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Reduced Atrial Contribution to Left Ventricular Filling in Patients With Severe Tricuspid Regurgitation After Tricuspid Valvulectomy: A Doppler Echocardiographic Study

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Patients undergoing valvulectomy for isolated tricuspid valve endocarditis offer the unique opportunity to study the effects of acquired right ventricular volume overload on left ventricular filling in persons free of pulmonary hypertension and preexisting left heart disease. Eleven patients who had undergone total or partial removal of the tricuspid valve were compared with 11 age-matched control subjects; Doppler echocardiographic techniques were used to quantify changes in left ventricular filling and to relate them to changes in left ventricular and left atrial geometry caused by right ventricular and right atrial distension.

The late diastolic fractional transmitral flow velocity integral, a measure of the left atrial contribution to left ventricular filling, was significantly decreased in patients undergoing tricuspid valvulectomy compared with control subjects (0.22 ± 0.11 versus 0.32 ± 0.09; $p < 0.04$). Severe tricuspid regurgitation in these patients resulted in marked right atrial distension, reversal of the normal interatrial septal curvature and compression of the left atrium such that left atrial area was significantly smaller than in control subjects (5.9 ± 2.2 versus 8.6 ± 1.2 cm^2/m^2; $p < 0.005$).

Acting as a receiving chamber, the left ventricle was maximally compressed by the volume-overloaded right ventricle in late diastole coincident with the timing of atrial systole, resulting in a significant increase in the left ventricular eccentricity index compared with that in control subjects (1.35 ± 0.14 versus 1.03 ± 0.1; $p < 0.001$). Thus, right ventricular volume overload due to severe tricuspid regurgitation results in left heart geometric alterations that decrease left atrial preload, impair left ventricular receiving chamber characteristics and reduce the atrial contribution to total left ventricular filling.

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Right ventricular volume overload results in diastolic geometric distortion of the left ventricle as a consequence of the parallel arrangement of the two ventricles within the heart (1–6). In the unloaded nonbeating human heart, the ventricular septum assumes a relatively flat neutral position between the two ventricles (7). In contrast, in the normally loaded beating heart, it assumes a more nearly circular arc concave with respect to the center of the left ventricle as a result of the normal positive diastolic left to right transseptal pressure gradient (7,8). With right ventricular volume loading, the diastolic transseptal pressure gradient between the left and right ventricles decreases (without necessarily reversing), resulting in ventricular septal flattening and displacement toward the center of the left ventricle (8–10). Dexter (11) first postulated that this diastolic interaction between the volume-overloaded right ventricle and the normal left ventricle might result in interference with left ventricular filling by a "reverse Bernheim's syndrome" (11,12).

Recent echocardiographic studies (6,13) of left ventricular geometry and function in patients with right ventricular volume overload have included patients with congenital defects that might alter left ventricular diastolic filling through mechanisms independent of right ventricular volume loading or patients with acquired conditions such as chronic obstructive pulmonary disease (13) in which an element of right ventricular pressure overload could not be excluded. Thus, in the present study, patients who underwent tricuspid valvulectomy for isolated tricuspid valve endocarditis were studied to evaluate the changes in left

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ventricular geometry and diastolic filling associated with acquired right ventricular volume overload due to severe tricuspid regurgitation. Although others have examined the hemodynamics (14) and clinical course (15) after total or partial removal of the tricuspid valve, we present findings regarding the geometric interactions between the left and right heart chambers and their impact on the pattern of left ventricular diastolic filling.

Methods

Study patients. Eleven patients (25 to 45 years of age, mean 33 ± 8) with severe tricuspid regurgitation after tricuspid valvulectomy for isolated involvement of the valve by infective endocarditis form the study group. Eight of the 11 patients were studied in an ambulatory hemodynamically stable state 1 week after tricuspid valvulectomy and just before discharge from the hospital or transfer to the medical service for completion of their course of antibiotic therapy. The remaining three patients were studied 4 to 47 months after tricuspid valvulectomy. Because Doppler and echocardiographic findings were comparable in these two subgroups, the data were pooled for purposes of analysis.

Six of the 11 patients had undergone total excision of all three leaflets of the tricuspid valve, 3 had undergone excision of two leaflets and 2 had undergone excision of a single leaflet; in each case, this resulted in the development of severe tricuspid regurgitation. In each instance, macroscopic surgical examination revealed that all tricuspid valve vegetations were excised and the underlying leaflet tissue appeared to be intrinsically normal. In no patient was there historical or echocardiographic evidence of prior pulmonary, myocardial, valvular or coronary artery disease or systemic hypertension.

All patients were studied in a hemodynamically stable ambulatory state, at which time right atrial pressure estimated from the vertical height of the meniscus of the internal jugular venous pulsations (assuming the sternal angle was 5 cm above the center of the right atrium) was estimated to be 14 ± 4 mm Hg. No patient was judged to have significant right ventricular systolic hypertension because the peak systolic velocity of tricuspid regurgitation assessed by continuous wave Doppler ultrasound was ≤2 m/s (right ventricle to right atrium peak systolic pressure differential ≤16 mm Hg) and hence right ventricular peak systolic pressure was <30 ± 4 mm Hg for each patient.

Eleven age-matched normal persons (25 to 47 years of age, mean 32 ± 8) without myocardial or valvular disease by pulsed Doppler and two-dimensional echocardiography served as control subjects. The study protocol was approved by the University of Illinois Institutional Review Board. Informed consent was obtained from all subjects.

Two-dimensional echocardiographic examination. Two-dimensional echocardiograms from standard left parasternal and apical windows and pulsed Doppler recordings were performed with use of a Diasonics CV 400 sector scanner interfaced with a 3.5 MHz mechanical transducer. The studies were performed while the subjects were in sinus rhythm, breathing quietly in the left lateral recumbent position. Parasternal short-axis images of the left ventricular cavity at the mitral leaflet, mitral chordal and papillary muscle levels were obtained by angling the transducer from base to apex. Orthogonality of these imaging planes to the left ventricular long axis was assessed by adjusting transducer angulation such that the medial and lateral aspects of the mitral commissures were symmetric at the mitral leaflet level. All studies were recorded on 0.5 in. (1.27 cm) videotape for subsequent real time playback and stop frame analysis.

Stop frame images from the mitral chordal level of the left ventricle were selected for analysis as follows: 1) end-systole: the stop frame demonstrating the smallest left ventricular cavity area just before the opening motion of the mitral chordae and leaflet tips; 2) end-diastole: the stop frame demonstrating the maximal left ventricular cavity area just before the closing motion of the mitral chordae and leaflet tips; and 3) mid-diastole: the stop frame image of the left ventricle at the end of the first half of diastole (determined by counting forward from the end-systolic frame one-half of the total number of frames encompassing diastole). The boundaries of the left ventricular endocardium were defined by the internal interface of the left ventricular endocardium with the blood pool. To assess left ventricular cavity shape, the short-axis left ventricular two-dimensional images proximal and distal to the mitral chordal level were examined to ascertain that the cavity profile at the chordal level was representative of left ventricular shape at all levels in each patient.

Standard apical four chamber images of the left ventricle and atrium were obtained, with careful attention directed toward ensuring that the imaging planes were aligned with the long axis of the left ventricular inflow tract such that maximal left ventricular and atrial cavity areas were obtained. Left atrial cavity area was measured from the apical four chamber image by tracing the internal interface between the left atrial wall and the blood pool (transecting pulmonary veins at their orifices and using the plane of the mitral anulus to define the boundary between the left ventricle and left atrium) (16,17). These measurements were obtained at the time of maximal left atrial cavity size.

Pulsed Doppler echocardiographic examination. Pulsed Doppler measurements were performed by positioning the Doppler cursor within the 80° two-dimensional sector scan at varying range gates to a maximal depth of 16 cm with a sample volume length of 4.1 mm.* Doppler output (filtered

*With the Diasonics instrument used in this study, a pulsed wave Doppler sample volume of 4.1 mm represents the best compromise between spatial specificity and adequate sensitivity for Doppler signals to provide clear spectra for accurate measurements.

with a 400 Hz high pass filter) produced an audio signal, as well as a spectral display derived by a fast Fourier transform algorithm. Transmitral inflow was recorded by positioning the transducer at the apical window to obtain a standard four chamber view of the heart with clear visualization of the ventricular apex, mitral valve and left atrium. The pulsed Doppler cursor was positioned parallel to the left ventricular long axis, running from the ventricular apex to the midpoint of the mitral orifice, and the sample volume was positioned at the level of the mitral anulus. In each patient, the presence of mitral regurgitation was excluded by detailed interrogation of the left atrium with pulsed Doppler ultrasound. Left ventricular outflow was recorded from the same apical window by angling the transducer anteriorly to record a four chamber view of the heart, including the left ventricular outflow tract. The pulsed Doppler cursor was aligned parallel to the outflow tract along a line running from the left ventricular apex to the midpoint of the aortic orifice, with the sample volume positioned just below the aortic valve. Minor angulation of the transducer was permitted to ensure that the interrogating Doppler beam was aligned with mitral or aortic flow.

Optimal alignment of the Doppler beam was judged to have been attained (18,19) when 1) a relatively pure audio signal was appreciated; 2) a reproducible display of the flow velocity envelope with minimal (<0.5 kHz) spectral broadening could be recorded; and 3) maximal peak flow velocities were measured. No correction was attempted for the angle between the Doppler cursor and the apparent direction of blood flow and in all instances that angle was estimated to be <20°. In no instance did the recorded Doppler frequency shift exceed the Nyquist limit for the range gate utilized. Pulsed Doppler spectra were recorded with a simultaneous lead II electrocardiogram (ECG) by a strip chart recorder scrolling at 50 mm/s. All subjects were in sinus rhythm with a normal PR interval.

Measurements and calculations. Left ventricular geometry and ventricular septal displacement were quantified by measuring the eccentricity index of the short-axis profile of the left ventricular cavity (mitral chordal level) at end-systole, mid-diastole and end-diastole (3,6,13). The ventricular septum was defined as the arc of the left ventricular circumference delimited by the attachments of the right ventricular free walls. A chord connecting the anterior and posterior limits of the ventricular septum at the left ventricular endocardial surface was constructed. The perpendicular bisector to that chord was constructed and defined the distance from the ventricular septal endocardium to left ventricular free wall endocardium as the minor-axis diameter (D_1). The orthogonal minor-axis diameter (D_2), measuring the distance between anterior and posterior left ventricular endocardial surfaces, was measured and D_2/D_1 (which equals the left ventricular cavity eccentricity index) was calculated. For an ideal circular left ventricular cavity, the eccentricity index would be equal to 1; however, with progressive flattening of the ventricular septum due to leftward shift toward the center of the left ventricle, the eccentricity index would be >1.

Maximal left atrial area normalized for body surface area was derived from the left atrial stop frame image corresponding approximately to end-ventricular systole. All two-dimensional echocardiographic measurements were performed with a video screen digitizer interfaced to a computerized graphics analyzer (Nova MicroSonics Data Vue System, MicroSonics, Inc.).

Doppler spectra of left ventricular outflow and mitral inflow were analyzed by digitizing the portion of the Doppler flow velocity spectrum with greatest intensity, defining the instantaneous modal flow velocity profile from which all subsequent measurements were derived. The time interval from the onset of the QRS complex on the ECG to the end of the systolic left ventricular outflow velocity profile (QA) and the time interval from the onset of the QRS complex to the beginning of the diastolic transmitral flow velocity profile (QM) were measured from separate tracings with matched cycle lengths. The time interval from aortic valve closure to mitral valve opening was calculated as QM−QA (18,20,21).

Peak mitral flow velocities in early (V_E) and late (V_A) diastole were measured from the midpoint of the Doppler spectral envelope at the time of maximal Doppler shift during the *early* rapid filling phase and *atrial* systolic phase of left ventricular diastole. Stroke distance (SD) was measured by numeric integration of the area under the transmitral flow velocity profile. Assuming the effective hemodynamic cross-sectional area at the mitral anulus (CSA) to be constant throughout diastole, peak left ventricular filling rate in early diastole (PFR[E]) expressed as stroke volumes per second was computed as (22):

$$PFR(E) = \frac{V_E \times CSA}{SD \times CSA} = \frac{V_E}{SD}.$$

Similarly, peak filling rate in late diastole (PFR[A]) was computed as V_A/SD.

To quantitate the relative contribution of the late (atrial systolic) filling phase of diastole to total left ventricular filling, the fractional transmitral flow velocity integral for atrial systolic left ventricular filling was determined by numeric integration (18). The onset of the late (atrial systolic) filling phase was defined as the onset of increasing velocities attributable to atrial systole. All Doppler echocardiographic measurements were performed by digitizing hard copy data with a Hitachi tablet digitizer (HDG-111B) interfaced to an IBM AT microprocessor using digitizing utility (VISHNU, Visual Numerics).

Statistical methods. Where appropriate, data were analyzed with Student's paired or unpaired *t* test. Probability

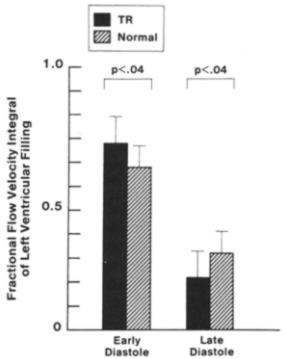


Figure 1. Doppler-derived fractional flow velocity integral of left ventricular filling in early and late diastole in 11 normal subjects and 11 patients with severe tricuspid regurgitation (TR) after tricuspid valvulectomy. In patients with severe right ventricular volume overload due to tricuspid regurgitation, the late diastolic fractional flow velocity integral of left ventricular filling is significantly reduced (0.22 ± 0.11) relative to that in the normal subjects (0.32 ± 0.09; $p < 0.04$).

values <0.05 were considered statistically significant. Data are reported as mean values ± 1 SD.

Results

Alterations in transmitral flow velocity profile (Fig. 1). For the 11 normal control subjects, the onset of transmitral flow into the left ventricle began 70 ± 25 ms after the cessation of aortic outflow. Peak transmitral flow velocity in early diastole during the rapid filling period was 52.4 ± 9.8 cm/s, and peak transmitral flow velocity in late diastole after atrial systole was 37.8 ± 8.2 cm/s.

For the 11 patients with severe isolated tricuspid regurgitation after tricuspid valvulectomy, the onset of forward transmitral flow velocities began 52 ± 32 ms (p = NS compared with normal subjects) after cessation of aortic outflow. In these patients with severe tricuspid regurgitation, peak transmitral flow velocity in early diastole (50.7 ± 13.2 cm/s) was not significantly different from that found in normal subjects. However, the peak transmitral flow velocity in late diastole resulting from active left atrial contraction (27.5 ± 11.1 cm/s) was significantly reduced ($p < 0.03$) compared with values in normal subjects.

Derived left ventricular peak filling rates (expressed as stroke volumes per second [SV/s]) in early and late diastole demonstrated a trend toward more rapid filling in early diastole and less rapid filling in late diastole in patients with severe tricuspid regurgitation compared with normal subjects. In the former, peak filling rates in early diastole were 7.9 ± 2.3 compared with 6.7 ± 2.0 SV/s in normal subjects (p = 0.22), whereas peak filling rates in late diastole were 4.0 ± 1.1 compared with 4.9 ± 1.6 SV/s in normal subjects (p = 0.17). The ratio of late to early peak filling rates in patients with right ventricular volume overload due to severe tricuspid regurgitation (0.54 ± 0.20) was significantly less than that found in normal subjects (0.72 ± 0.12, $p < 0.02$).

To further characterize the relative contribution of left atrial systole to total left ventricular filling, fractional flow velocity integrals for late diastolic left ventricular filling were measured (Fig. 1). The late diastolic fraction of the transmitral flow velocity integral attributable to atrial systole was significantly lower in patients with severe tricuspid regurgitation (0.22 ± 0.11) than in normal subjects (0.32 ± 0.09, $p < 0.04$). Consequently, left ventricular filling was redistributed to the rapid filling period, such that the early diastolic fraction of the transmitral flow velocity integral was greater in patients with severe tricuspid regurgitation (0.78 ± 0.11) compared with normal subjects (0.68 ± 0.09, $p < 0.04$).

Impact of right ventricular volume overload on left ventricular diastolic geometry (Fig. 2). The short-axis two-dimensional echocardiographic image of the left ventricular cavity exhibited a relatively circular profile throughout diastole in normal persons. Accordingly, the normal eccentricity index (minor axis parallel to the ventricular septum/minor axis perpendicular to the ventricular septum) assumed a value close to unity during left ventricular filling. For the 11 normal control subjects in this study, left ventricular cavity eccentricity index was 1.00 ± 0.05 at end-systole and did not change significantly at mid-diastole (1.03 ± 0.09) or at end-diastole (1.03 ± 0.10).

In the 11 patients with right ventricular volume overload due to tricuspid regurgitation, the short-axis profile of the left ventricular cavity was relatively circular at end-systole. Through the course of diastole, however, the left ventricular cavity was progressively compressed as the ventricular septum shifted toward the center of the cavity, becoming "D"-shaped rather than circular as a result of abnormal flattening of the septal curvature (Fig. 2). This encroachment on the left ventricular cavity is reflected by a change in the eccentricity index from near unity (1.07 ± 0.09) at end-systole to 1.30 ± 0.23 at mid-diastole ($p < 0.02$) and to 1.35 ± 0.14 at end-diastole ($p < 0.001$). Thus, in the latter half of diastole, there was maximal distortion of left ventricular geometry as a result of leftward ventricular septal displacement, resulting in an end-diastolic left ventricular eccentricity index significantly greater than that measured in normal subjects (1.35 ± 0.14 versus 1.03 ± 0.10, $p < 0.001$). The late diastolic timing of the maximal distortion of left ventricular geometry in these patients with right ventricular volume overload coincides with the observed decrement in peak

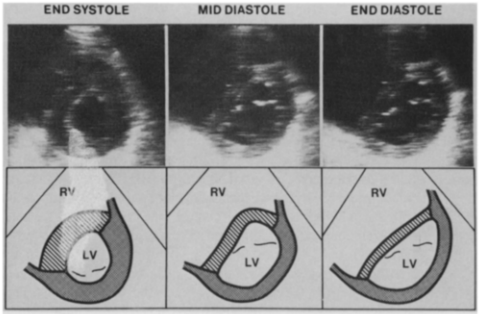


Figure 2. Two-dimensional echocardiographic images (with accompanying diagrams) from the short-axis plane of the left ventricle (LV) at the chordal level in a patient with severe tricuspid regurgitation due to tricuspid valvulectomy. In contrast to normal patients whose left ventricular cavity maintains a circular profile throughout diastole, this patient with right ventricular (RV) volume overload exhibits a circular left ventricular profile at end-systole and a progressive leftward shift of the ventricular septum toward the center of the left ventricle at mid-diastole and end-diastole. The left ventricle is maximally compressed into a D-shaped configuration by the distended right ventricle at end-diastole.

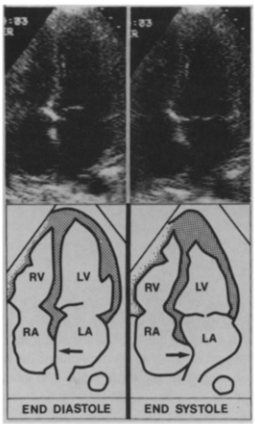
transmitral flow velocities and fractional flow velocity integrals during late diastole.

Impact of severe tricuspid regurgitation on maximal left atrial area (Fig. 3). In the 11 normal subjects, maximal left atrial area (at the end of ventricular systole) normalized for body surface area was 8.6 ± 1.2 cm^2/m^2. Whereas in normal subjects the interatrial septum always bowed slightly toward the right atrium (resulting in a concave contour with respect to the center of the left atrium), in each of the patients with severe tricuspid regurgitation due to tricuspid valvulectomy, the interatrial septum was convex with respect to the center of the left atrium in either the apical four chamber or parasternal short-axis view. This abnormality was most striking when the right atrium attained its maximal cross-sectional area, coinciding closely with the timing of maximal left atrial expansion (Fig. 3). This encroachment on the left atrium by the right atrium in patients with severe tricuspid regurgitation was associated with significantly decreased maximal left atrial area compared with that in normal subjects (5.9 ± 2.2 versus 8.6 ± 1.2 cm^2/m^2, $p < 0.005$). Thus, marked right atrial enlargement due to severe tricuspid regurgitation results in displacement of the interatrial septum toward the center of the left atrium and is associated with a decrease in maximal left atrial filling.

Discussion

Alterations in left ventricular and left atrial geometry due to severe tricuspid regurgitation. In this study, we examined the effects of acquired right ventricular volume overload on left ventricular geometry and diastolic filling in a group of patients free of left heart disease and pulmonary hypertension. Total or partial valvulectomy for isolated tricuspid valve endocarditis in each instance resulted in severe diastolic overload of the right ventricle and overfilling of the right atrium. Mid- to late-diastolic shift of the ventricular septum toward the center of the left ventricle resulted in late diastolic compression of the left ventricular cavity, such that its normal circular short-axis cavity profile became

Figure 3. Two-dimensional echocardiographic images (with accompanying diagrams) from the apical four chamber view of the heart. In this patient with tricuspid regurgitation due to tricuspid valvulectomy, distension of the right atrium (RA) in ventricular systole results in progressive leftward shift of the interatrial septum (arrow) toward the center of the left atrium (LA). Although the interatrial septum (arrow) is normally positioned at ventricular end-diastole, by ventricular end-systole the left atrium is maximally compressed and the interatrial septum is convex with respect to the center of the left atrium. LV = left ventricle; RV = right ventricle.



D-shaped, with progressive flattening of the ventricular septum. This distortion of left ventricular geometry was reflected in the relative reduction in the ventricular septal to free wall minor-axis dimension of the left ventricle, resulting in an end-diastolic left ventricular eccentricity index of 1.35, which was 31% greater than that (1.03) in control subjects. Thus, mid- to late-diastolic distortion of the left ventricular chamber as a result of right ventricular volume overload resulted in maximal left ventricular deformation during late diastole at the time of active left atrial contraction. These observations are consistent with echocardiographic measurements in patients with a variety of clinical conditions characterized by predominant right ventricular volume overload (1–3,5,6,8,9,13).

In right ventricular volume overload due to severe tricuspid regurgitation, there is the potential for right and left heart interdependence not only at the ventricular level, but also at the atrial level. In each of our patients, marked distension of the right atrium due to severe tricuspid regurgitation resulted in reversal of the normal atrial septal curvature from concave to convex with respect to the center of the left atrium. Thus, during right ventricular systole, the right atrium becomes maximally distended by severe tricuspid regurgitation, resulting in leftward displacement of the interatrial septum and compression of the left atrium at the point in the cardiac cycle when the left atrium should be achieving maximal filling. As a consequence, maximal left atrial area in these patients with severe tricuspid regurgitation averaged only 5.9 cm^2/m^2, 31% less than that in control subjects (8.6 cm^2/m^2).

Previous investigators studying animal models (23–25) and humans (26) demonstrated that within the limits of normal physiologic distension of the atria (23,24), active atrial systolic shortening increases with progressive volume loading of the atria, consistent with the operation of Frank-Starling forces in response to atrial distension. It appears that in patients with severe tricuspid regurgitation left atrial preload and atrial systolic function are reduced because left atrial maximal volume is reduced as a result of mechanical encumbrance by the volume-overloaded right atrium. Hence, these patients with severe isolated tricuspid regurgitation manifest interactions between the right and left heart chambers, resulting in distortion of left atrial and left ventricular geometry, both of which may foster a reduction in the active contribution of the left atrium to diastolic filling of the left ventricle.

Pulsed Doppler quantitation of the left atrial systolic contribution to left ventricular filling in right ventricular volume overload. To assess the impact of these geometric factors on the contribution to late diastolic filling of the left ventricle, we utilized pulsed Doppler measurements of the transmitral flow velocity integral at the mitral anulus to quantitate left atrial booster function in our patients with severe tricuspid regurgitation after tricuspid valvulectomy. We (18) and others (27–30) have demonstrated that such Doppler measurements correlate closely with those obtained by cineangiographic (27), radionuclide angiographic (28–30) and two-dimensional echocardiographic (18) techniques. In our patients, left atrial systole contributed approximately 22% of total left ventricular filling, which was almost 33% less than that measured in age-matched control subjects. Thus, it appears that the combination of 1) late diastolic left ventricular compression by the volume-overloaded right ventricle, and 2) reduced left atrial systolic ejection due to reduced left atrial preload, resulting from right atrial compression of the left atrium, combined to reduce the relative contribution of left atrial systole to total left ventricular filling in these patients.

Alternative mechanisms for the reduction in left atrial systolic contribution to left ventricular filling in right ventricular volume overload. Although the right and left heart chambers are anatomically juxtaposed in parallel, they pump in series. Thus, one might postulate that the altered pattern of left ventricular filling seen in patients with severe tricuspid regurgitation could result from reduced forward right ventricular stroke volume and, hence, reduced input to the left side of the heart. Although we cannot exclude the possibility that reduced right heart stroke output contributed to the underfilling of the left atrium in this study, several considerations suggest that this is not the only explanation for the reduction in the left atrial contribution to left ventricular filling seen in our patients. 1) Right ventricular forward stroke volume (as estimated by transmitral stroke distance) was not significantly different between our patients with severe tricuspid regurgitation and control subjects (7.1 ± 2.9 versus 8.4 ± 2.8 cm), suggesting that stroke volume and hence total left ventricular filling were not different between the two groups. 2) We predict that reduced right ventricular forward output decreases left heart volume loading and left atrial filling pressure, conditions that result in a *relative increase* in the contribution of atrial systole to total left ventricular diastolic filling (31–33) and that therefore do not explain the *decrease* in the late diastolic fractional transmitral flow velocity integral demonstrated in our patients.

We have related observed geometric distortions of the left ventricle and atrium to measurements of reduced left atrial contribution to left ventricular filling, but we cannot prove a causal relation. One could argue that the abnormal filling pattern might be the cause for the observed geometric changes. This hypothesis seems unlikely, however, in view of the temporal sequence of events. For instance, while left ventricular geometry was maximally distorted at end-diastole in our patients with right ventricular volume overload, the deformation was established by mid-diastole (eccentricity index 1.30) long before left atrial contraction. Thus, it is more plausible to postulate that left ventricular distortion due to right ventricular distension impedes late diastolic left ventricular filling, rather than proposing that it

results from impaired atrial contribution to ventricular filling.

Methodologic considerations. Doppler-derived fractional transmitral flow velocity integrals characterize the time-varying pattern of left ventricular filling velocities and not volumetric flow rate. The correlation between volumetric flow rate and filling velocities is predicated on the assumption that systematic errors are not incurred by variations in the effective hemodynamic cross-sectional flow area. Similarly, our calculations of peak filling rates normalized for stroke volume were based on the assumption of relative constancy of mitral annular cross-sectional flow area throughout diastole (22). It could be argued that the demonstrated distortions in left ventricular geometry in our patients with severe right ventricular volume overload might result in distortion of the mitral anulus and hence indirectly influence the transmitral flow velocity profiles. This potential source of error is unlikely to account for our results for several reasons. 1) Our previous studies (18) in patients with isolated right ventricular pressure overload and marked *early* diastolic left ventricular geometric distortion demonstrated a strong correlation ($r = 0.74$) between Doppler-derived early diastolic fractional filling (which presumably might be influenced by alterations in mitral annular geometry) and echocardiographically measured early diastolic left ventricular cavity expansion (which is a measure of ventricular filling independent of assumptions regarding mitral annular geometry). 2) Presumably, late diastolic left ventricular compression would, if anything, serve to decrease the effective hemodynamic cross-sectional flow area at the mitral anulus. For any given fixed rate of volumetric flow, we would expect these geometric changes to result in an increase in late diastolic filling velocities, not the decrease documented in this study. 3) If, in fact, the effective hemodynamic cross-sectional flow area for mitral inflow is reduced in late diastole relative to early diastole, then the volumetric flow rates in late diastole (product of cross-sectional flow area and flow velocity) would be proportionately decreased and would magnify our results. Accordingly, by using fractional flow velocity integrals to quantitate relative left ventricular filling, and then assuming relative constancy of effective cross-sectional flow area, we may have overestimated (not underestimated) the relative volumetric contribution of atrial systole to total left ventricular filling in our patients with right ventricular volume overload. Accordingly, correction of our data for the potential effects of late diastolic left ventricular compression on cross-sectional flow area would serve to strengthen our observation that the atrial contribution to left ventricular filling is significantly reduced in patients with right ventricular volume overload.

Clinical implications. In this study, we examined the geometric and functional impact of relatively pure right ventricular volume overload on left ventricular diastolic filling in patients with severe tricuspid regurgitation without preexisting pulmonary hypertension or left heart disease. We confirmed the late diastolic left ventricular geometric distortion previously documented (1–6,13) in disease states characterized by predominant right ventricular volume overload associated with mild pulmonary hypertension, intracardiac shunts and other congenital anomalies. During late diastole, the left ventricle fills passively as a result of active left atrial contraction, and the magnitude of that contribution to left ventricular filling is governed by a complex interplay of passive left ventricular chamber compliance, atrial preload and the atrial contractile state (31,34). In our study patients with severe tricuspid regurgitation, interactions between the right and left heart chambers combine to impair the ability of the left ventricle and atrium to act as receiving chambers, resulting in a reduction in the atrial contribution to left ventricular filling at a critical time in the diastolic filling period. These observations serve to highlight the clinical paradox of systemic venous engorgement in the setting of impaired late diastolic left ventricular filling in these patients.

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