heart beats, and the mean values for each grade from TR-0 to TR-IV were; 10.6 ± 2.2; 15.5 ± 3.4; 26.4 ± 6.3; 37.7 ± 9.2; and 56.4 ± 7.1, respectively (Fig. 8). Significant progressive delayed disappearance of contrast echo was observed in each grade from TR-I to TR-IV, but overlapping of the disappearance time between each grade was also noted to some extent.

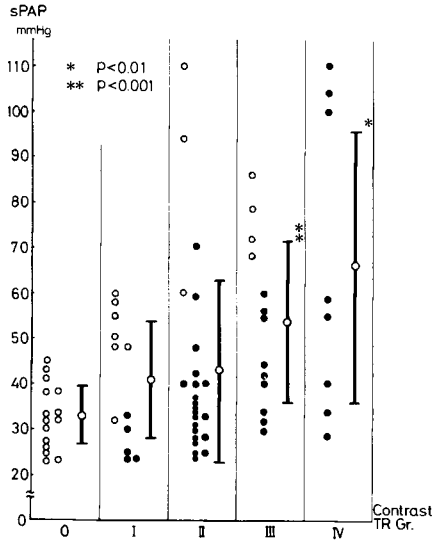

Fig. 9
Systolic pulmonary arterial pressures of 75 patients in each grade of contrast TR.
sPAP : Systolic pulmonary arterial pressure.
Open circles represent sinus cases and closed circles atrial fibrillation cases.

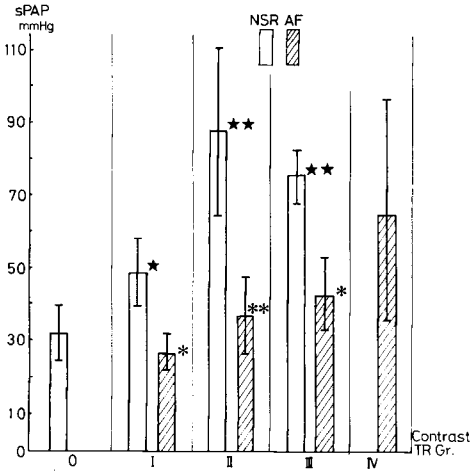

Fig. 10.
Systolic pulmonary arterial pressures (expressed by mean ± SD) of sinus and AF cases in each grade of contrast echocardiographic TR.
sPAP : Systolic pulmonary arterial pressure. NSR : Normal sinus rhythm. AF : Atrial fibrillation.
★ Compared to non-TR group, $p<0.01$
★★ Compared to non-TR group, $p<0.001$
* Compared to NSR group of the same TR grade, $p<0.01$
** Compared to NSR group of the same TR grade, $p<0.001$

Hemodynamic variables

1. Pulmonary arterial pressure (PAP)

The mean systolic PAPs of non-TR group and TR groups were 32.6 ± 7.2 mmHg and 48.2 ± 24.1 mmHg, respectively. As shown in Fig. 9, the PAPs of TR-III & TR-IV were significantly higher than those of TR-0, while no significant difference was found between the PAPs of TR-I & TR-II and of TR-0. The overlapping of PAPs between each TR grade was rather prominent. The mean values of

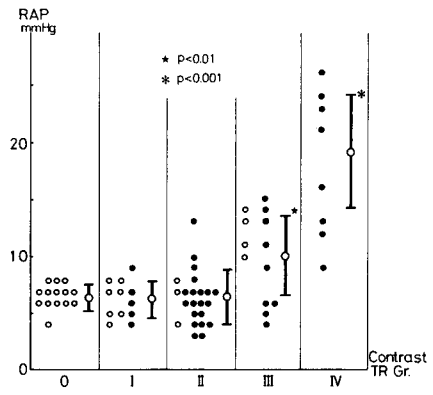

Fig. 11
Right atrial pressures (V-wave) of 75 cases in each grade of contrast echocardiographic TR.
RAP : Right atrial pressure.

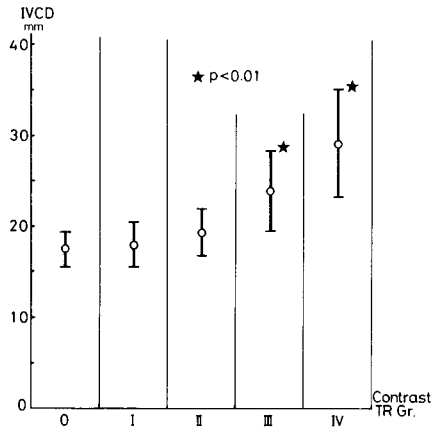

Fig. 12
Echocardiographic diameters of inferior vena cava (IVCD) in each grade of contrast TR (expressed by mean ± SD).

PAPs in each grade were: TR-0, 32.6 ± 7.1 mmHg; TR-I, 40.6 ± 13.9 mmHg; TR-II, 42.9 ± 21.7 mmHg; TR-III, 53.7 ± 18.6 mmHg; and TR-IV, 66.3 ± 33.7 mmHg. However, when cases of sinus rhythm were separated from AF cases, the PAPs of sinus cases were significantly higher than those of AF cases in the same group. As shown in Fig. 10, the PAPs of sinus cases in each grade were: TR-0, 32.6 ± 7.2 mmHg; TR-I, 50.1 ± 9.3 mmHg; TR-II, 88.0 ± 22.5 mmHg; TR-III, 76.5 ± 7.6 mmHg; and the PAPs of AF cases in each grade were: TR-I, 27.2 ± 4.1 mmHg; TR-II, 36.4 ± 11.4 mmHg; TR-III, 43.5 ± 11.1 mmHg; and TR-IV, 66.2 ± 33.7 mmHg. The PAPs of AF cases in TR-I, TR-II and TR-III were not significantly higher than the PAPs in TR-0, while the PAPs of sinus cases in TR-I, TR-II and TR-III were significantly higher than those in TR-0.

2. Right atrial pressure (RAP) and diameter of inferior vena cava (IVCD)

As shown in Fig. 11, the mean values of RAP (V-wave) in each grade from TR-0 to TR-IV were 6.4 ± 1.1 mmHg, 6.3 ± 1.6 mmHg, 6.4 ± 2.3 mmHg, 10.1 ± 3.7 mmHg and 19.2 ± 5.5 mmHg, respectively. No case in TR-0 or TR-I had a RAP higher than 10 mmHg and there was only one case in TR-II with a RAP higher than 10 mmHg. The

RAPs in 62% of TR-III cases and 88% of TR-IV cases were higher than 10 mmHg. The RAPs were not significantly elevated in TR-I and TR-II, while significant elevation of RAPs was observed in TR-III and TR-IV. This backward load of TR was seen to be reflected in the IVCD (TR-III & TR-IV). The mean IVCDs in each grade from TR-0 to TR-IV were 17.6 ± 2.0 mm, 17.9 ± 2.5 mm, 19.3 ±2.6 mm, 23.9 ± 4.4 mm, and 29.2 ± 6.0 mm, respectively (Fig. 12). The mean IVCDs of TR-I and TR-II were not larger than 20 mm and were not significantly larger than that of TR-0. A significant increase in IVCDs was observed only in TR-III and TR-IV.

DISCUSSION

The M-mode patterns of contrast echocardiograms at the tricuspid orifice have been studied by many investigators.[1, 12, 22, 31] Nakamura et al.[20] classified the patterns of contrast echocardiograms into Type I (with early systolic forward flow) and Type II (without early systolic forward flow) and they concluded that Type II was possibly associated with severe TR. Kuwako et al.[15] classified the echo patterns into AB, B, and N types according to the presence or absence of regurgitant echo in RA and/or RV. But our results with these criteria were not necessarily encouraging. Amano et al.[1] indicated that the duration of contrast echo observed in IVC was a better index for detecting TR. In the present study, we noted that the appearance of contrast echo in IVC was not a constant finding in TR especially when the degree of TR was mild. However, we did observe a parallel relationship between the duration of IVC contrast echo and the severity of TR, but overlapping between each grade was great. The duration of contrast echo in IVC is apparently influenced not only by TR, but also by cardiac rhythm and heart failure etc. The diameter of IVC might serve as an indicator of severity of TR.[1] When the diameter of IVC is greater than 22 mm, the presence of TR should be suspected, while the diameters of IVC in mild TR are usually not dilated (below 20 mm). As stated previously, classifying the M-mode contrast echocardiograms solely on the regurgitant flow patterns is often inadequate for proper evaluation of TR severity. It is necessary to take the regurgitant flow patterns and the flow amount at the tricuspid

orifice into consideration if proper TR grading is desired. With our new classification, i.e., no RCF is observed in TR-0, non-pansystolic RCF is observed in RA in TR-I, pan-systolic RCF is observed in RA but is not larger than FCF in TR-II, pan-systolic RCF in RA is larger than FCF in TR-III (A), pan-systolic RCF is not larger than FCF but is present in both RA and RV in TR-III (B), and pan-systolic RCF is observed in both RA and RV and is larger than FCF in TR-IV, we were able to obtain very satisfactory results for contrast TR grading as compared with the angiocardiographic TR gradings. The Doppler mapping of RA was also useful in evaluating the severity of TR but it appeared inferior to the contrast method in our study, and there were several reasons for this. Firstly, some of our cases had a giant left atrium which displaced RA and interfered with adequate RA mapping. Secondly, when a large RA was present, Doppler sonographic interference in the posterior area of RA was usually great enough to obscure the detection of turbulent flow.

The annular dilatation in functional TR has been reported in many surgical and autopsy studies and generally accepted as one of the mechanisms of TR. In this study, we also observed a progressive annular dilatation with simultaneous reduction of %FS of TAD in parallel with TR grades from I to IV. Since groups with severe TR (TR-III & TR-IV) had mean minimum diameters greater than 30 mm, which was the mean maximum diameter of the non-TR gourp, a minimum diameter over this value might suggest the existence of moderate to severe TR. The %FS of TAD may play as important a role as annular dilatation does in TR. Tsukiris et al.[29] demonstrated in their dog experiments the role of phasic shortening of the tricuspid annulus in normal valve closure. Tei et al.[28] and others[12, 32] associated TR with both annular dilatation and reduced %FS. These reports are consistent with what we observed in this study.

The relationship between the severity of TR and hemodynamic changes has not been clear. Kawashima et al.[14] demonstrated a significant difference of RAP between TR and non-TR groups. But Cairns et al.[4] and others[7, 25] failed to find such a difference. Tamaki[27] stated that TR was definitely associated with a RAP higher than 14 mmHg. We believe that in cases of mild TR, RAP is not necessarily affected by regurgitant flow and accordingly it can not be used to predict the presence of TR. However, when RAP

reaches a critical level (>10 mmHg), TR of more than moderate degree is usually present.

The relationship between PAP and functional TR is also controversial. Salazar et al.[23] pointed out that an elevated PAP might be related to functional TR. On the other hand, Daniels et al.[8] failed to demonstrate a close relationship between functional TR and hemodynamic variables. Although our data disclosed a significant elevation of mean PAP in TR groups, in many cases a normal PAP could not rule out the possibility of TR. However, we observed that the PAP in patients with normal sinus rhythm was significantly higher than that in cases with AF in each corresponding group. In fact, only one case of sinus rhythm with TR had a PAP lower than 45 mmHg, while most AF cases with TR (74.4%) had PAPs lower than that level. This finding suggests that atrial fibrillation may facilitate the occurrence of tricuspid regurgitation under relatively low pressure loads. It was recognized by Salazar et al.[23] that in atrial fibrillation, normal atrial relaxation might be absent and consequently atrioventricular valve closure incomplete. This incomplete valve closure in AF may be associated not only with poor apposition of valves but also inadequate contraction of atrioventricular ring and ventricle. In other words, the altered tricuspid apparatus in cases with AF facilitates the occurrence of TR.

It is reasonable to conclude that tricuspid valve regurgitation may be produced not only by an elevated PAP (greater than 45 mmHg), but also by a number of other factors such as cardiac rhythm, annular diameter and contraction, valve apposition and ventricular contraction. In addition to the other conventional clinical examinations, contrast echocardiography according to the above mentioned criteria can provide a reliable and convenient method for the evaluation of TR severity.

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REFERENCES

1. Amano, K., Sakamoto, T., Yamaguchi, T., Ishimitsu, T., Hada, Y., and Adachi, H. J. Cardiography 1979. 9. 697/716. Contrast echocardiography: Application for valvular incompetence.
2. Baron, M.G. Circulation 1971. 43. 599/605. Angiocardiographic evaluation of valvular insufficiency.
3. Breyer, R.H., McClenathan, J.H., Michaelis, L.L., McIntosh, C.L., and Morrow, A.G. J. Thoracic & Cardiovasc. Surg. 1976. 72. 868/874. Tricuspid regurgitation, a comparison of nonoperative management, tricuspid annuloplasty, and tricuspid valve replacement.
4. Cairns, K.B., Kloster, F.E., Bristow, J.D., Lees, M.H., and Griswold, H.E. Am. Heart J. 1968. 75. 173/179. Problems in the hemodynamic diagnosis of tricuspid insufficiency.
5. Carpentier, A., Deloche, A., Hanania, G., Sellier, Ph., Piwnica, A., Dubost, Ch., and McGoon, D.C. J. Thoracic & Cardiovasc. Surg. 1974. 67. 53/65. Surgical management of acquired tricuspid valve disease.
6. Cha, S.D., Gooch, A.S., and Maranhao, V. Am. J. Cardiol. 1981. 48. 578/583. Intracardiac phonocardiography in tricuspid regurgitation: Relation to clinical and angiographic findings.
7. Collins, N.P., Braunwald, E., and Morrow, A.G. Circulation 1959. 20. 561/568. Detection of pulmonic and tricuspid valvular regurgitation by means of indicator solutions.
8. Daniels, S.J., Mints, G.S., and Kotler, M.N. Am. J. Cardiol. 1983. 51. 492/496. Rheumatic tricuspid valve disease : Two-dimensional echocardiographic, hemodynamic, and angiographic correlations.
9. Eckner, F.A., Brown, B.W., Davidson, D.L., and Glagov, S. Arch. Pathol. 1969. 88. 497/507. Dimensions of normal heart.
10. Gould, L., Schaffer, A.I., and O'Connor, R.A. Am. J. Cardiol. 1965. 16. 189/194. Does atrial fibrillation lead to tricuspid insufficiency?
11. Grossman, W., and Dexter, L. In : Grossman, W., ed., Cardiac Catheterization and Angiography, 2nd ed., (Lee & Febiger, Philadelphia). 1980. 305/335. Profiles in valvular disease.
12. Jinbo, M., Sugimoto, T., Uraoka, T., Terada, Y., Yokawa, S.,

Iuchi, K., Akiyama, M., and Kanaki, E. J. Cardiography 1981. 11. 457/466. Relationship between vascular diameter and intracardiac pressure in tricuspid or pulmonary insufficiency detected by contrast echocardiography.

13. Kato, H., Kudo, M., Tsuji, K., Yamaoka, S., Yoshida, K., Takagi Y., Okumachi, F., Yanagihara, K., Owaki, T., and Yoshikawa, J. J. Cardiography 1981. 11. 209/215. Tricuspid valve motion and tricuspid valve ring size in normals and patients with atrial septal defect.
14. Kawashima, Y., Nakano, S., and Manabe, H. Jpn. Circ. J. 1974. 38. 853/859. Tricuspid insufficiency : A study of hemodynamics.
15. Kuwako, K., Tohda, E., Ino, T., Umeda, T., Furuta, S., and Machii, K. J. Cardiography 1980. 10. 947/966. Echocardiographic evaluation of the tricuspid valve and ring : Diagnosis of tricuspid stenosis and regurgitation.
16. Lieppe, W., Behar, V.S., Scallion, R., and Kisslo, J.K. Circulation 1978. 57. 128/132. Detection of tricuspid regurgitation with two-dimensional echocardiography and peripheral vein injections.
17. Meltzer, R.S., Hoogenhuyze, D.V., Serruys, P.W., Haalebos, M.P., Hugenholtz, P.G., and Roelandt, J. Circulation 1981. 63. 1093/1099. Diagnosis of tricuspid regurgitation by contrast echocardiography.
18. Mintz, G.S., Kotler, M.N., Parry, W.R., Iskandrian, A.S., and Kane, S.A. Circulation 1981. 64. 1018/1025. Real-time inferior vena caval ultrasonography : normal and abnormal findings and its use in assessing right-heart function.
19. Nakasone, I., Sakakibara, H., Hasuda, Y., Okamoto, M., Miyatake, K., Kinoshita, N., Nagata, S., Beppu, S., and Nimura, Y. JSUM Proceedings 1982. 40. 527/528. Assessment of severity of tricuspid regurgitation by contrast M-mode echocardiography.
20. Nakamura, K., Kuroiwa, N., Kataoka, H., Fukuda, Y., Miyahara, K., and Hashimoto, S. JSUM Proceedings 1980. 36. 345/346. Diagnosis of tricuspid regurgitation by means of linear contrast echocardiogram with special emphasis on an attempt to evaluate severity of the disease.
21. Nakamura, K., Sanada, J., Minami, Y., Okumiya, K., Tabuchi, H., Furukawa, S., Miyahara, K., and Hashimoto, S. JSUM

Proceedings 1980. 37. 13/14. The linear contrast echogram in the direction of the right ventricle, tricuspid valve and the right atrium for estimating right ventricle end-diastolic pressure.

22. Ozaki, M., Handa, Y., Okabe, M., Naito, H., Hesaka, K., Takahashi, T., Sota, K., and Ohta, N. J. Cardiography 1980. 10. 173/186. Problem in the diagnosis of tricuspid insufficiency by contrast echocardiography injected into the peripheral vein : On the false positive findings in normal subjects.
23. Salazar, E., and Levine, H.D. Am. J. Medicine 1962. 33. 111/129. Rheumatic tricuspid regurgitation.
24. Silver, M.D., Lam, J., Rauganathan, N., and Wigle, E.D. Circulation 1971. 43. 333/348. Morphology of the tricuspid valve.
25. Takabatake, Y., and Iizuka, M. Jpn. Circ. J. 1974. 38. 843/851. Pathophysiology of tricuspid insufficiency.
26. Takamoto, T., Nitta, M., Miyahara, Y., Niwa, A., and Taniguchi, K. JSUM Proceedings 1980. 37. 11/12. Contrast echocardiographic evaluation of the right ventricular filling pattern in the patients with atrial septal defect.
27. Tamaki, E. Jpn. Circ. J. 1968. 32. 1515/1530. Studies on tricuspid regurgitation.
28. Tei, C., Pilgrim, J.P., Shah, P.M., Ormiston, J.A., and Wong, M. Circulation 1982. 66. 665/671. The tricuspid valve annulus : Study of size and motion in normal subjects and in patients with TR.
29. Tsakiris, A.G., Mair, D.D., Seki, S., Titus, J.L., and Wood, E.H. Circulation Research 1975. 36. 43/48. Motion of the tricuspid valve annulus in anesthetized dogs.
30. Tsuiki, K., Miyazawa, K., Ishikawa, K., and Katori, R. Jpn. Heart J. 1975. 16. 11/21. A method for quantitative detection of tricuspid regurgitation with double injection-single sampling dye-dilution technique.
31. Tsuyuguchi, N., Nohara, R., Fujita, M., Shigeta, H., Yoshimatsu, S., Suzuki, M., and Suwo, M. JSUM Proceedings 1980. 36. 347/348. Analysis of contrast echo disappearance time and its relation to hemodynamic parameters of radiocardiogram.
32. Ubago, J.L., Figueroa, A., Ochoteco, A., Colman, T., Duran,

R.M., and Duran C.G. Am. J. Cardiol. 1983. 52. 155/158. Analysis of the amount of tricuspid valve annular dilatation required to produce functional tricuspid regurgitation.

33. Veyrat, C., Kalmanson, D., Farjon, M., Manin, J.P., and Abitbol, G. Br. Heart J. 1982. 47. 596/605. Non-invasive diagnosis and assessment of tricuspid regurgitation and stenosis using one and two dimensional echo-pulsed Doppler.
34. Waggoner, A.D., Quinones, M.A., Young, J.B., Brandon, T.A., Shah, A.A. Verani, M.S., and Miller, R.R. Am. J. Cardiol. 1981. 47. 279/286. Pulsed Doppler echocardiographic detection of right-sided valve regurgitation
35. Wooley, C.F. Circulation 1978. 57. 1039/1053. Intracardiac phonocardiography.