Correlation of Right and Left Ventricular Ejection Fraction and Volume Measurements

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First-pass radiocardiography and biplane angiocardiography were performed on 13 patients with left-sided regurgitant valvular disease (R+) and 7 patients without regurgitation but with coronary artery disease and/or cardiomyopathy (R−). Right and left ventricular volumes and ejection fractions were calculated and compared. In the R− group, corresponding right and left ventricular volumes and ejection fractions correlated highly with each other ($r = 0.86$–0.89, $p \simeq 0.01$). Ejection fractions in the R+ group correlated ($r = 0.64$, $p < 0.05$) only because stroke volume correlation was very high ($r = 0.93$), with end-diastolic and end-systolic volumes showing no significant correlation. Right ventricular ejection fraction (RVEF) decreased significantly with increasing mean pulmonary artery pressure (PAP) in both R− and R+ groups. The correlation of RVEF and LVEF in the R− group appears to be multifactorial in origin, consisting of effects of increased PAP, the mechanical interference of an enlarged left ventricle on the right ventricle, and direct biventricular ischemic effects. In the R+ group, the correlation appears to be due to only increased PAP and its sequelae.

J Nucl Med 29:1114–1117, 1988

The heart is frequently regarded as a simple pump, with its right and left sides considered to be in a series configuration but essentially independent of each other. As a result, the forward stroke volume must be equal for both sides if homeostasis is to maintain. Despite this tendency to consider the right and left sides of the heart as independent entities, it seems reasonable to suppose that deranged morphology and function of one cardiac chamber may indeed affect one or more of the other chambers (*1–4*).

We have studied the correlations of right ventricular and left ventricular ejection fractions (RVEF and LVEF) and volume measurements in patients with left-sided regurgitant valvular disease (R+) and in patients with coronary artery disease (CAD) and/or cardiomyopathy but without regurgitant valvular disease (R−) in order to define these interrelationships and to elucidate their mechanisms.

METHODS

The patient population and experimental methods were described in detail elsewhere (*5*). Briefly, the study group consisted of 20 patients in whom first-pass radionuclide angiography was performed within 48 hr of cardiac catheterization. Of these, 13 patients (R+) had left-sided (predominantly mitral) regurgitant valvular disease, and 7 patients (R−) had CAD and/or cardiomyopathy but did not have regurgitant valvular disease.

During cardiac catheterization, pulmonary artery pressure (PAP) was measured. Right ventricular cardiac output was determined using both the Fick principle and the indocyanine green indicator dilution method, and standard angiocardiographic geometric methods were used to obtain left ventricular end-diastolic volume (EDV), end-systolic volume (ESV), stroke volume (SV), and ejection fraction (EF) to validate the first-pass techniques employed (*5*). These angiocardiographic data were not used in this present paper.

Blood volume was determined using 5–10 μCi (200–400 KBq) of iodine-125 (^{125}I) albumin. First-pass radionuclide angiography was performed in the LAO projection using an Anger camera and a bolus injection of 8–10 mCi (300–400 MBq) of technetium-99m- (^{99m}Tc) albumin. First-pass RVEF and LVEF were calculated on a beat-by-beat basis using 25 frames per second images. The first-pass data were reformatted into two-frames-per-second images and used to calculate chamber cardiac output by the Stewart-Hamilton method. The radionuclide cardiac output data were combined with the first-pass EF data to calculate RV and LV SV, EDV, and ESV. Lung data were taken to be representative of RV performance (*5*). Left ventricular SV was corrected for regurgitation and bolus smearing effect (*5*).

Standard linear regression techniques were used to compare

Received July 22, 1987; revision accepted Nov. 12, 1987.

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data (6). The correlation coefficients were considered to be statistically significant if the p value was <0.05 (6).

RESULTS

Patients Without Regurgitant Valvular Disease (R−)

RVEF and LVEF correlated highly ($r = 0.86$, $p \simeq 0.01$; Fig. 1). Because of the limited number of patients and the absence of cases with LVEF < 40% in our series, we analyzed raw data presented but not analyzed by Korr et al. (7). In their series of 54 patients, ranging from normals to patients with severe CAD, RVEF and LVEF again correlated ($r = 0.77$, $p < 0.001$; Fig. 2). Our data and the data from Korr et al. agree well with data from Steele et al. (8), who demonstrated a highly significant correlation ($p < 0.001$) of RVEF and LVEF in 96 men with CAD.

Correlations of radionuclide RV and LV SV, EDV, and ESV were all significant at the $p \simeq 0.01$ level and are summarized in Table 1.

As expected, there is a good reciprocal relationship between LVEF and LVEDV ($r = -0.87$, $p \simeq 0.01$; Table 2). This indicates that in our group of patients with LVEF over 40%, SV is well maintained. Since none of our R− patients exhibited LVEF < 40%, we analyzed the data of Korr et al. (7). These data show the fall in LVEF that accompanies decompensation, chamber enlargement, and resultant increased PAP (Fig. 3; $p < 0.001$). Both in our series and that of Korr et al., RVEF fell as PAP increased; the fall in RVEF was also associated with a rise in RVEDV (Table 2).

Patients With Regurgitant Valvular Disease (R+)

In R+ patients, the correlation of RVEF with LVEF was significant because the RVSV-LVSV correlation was highly significant; EDV and ESV were not correlated in this group of patients with regurgitant valvular disease (Table 3).

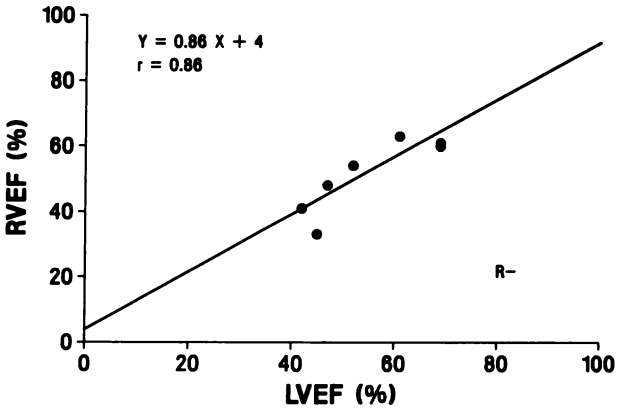

FIGURE 1
Correlation of LVEF and RVEF in patients with coronary artery disease and/or cardiomyopathy but without regurgitant valvular disease.

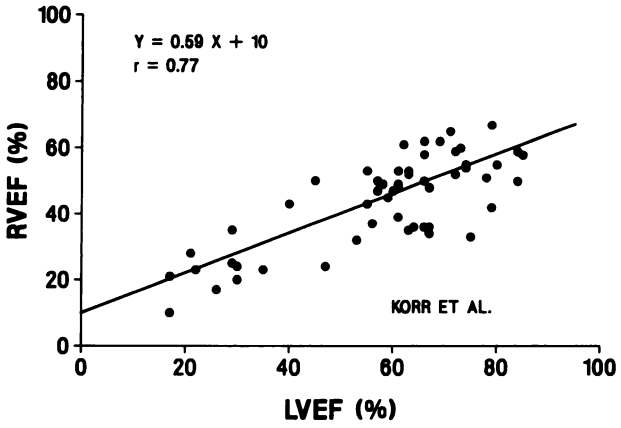

FIGURE 2
Correlation of LVEF and RVEF in normals and in patients with varying degrees of coronary artery disease; data presented but not analyzed in Ref. 7.

Regurgitant valvular disease causes increased LVEDV and consequent decreased LVEF (Table 4); there was no correlation of LVSV with LVEF. As decompensation occurs and LVSV decreases, the mean PAP increases (Table 4). Associated with the rise in PAP was a decrease of RVSV (Table 4); this rise in PAP and decrease in RVSV leads to a decrease in RVEF (Table 4).

DISCUSSION

Since the cardiovascular system is a closed loop and since the two sides of the heart are in direct apposition, it is reasonable to expect that disruptions in one portion of the system will cause disruptions in other portions. Thus, failure of the left ventricle should ultimately lead to malfunction of the right ventricle. Although the two sides of the heart have been traditionally treated as essentially independent pumps (9), in fact, both their structure and function are interrelated in a complex manner. We have investigated these interrelationships in two distinct patient populations, namely patients

TABLE 1
Correlation of Right Ventricular and Left Ventricular Ejection Fraction and Volumes in 7 Patients Without Regurgitant Valvular Disease (R−)

Linear regression equation	Correlation coefficient
RVEF = 0.86 [LVEF] + 4%	0.86**
RVSV = 0.79 [LVSV] + 8 ml	0.87**
RVEDV = 0.72 [LVEDV] + 38 ml	0.89**
RVESV = 0.77 [LVESV] + 18 ml	0.87**

** $p < 0.01$

TABLE 2
Correlation of Ejection Fraction, End Diastolic Volume, and Pulmonary Artery Pressure in Patients Without Regurgitant Valvular Disease (R−)

Linear regression equation	Correlation coefficient
LVEF = −0.14 [LVEDV] + 81 ml	−0.87**
RVEF = −1.2 [PAP] + 70%	−0.77*
RVEF = −0.95 [PAP] + 64% (Ref. 7)	−0.82***
RVEF = −0.15 [RVEDV] + 77%	−0.75*

*p < 0.05.
**p < 0.01.
***p < 0.001.

TABLE 3
Correlation of Right Ventricular and Left Ventricular Ejection Fractions and Volumes in 13 Patients with Regurgitant Valvular Disease (R+)

Linear regression equation	Correlation coefficient
RVSV = 0.73 [LVSV] − 1 ml	0.93***
RVEF = 0.65 [LVEF] + 12%	0.64*
RVEDV = 0.17 [LVEDV] + 153 ml	0.35 NS
RVESV = 0.23 [LVESV] + 83 ml	0.35 NS

*p < 0.05.
***p < 0.001.

with CAD and patients with regurgitant valvular disease.

Patients Without Regurgitant Valvular Disease (R−)

This group of patients had varying degrees of CAD and/or cardiomyopathy, but with angiocardiographically proven absence of valvular regurgitation. The data we and others have obtained from this patient population allow us to propose three mechanisms by which left ventricular and right ventricular performance decrements may be related.

In CAD, the cause of left ventricular failure is ischemia and necrosis, with increased PAP as an ultimate consequence of ischemia. The ischemic myocardium exhibits decreased contractility, requiring the ventricle to dilate in order to maintain stroke volume. Eventually, stroke volume becomes compromised as left ventricular contractility decreases with progressive disease. The reduced forward stroke volume and/or the increased LVEDV causes reduced LVEF and results in increased resistance to flow and subsequent increased PAP (Fig. 3). In order to compensate for the increased pressure and maintain forward stroke volume, the right ventricle becomes dilated (increased EDV) and, as a result, RVEF falls. Thus, left ventricular failure eventually leads to right ventricular malfunction by the PAP mechanism. Patients studied sequentially as their CAD worsens would show normal RVEF and RVEDV early, increasing RVEDV in later stages, and frank failure of the RV in the late stages. Our data and the data of others (*7–8*) support this hypothesis (Figs. 1 and 2; Tables 1 and 2).

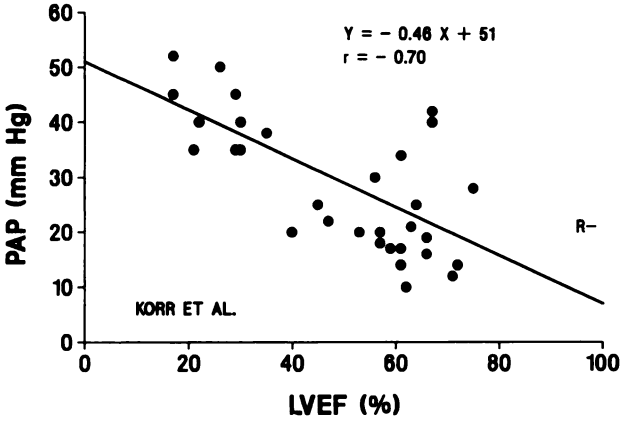

FIGURE 3
Correlation of LVEF and mean pulmonary artery pressure in patients with varying degrees of coronary artery disease; data presented but not analyzed in Ref. 7.

As a second mechanism, ischemic or other cardiomyopathies may result in decreased RV and LV performance due to mechanical interference between the two chambers (the Bernheim effect; *1*). As the LV enlarges, it displaces the septum to the right and interferes with RV contractile function. The RV would thus enlarge to compensate for impaired contractility and the increased EDV would lead to a decreased RVEF (Figs. 1 and 2; Tables 1 and 2).

A third mechanism for correlated biventricular failure is multivessel CAD, in which both ventricles are ischemic simultaneously. In this instance, each ventricle will dilate and ultimately fail because the ischemic process affects both ventricles simultaneously.

In any event, the diminution in RVEF in this group of patients is related to both the decrease in stroke volume and the increase in end diastolic volume as the disease progresses.

Patients with Regurgitant Valvular Disease (R+)

Early in its course, regurgitant valvular disease with its attendant volume overload causes enlargement of

TABLE 4
Correlation of Left Ventricular and Right Ventricular Ejection Fractions and Volumes and Pulmonary Artery Pressures in 13 Patients with Regurgitant Valvular Disease (R+)

Linear regression equation	Correlation coefficient
LVEF = −0.09 [LVEDV] + 63%	−0.73**
PAP = −0.38 [LVSV] + 65 mm Hg	−0.84***
RVSV = −1.4 [PAP] + 110 ml	−0.83**
RVEF = −0.96 [PAP] + 64%	−0.76**
RVEF = 0.42 [RVSV] + 7.3%	0.58*

*p < 0.05.
**p < 0.01.
***p < 0.001.

the left ventricle. This increase in LVEDV subsequently leads to decreased LVEF (Table 4). Since stroke volume is preserved in this early stage, there is no correlation between LVEF and LVSV in the R+ group.

In later stages, as decompensation occurs, LVSV decreases and PAP increases (Table 4). The higher PAP leads to decreased RVSV, since the forward stroke volumes for both ventricles must be the same at equilibrium (Table 4). The concomitant decrease in both RVSV and LVSV is very highly correlated ($r = 0.93$, $p < 0.001$; Table 3). Decreased RVSV due to the higher PAP results in decreased RVEF (Table 4). As a consequence, there is a correlation between RVEF and LVEF in regurgitant valvular disease (Table 3).

Thus, the major mechanism of the interrelationship of right and left ventricular function in regurgitant valvular disease appears to be through pulmonary afterload effects, as in chronic obstructive pulmonary disease (*10*). Mechanical factors, which are important in our CAD (R−) patients, do not appear to be important in the R+ group, as shown by the failure of correlation between right and left EDV and ESV (Table 3).

In contrast to the R− group, the diminution of RVEF in regurgitant valvular disease is predominantly due to alterations in stroke volume alone.

CONCLUSIONS

In CAD, the correlation of RV and LV ejection fractions and volumes appears to be multifactorial in origin, consisting of the effects of increased PAP, mechanical interference (Bernheim effect), and, ischemic effects.

The correlation of RVEF and LVEF in regurgitant valvular disease appears to be mediated predominantly through increased PAP and its sequelae. The right ventricular findings are similar to those seen in chronic obstructive lung disease.

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