Asymmetry of Right Ventricular Enlargement in Response to Tricuspid Regurgitation

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**Background**—Analysis of right ventricular adaptation to tricuspid regurgitation was studied in 10 heart transplant recipients following inadvertent endomyocardial biopsy disruption of the tricuspid apparatus.

**Methods and Results**—Echocardiography demonstrated progressive diastolic right ventricular cavity enlargement (19.5±5.0 to 30.3±5.4 cm², P<0.0002), with disproportionate elongation along the midminor axis (3.5±0.6 to 5.0±0.5 cm, P<0.001). As the right ventricle remodeled to more spherical (and less elliptical) proportions, the end-diastolic right ventricular midminor axis/long axis ratio increased significantly from 0.52±0.10 to 0.68±0.07, P<0.005.

**Conclusions**—Ventricular enlargement due to right ventricular volume overload results in disproportionate dilation along the free wall to septum minor axis. (Circulation. 1999;100:465-467.)

**Key Words:** ventricles • regurgitation • right ventricular volume overload • tricuspid regurgitation • orthotopic heart transplantation

R ight ventricular volume overload distorts ventricular septal geometry,1 alters left ventricular filling,1,2 and impairs left ventricular systolic function.3–7 Inadvertent tricuspid valve trauma due to endomyocardial biopsy in heart transplant recipients results in the abrupt onset of severe tricuspid regurgitation, providing the opportunity to measure the progressive longitudinal impact of right ventricular volume overload on chamber geometry and function.

**Methods**

**Study Population**

Survveillance endomyocardial biopsy, right heart catheterization, and postprocedural echocardiography were prospectively performed during the first 4 years after cardiac transplantation in 365 patients; the study was conducted from 1984 to 1994. Review of the echocardiograms identified 54 patients in whom endomyocardial biopsy resulted in a flail tricuspid apparatus (index study). The immediately preceding echocardiogram demonstrating normal tricuspid valve anatomy (baseline study) and the succeeding echocardiograms at 6, 12, and 24 months following the index study were selected for longitudinal analysis. This study was approved by the Institutional Review Board and all patients gave informed consent for the diagnostic procedures.

Subjects were excluded from this study if they had any of the following conditions which might independently affect ventricular function: (1) systemic hypertension: blood pressure >160/104 mm Hg or an increase >25 mm Hg unresponsive to medication; (2) pulmonary hypertension: systolic pulmonary artery pressure >30 mm Hg or transpulmonary gradient >15 mm Hg; (3) diabetes mellitus requiring pharmacological therapy; (4) moderate to severe cardiac rejection: endomyocardial biopsy grade ≥48 requiring treatment with intravenous methyl prednisolone and/or OKT 3 antithymocyte globulin; (5) allograft coronary arteriopathy detected by annual coronary angiography; and (6) left ventricular ejection fraction <50% at baseline.

Ten subjects were identified who met these exclusion criteria and had technically superior echocardiograms suitable for quantitative measurements. At baseline, this study population ranged in age from 21 to 65 years (mean 46±15); 7 subjects were male.

**Echocardiographic Measurements**

Transthoracic echocardiograms were performed within 3 hours after endomyocardial biopsies and right heart catheterization using standard techniques. The transducer was positioned to maximize right ventricular cavity size and end-systolic images were selected at minimal ventricular cavity size. Mid-systole was identified by counting half the number of image frames between end diastole and end systole. A flail tricuspid valve was defined by9 (1) lack of normal coaptation of its leaflets, (2) whiplike systolic prolapse of its leaflet tips into the right atrium, and (3) erratic motion of its malcoapting leaflet segments.

All measurements of right ventricular geometry,10 RVA, and tricuspid annulus size11 were obtained from apical 4-chamber views of the heart through the cardiac crux. The right ventricular long axis (L), maximal minor axis (S1), and midminor axis (S2) dimensions are defined in Figure 1. Percent systolic fractional shortening for RVA, L, S1, and S2 was calculated as 100×(end-diastolic measurement–end-systolic measurement)/end-diastolic measurement. The ratio of right ventricular minor axis dimension to L dimension was calculated for both S1 and S2 as an index of asymmetry of right ventricular enlargement.

**Statistical Methods**

For the predetermined time points (index, 6, 12, and 24 months), measurements were compared with baseline by a repeated measures
Hemodynamic and Clinical Parameters

Relative to baseline, there were no significant changes in body weight (64±12 versus 68±9 kg), cardiac index (2.7±0.5 versus 2.5±0.8 L·min⁻¹·m⁻²), mean systemic arterial pressure (95±8 versus 107±12 mm Hg), systolic pulmonary artery pressure (26.5 versus 22±4 mm Hg), mean pulmonary artery pressure (17±3 versus 14±3 mm Hg), or mean pulmonary artery capillary wedge pressure (10.4 versus 8±3 mm Hg) at 2-year follow-up.

Impact of Right Ventricular Volume Overload on Right Ventricular Geometry

Tricuspid annular diameter was significantly enlarged at 24 months relative to baseline for end-diastolic (4.0±0.5 versus 3.2±0.4 cm, P<0.01), mid-systolic (3.6±0.5 versus 2.8±0.3 cm, P<0.001), and end-systolic (3.3±0.4 versus 2.5±0.3 cm, P<0.01) measurements. End-diastolic RVA (30.3±5.4 versus 19.5±5.0 cm², P<0.0002), and end-systolic RVA (16.7±3.7 versus 11.0±3.2 cm², P<0.001) were also increased significantly at 24 months relative to baseline. The right ventricular L dimension remained relatively unchanged over this period (7.4±0.9 versus 6.9±0.8 cm, P=NS), whereas the right ventricular S1 dimension increased from 4.0±0.4 to 5.1±0.7 cm (P<0.001), and the right ventricular S2 dimension increased from 3.5±0.6 to 5.0±0.5 cm (P<0.001; Figure 2). As a result, the ratio of right ventricular S2 to L at end diastole (0.68±0.07 versus 0.52±0.10, P<0.005) was significantly greater at 24 months than at baseline, as were the midsystolic (0.63±0.06 versus 0.47±0.09, P<0.005) and end-systolic minor axis/L ratios (0.56±0.06 versus 0.42±0.11, P<0.05).

Discussion

Severe tricuspid regurgitation due to the development of a flail tricuspid valve resulted in a 25% increase (P<0.01) in end-diastolic tricuspid annular diameter and a 55% (P<0.0002) increase in end-diastolic RVA at 2-year follow-up. The end-diastolic L of the right ventricle did not increase significantly compared with baseline (7% increase), whereas the S1 dimension increased 28% (P<0.001) and the S2 dimension increased 43% (P<0.001) over the same period. Thus, the right ventricle preferentially dilates in the free wall
to ventricular septal axis in response to right ventricular volume overload.

Comparison to Earlier Studies
In a cross-sectional study comparing 10 orthotopic heart transplant recipients with flail tricuspid valves to 62 orthotopic heart transplant recipients with normal tricuspid valves, the magnitude of right ventricular enlargement due to right ventricular volume overload was comparable to that found in our longitudinal study over 2 years. Our longitudinal study builds on these observations by demonstrating that the right ventricular enlargement is asymmetric with preferential dilation along the right ventricular minor axis. The resultant distortion of ventricular septal geometry may have important implications for our preliminary observation of progressive depression in left ventricular ejection fraction seen in these and other patients with severe tricuspid regurgitation.3–7

References
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