Right Ventricular Ejection Fraction During Exercise in Patients with Coronary Artery Disease

LYNNE L. JOHNSON, M.D., DAVID M. MCCARTHY, M.D., ROBERT R. SCIACCA, ENG.SC.D., and PAUL J. CANNON, M.D.

SUMMARY The effect of the location of right coronary artery (RCA) lesions upon right ventricular ejection fraction (RVEF) during exercise was investigated using first-pass radionuclide angiography. Thirty-seven patients were studied at rest and during upright bicycle exercise: 11 controls (group 1) and 26 patients with significant (>70%) coronary artery stenosis, divided into those without lesions of the RCA proximal to the acute margin (group 2, n = 14) and those with proximal RCA lesions (group 3, n = 12).

In group 1, left ventricular ejection fraction (LVEF) increased from 0.68 ± 0.07 (SD) at rest to 0.76 ± 0.06 during exercise (p < 0.01), and RVEF increased from 0.47 ± 0.04 to 0.57 ± 0.06 (p < 0.01). In group 2, LVEF fell from 0.63 ± 0.13 to 0.58 ± 0.11 during exercise (p < 0.01), while RVEF increased from 0.49 ± 0.06 to 0.58 ± 0.09 (p < 0.01). The LVEF response to exercise in group 2 was significantly different from that in group 1 (p < 0.01); the RVEF response did not differ significantly. When group 2 was subdivided into patients without RCA lesions and those with distal RCA lesions, there was no significant difference in the RVEF response to exercise. In group 3, LVEF fell from 0.53 ± 0.17 at rest to 0.45 ± 0.14 during exercise (p < 0.01), while RVEF did not change significantly (0.50 ± 0.07 at rest and 0.46 ± 0.10 during exercise). The LVEF response to exercise in group 3 was not significantly different from that in group 2; however, the RVEF response differed significantly from that in groups 1 and 2 (p < 0.01).

Proximal RCA lesions are associated with a fall in LVEF and no change in RVEF during exercise, while coronary artery disease that does not involve the proximal RCA results in a fall in LVEF and a rise in RVEF. Thus, there can be a dissociation of the exercise response of the right and left ventricle, determined by the location of coronary artery lesions.

RADIONUCLIDE ANGIOGRAPHY has been shown to be a useful method for evaluating left ventricular function at rest and during exercise in patients with coronary artery disease.1-3 During myocardial ischemia induced by exercise, a fall in left ventricular ejection fraction (LVEF) and the appearance of regional wall motion abnormalities has been reported in patients with coronary artery lesions in studies using either first-pass or multiple-gated blood pool techniques.1-3 This method has also been used to measure right ventricular ejection fraction (RVEF) at rest in patients with recent myocardial infarction4, 5 and in patients with chronic obstructive lung disease.6 Radionuclide angiography is potentially useful for evaluating right ventricular function during exercise-induced ischemia in patients with coronary artery disease.

The right ventricle is composed of the free wall and the interventricular septum. Echocardiography has shown that the interventricular septum normally contributes to left ventricular ejection;7 therefore, the free wall of the right ventricle must make the major contribution to right ventricular ejection. Anatomic studies have indicated that an average of 60% of the anterior right ventricular free wall from the acute margin to the anterior interventricular septum is supplied by branches originating from the right coronary artery before the acute margin.8 Significant obstruction of the right coronary artery proximal to the acute margin may lead to inadequate blood supply to the free wall of the right ventricle during exercise and result in a fall in RVEF.

We tested the hypothesis that the location of right coronary artery lesions influences the RVEF response to exercise. RVEF and LVEF were measured by first-pass radionuclide angiography at rest and during upright bicycle exercise in control subjects and in patients with coronary artery disease. The RVEF response of patients with proximal right coronary artery lesions was compared with the response of patients without right coronary disease or with right coronary lesions distal to the acute margin.

Methods

Patient Selection

All patients with known or suspected coronary artery disease who underwent diagnostic coronary arteriography and ventriculography for clinical indications were candidates for the study. Patients with recent myocardial infarction (within 3 months), unstable angina or signs of pulmonary congestion at rest were excluded because of the risks of exercise. Informed written consent was obtained from all patients under a protocol approved by the Institutional Review Board, Health Sciences, Columbia University.

Coronary Arteriography and Ventriculography

Selective coronary arteriography was performed in multiple projections using a modified Judkins tech-
nique. The coronary arteriograms and left ventriculograms were interpreted by a cardiologist and a cardiovascular radiologist without knowledge of the results of radionuclide angiography. A patient was considered to have significant coronary artery disease if one or more of the major coronary arteries or branches was narrowed by at least 70% of its luminal diameter. In addition, patients were classified as having jeopardized right ventricular blood supply if there was a significant obstructive lesion of the right coronary artery before the acute margin. The classification represents a consensus between the two observers.

Study Population

The study population consisted of 37 patients (Table 1). The control group (group 1) consisted of 11 subjects, eight males and three females, with a mean age of 42 years (range 25–69 years). Three of the subjects had an atypical chest pain syndrome (nos. 1–3) two had normal coronary arteriograms and one had an insignificant LAD lesion. All had normal left ventriculograms and none were receiving propranolol. Eight healthy volunteers (nos. 4–11) did not undergo catheterization; all had normal physical examinations. ECGs and electrocardiographic stress tests were normal by the criteria of the American Heart Association,* except for subject no. 8, who was an asymptomatic pilot with a right bundle branch block.

Group 2 consisted of 14 patients with coronary artery disease that did not jeopardize the blood supply to the right ventricular free wall, i.e., they had no significant right coronary lesions proximal to the acute margin. There were 13 males and one female with a mean age of 51 years (range 34–68 years). The distribution of coronary artery lesions is shown in table 1. Seven patients had no significant right coronary artery lesions and seven patients had significant lesions of dominant right coronary arteries distal to the acute margin. Seven patients had ECG evidence of prior myocardial infarction (inferior in three, anterior in three and posterior in one). Eight patients were receiving propranolol at the time of the study.

Group 3 consisted of 12 patients with significant coronary artery lesions that jeopardized the blood supply to the right ventricular free wall, i.e., the lesions of the right coronary artery were proximal to the acute margin. There were 11 males and one female with a mean age of 58 years (range 38–67 years). All patients had dominant right coronary arteries. The locations of the right coronary lesions and the associated left coronary lesions are shown in table 1. Three patients had isolated lesions of right coronary arteries and nine had multivessel disease. Six patients had ECG evidence of prior myocardial infarction (inferior in five and anteroseptal in one). Seven patients in group 3 were receiving propranolol at the time of the study.

Radionuclide Angiography

Radionuclide angiography during upright bicycle exercise and at rest was performed by means of the first-pass technique using a multiple-crystal scintillation camera computer system (Baird-Atomic System 77). The detector of this instrument consists of a rectangular matrix of 294 individual NaI (T1) scintillation crystals equipped with a 1-inch, multichannel collimator. An 18-gauge Teflon catheter was inserted percutaneously into an antecubital vein and connected to an intravenous tubing filled with isotonic saline. The patient was seated on a bicycle ergometer (Quinton Instruments Model 862) and the scintillation camera detector was positioned parallel and anterior to the patient's chest. Blood pressure was measured with a sphygmomanometer. Lead CS, of the ECG was monitored continuously and recorded at 1-minute intervals using a Hewlett-Packard recorder. Exercise was begun at 0, 150 or 300 kilopond-meters (kpm)/min, depending on the physician's estimation of the patient's ability to exercise, and was increased in 150-kpm/min increments until the target heart rate (90% of predicted maximal heart rate for age) was attained or symptoms occurred. At this point 15 mCi of technetium-99m pertechnetate (99mTc) (Mallinckrodt, Inc.) dissolved in ≤1 ml of isotonic saline was injected rapidly into the indwelling catheter and followed with a flush of 15–20 ml of saline, while the patient continued to exercise for 1 minute. During the initial passage of the radionuclide through the heart and great vessels, counts were recorded at a 50-msec framing interval, using a 100–250 keV window encompassing the energy peak of 99mTc (140 keV). The data were stored on computer disc. The patient was allowed to rest in a supine position after exercise until heart rate and blood pressure returned to preexercise values (average time 20 minutes). Radionuclide angiography was then repeated in the postrecovery resting state with the patient seated on the bicycle ergometer. An appropriate background frame was acquired before the second 99mTc injection.

Data Processing

Using the system's dedicated computer, all data were corrected for heterogeneity of crystal efficiency by use of a uniform cobalt-57 pool; corrections for dead time of the instrument (2.4 μsec) were also introduced. The background frame obtained before the second (rest) injection was used to correct for background activity from the first injection. Each study was then replayed in serial fashion and displayed on the oscilloscope with each image representing 20 summed, 50-msec frames. The right and left ventricular phases were identified from the anatomic configuration, and the temporal appearance of the bolus dose in the central circulation and right and left ventricular regions of interest were identified.

RVEF was calculated by the method of Berger et al.⁶ Anatomic overlap of the right atrium and right ventricle in the anterior position results in the right atrial background contributing to the peak of the right ventricular time-activity curve. Correction for right atrial background was made and the RVEF was calculated on a beat-to-beat basis directly from the
**Table 1. Clinical Data and Ventricular Response to Exercise**

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age/sex</th>
<th>Cardiac catheterization</th>
<th>Significant (≥70%) coronary lesions</th>
<th>Ventriculogram</th>
<th>Resting ECG</th>
<th>Exercise</th>
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<td></td>
<td></td>
<td>Re</td>
<td>Ex</td>
<td>Re</td>
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</table>

**Group 1: controls**

1. 50/F  None  None  None  None  - - 64 68 55 55
2. 33/F  None  None  None  None  - - 78 81 47 64
3. 45/M  <50% LAD  None  None  - - 67 78 47 47
4. 41/M  *  *  None  - - 72 77 45 65
5. 35/M  *  *  None  - - 67 75 44 59
6. 28/M  *  *  None  - - 66 74 55 59
7. 42/M  *  *  None  - - 65 68 44 52
8. 69/M  *  *  RBBB  RBBB  - 69 78 46 60
9. 25/M  *  *  None  - - 71 81 45 55
10. 56/M  *  *  None  - - 74 86 43 62
11. 39/F  *  *  None  - - 52 65 45 51

**Group 2: coronary artery disease without proximal RCA lesions**

12. 63/M  LCF, RCA  Inf, Post  Inf  - + 59 61 46 52
13. 37/M  LAD  None  None  - + 68 61 60 71
14. 67/M  LCF  None  None  + - 82 66 43 60
15. 46/M  RCA  None  None  + + 73 72 56 64
16. 48/M  LAD  Ant  AS  + - 72 61 48 56
17. 58/M  LAD, RCA  Inf  Inf  + + 55 53 45 44
18. 52/M  LAD  Ant, Ap, Sept  AS  + + 43 43 53 66
19. 46/M  LCF, RCA  Inf  None  + - 60 57 41 60
20. 64/M  LCF  Post  Post  - - 66 59 52 59
21. 38/M  LAD, LCF, RCA  Inf  None  + + 68 72 52 55
22. 38/M  RCA  None  None  - + 75 67 47 69
23. 58/F  LAD  None  None  + + 75 58 45 36
24. 34/M  LAD  Ant, Ap, Sept  AS  - + 33 30 51 61
25. 68/M  LMCA, LAD, LCF, RCA  Inf  Inf  + + 53 50 42 61

**Group 3: coronary artery disease with proximal RCA lesions**

26. 45/M  RCA  None  None  + - 84 65 52 59
27. 59/M  LAD, LCF, RCA  None  None  + + 47 47 46 42
28. 56/M  LMCA, LAD, LCF, RCA  Ant, Ap, Inf  AL, Inf  - + 46 36 47 45
29. 67/M  LMCA, LAD, RCA  Inf  Inf  + + 50 36 49 43
30. 65/M  LAD, LCF, RCA  Inf  Inf  + + 41 31 67 54
31. 57/M  LMCA, LAD, RCA  Ant, Ap, Inf  Inf  + + 51 47 48 34
32. 63/M  LAD, LCF, RCA  Ant, Ap  AS  + + 27 26 45 46
33. 60/M  LAD, LCF, RCA  Inf  None  + - 47 46 44 42
34. 60/M  LAD, LCF, RCA  Inf  None  + - 79 64 59 66
35. 64/M  LMCA, LAD, LCF, RCA  Ant, Inf  None  + + 42 28 47 29
36. 38/M  RCA  None  None  - + 71 65 54 46
37. 57/F  RCA  Inf  Inf  + + 54 46 43 42

*Not done.

**Abbreviations:** WMA = wall motion abnormality; ST↓ = ischemic ST-segment depression; LVEF = left ventricular ejection fraction; RVEF = right ventricular ejection fraction; Re = rest; Ex = exercise; LMCA = left main coronary artery; LAD = left anterior descending coronary artery; LCF = left circumflex coronary artery; RCA = right coronary artery; Inf = inferior; Post = posterior; Ant = anterior; Ap = apical; Sept = septal; AS = anteroseptal; AL = anterolateral; RBBB = right bundle branch block; + = present; - = absent or negative.
final background-corrected, high-frequency time-activity curve using the formula

\[
RVEF = \frac{Cts_{ED} - Cts_{RS}}{Cts_{ED}}
\]

where \(Cts_{ED}\) = counts in an end-diastolic frame and \(Cts_{RS}\) = counts in the corresponding end-systolic frame. Only the beats at the peak of the right ventricular time-activity curve, with end-diastolic counts at least 70% of the maximal counts, were used; in general, one to three beats met this criterion. The final RVEF represents the numerical average of the data derived from all beats used.

LVEF was determined from the same radionuclide angiogram used to determine RVEF, using a modification of the method of Marshall et al.\(^{10}\) Summed end-diastolic and end-systolic frames were generated from the left ventricular time-activity curve. Sequential 50-msec background frames, corresponding to the number of cardiac cycles selected, were then chosen from the left ventricular time-activity curve just before the appearance of the radionuclide bolus in the left ventricle. This background represents overlying and scattered radiation from the left atrium and lungs. These background frames were summed and corrected for the decline in counts in the region around the heart during the transit of isotope through the body by multiplying by the ratio: \((\text{counts outside the left ventricle and aorta in summed ED frame})/(\text{counts outside left ventricle and aorta in summed background frame})\). This corrected summed background frame was subtracted from the summed end-diastolic frame derived from the individual cardiac cycles and displayed in analog mode. Using this background-corrected end-diastolic image, the initial zoning of the left ventricular region of interest was rechecked and adjusted if necessary. From the final left ventricular time-activity curve, the LVEF was calculated using the formula

\[
LVEF = \frac{Cts_{ED} - Cts_{RS}}{Cts_{ED} - \text{Background}}
\]

Only beats at the peak of the left ventricular time-activity curve, with end-diastolic counts at least 80% of the maximal end-diastolic counts, were used.

Statistical Analysis

Data for hypotheses relating to group differences in ejection fraction were analyzed using a multifactorial analysis of variance in which the ejection fractions at rest and during exercise for the left and right ventricle were considered to be repeated measures. Individual group differences were tested using Scheffe's procedure; differences with respect to the mean change in ejection fraction from rest to exercise for the left and right ventricle for groups 1, 2 and 3 were tested against the null hypothesis of no group differences. A separate analysis was performed comparing RVEF responses in the group 2 patients without right coronary artery lesions and with distal right coronary artery lesions. The relationship between the changes in LVEF and RVEF was tested by linear regression. In all cases, the null hypothesis was rejected when the test statistic exceeded the critical value for the 5% level.

Results

Group 1 — Controls

In the control subjects, the mean double product (heart rate \(\times\) systolic blood pressure) at peak exercise was \(28,000 \pm 4000\) (mean \(\pm\) sd). For the entire group, both LVEF and RVEF increased during exercise: the LVEF rose from \(0.68 \pm 0.07\) to \(0.76 \pm 0.06\) \((p < 0.01)\), and RVEF rose from \(0.47 \pm 0.04\) to \(0.57 \pm 0.06\) \((p < 0.01)\) (fig. 1). Three subjects in the control group had cardiac catheterizations because of symptoms of atypical chest pain. Two of these three patients (nos. 1 and 3) had no change in RVEF between rest and exercise. All subjects who were normal volunteers (nos. 4–11) had increased RVEF.

Group 2 — CAD Without Proximal RCA Lesions

The mean double product at peak exercise was \(18,000 \pm 4400\), which was significantly less than that in controls \((p < 0.01)\). LVEF fell from \(0.63 \pm 0.13\) to \(0.58 \pm 0.11\) \((p < 0.01)\), while RVEF increased from \(0.49 \pm 0.06\) to \(0.58 \pm 0.09\) \((p < 0.01)\). The LVEF response to exercise was significantly different from that of the controls \((p < 0.01)\); there was no significant difference in the RVEF response between group 1 and group 2. During exercise, all patients except one developed myocardial ischemia, defined as the occurrence of angina and/or horizontal or downsloping ST-segment depression \(\geq 0.1\) mV at the J point and lasting at least 80 msec. Patient no. 20, the one exception, had isolated circumflex disease and was only able to achieve a heart rate of 94 beats/min during exercise, and stopped because of leg fatigue.

In figure 2, the effect of the location of right coronary artery disease on the RVEF response is depicted in more detail. For this analysis, group 2 was further divided into patients without right coronary artery lesions (group 2A) and patients with distal right coronary artery lesions (group 2B). In group 2A, RVEF during exercise increased from \(0.50 \pm 0.06\) to \(0.58 \pm 0.11\) \((p < 0.01)\). All patients in this group had an increase in RVEF, except for patient no. 23, whose RVEF changed from 0.45 to 0.36; she had calcification of the proximal right coronary artery without a significant truncal stenosis and had abnormally few and small right ventricular branches. Patients with distal right coronary lesions (group 2B) also had a significant increase in RVEF during exercise, from \(0.47 \pm 0.05\) to \(0.58 \pm 0.08\) \((p < 0.01)\). There was no significant difference in the RVEF response to exercise between groups 2A and 2B.

Group 3 — CAD and Proximal RCA Lesions

The mean double product at peak exercise was \(17,000 \pm 3800\), which was significantly less than that
in group 1 ($p < 0.01$) but not significantly different from that in group 2. All patients became ischemic during exercise. During exercise, LVEF fell from $0.53 \pm 0.17$ to $0.45 \pm 0.14$ ($p < 0.01$), while RVEF during exercise ($0.46 \pm 0.10$) was not significantly different from that at rest ($0.50 \pm 0.07$; $0.05 < p < 0.10$). The LVEF response to exercise was significantly different from that of the controls ($p < 0.01$), but not significantly different from that in group 2. However, the RVEF response in group 3 was significantly different from that in both groups 1 and 2 ($p < 0.01$).

During exercise, three patients in group 3 had an increase in RVEF (nos. 26, 32 and 34) and three patients had a small decline (nos. 28, 33 and 37). Patients 26 and 34 had proximal occlusions of dominant right coronary arteries but had rich collateral supply reconstituting the right coronary artery back to the point of occlusion. The response of the other patients — either a small rise or fall in RVEF during exercise — was quantitatively different from the larger in-
crease in RVEF that occurred in the majority of patients in groups 1 and 2.

Figure 3 shows the right coronary arteriograms of individual patients from groups 2 and 3. Figure 3A shows the arteriogram of patient no. 22, who had an occlusion of the right coronary artery distal to the acute margin. In this patient, LVEF fell from 0.75 to 0.67 during exercise, while RVEF increased from 0.47 to 0.69. In contrast, figure 3B shows the arteriogram of patient no. 36, who had a proximal occlusion of the right coronary artery that compromised the blood supply to the right ventricular free wall. During exercise, ejection fractions of both ventricles fell, LVEF from 0.71 to 0.65, RVEF from 0.54 to 0.46.

**Figure 3.** Right coronary arteriograms from A) a patient in group 2 with distal occlusion of the right coronary artery (RCA) and B) a patient in group 3 with proximal occlusion of the RCA. See text for details.

**Relationship Between RVEF and LVEF**

The ejection fraction responses of the left and right ventricles were directionally similar within groups 1 and 3 (fig. 1). However, in group 2, LVEF fell significantly during exercise, while RVEF increased significantly. Using the data from all patients, there was not a significant correlation between the change in RVEF from rest to exercise and the change in LVEF ($r = 0.17$).

**Discussion**

Radionuclide angiocardiography allows simultaneous measurement of RVEF and LVEF and comparison of the functional reserve of both ventricles during exercise in patients with coronary artery disease.

Normal subjects increase their LVEF during exercise. This response has been shown by contrast ventriculography and by gated blood pool and first-pass radionuclide angiography. In our control group, LVEF increased from 0.68 to 0.76. A fall in LVEF has been reported during exercise in patients with coronary artery disease. All patients in groups 2 and 3 had an abnormal LVEF response to exercise, defined as either no change (<5% rise) or a fall in this measurement during exercise. In addition, all patients but one became ischemic and developed anginal pain or typical ST changes or both.

The proposed mechanism for the fall in LVEF during exercise in patients with coronary artery disease is the development of regional ischemia in the distribution of stenotic coronary arteries. The functional consequences of exercise-induced regional myocardial ischemia have been studied experimentally in animals. Studies in unanesthetized animals using ultrasonic crystals to measure segmental wall motion have shown that regional dysynergy occurs during exercise in the distribution of a partially constricted coronary artery, associated with a fall in global left ventricular function as measured by peak dP/dt.

Radionuclide angiography has also been applied to the study of RVEF. Validation of RVEF using contrast angiography is difficult because of the inherent difficulty in measuring right ventricular volume angiographically. Right ventricular shape does not conform to any simple geometric model. Simpson's rule has been the most commonly applied method for calculating right ventricular volume from cineangiograms. Gentzler et al. calculated right ventricular volume from postmortem casts of human hearts and compared the results with volume by water displacement. A regression equation was derived to apply to in vivo studies in man. In a group of nine normal adults, right ventricular volume was 81 ml/m², a value greater than the left ventricular volume, and RVEF was 0.51 ± 0.08, a value less than LVEF. Similar findings were reported in children by Fisher et al. Berger et al., using the first-pass technique and a multicrystal camera in 50 normal adults, found that RVEF was significantly lower than LVEF (0.55 ± 0.05 vs mean 0.69, range 0.58–0.70). Steele et al. found
similar results using radioisotope techniques. The results at rest in this study agree with these reports.

Theoretically, radionuclide techniques for determining RVEF eliminate some of the problems inherent in angiocraphic techniques, because ejection fraction is calculated from changes in counts and is not based upon geometry. One arbitrary assumption involved in the radionuclide calculation is the selection of the right atrial (background) region of interest. Although the absolute value for RVEF has not been validated, the method of calculation has been shown to be highly reproducible.6

We tested the hypothesis that the location of right coronary artery lesions influences the RVEF response to exercise. The blood supply to the free wall of the right ventricle comes predominantly from the right coronary artery. In the presence of a proximal critical stenosis of the right coronary artery, the blood supply to the right ventricle is limited and the functional reserve is diminished.

In the right ventricle, as in the left ventricle, performance is impaired in the distribution of the stenotic vessel when demand is increased. In the group with significant proximal right coronary artery lesions, there was no significant change in RVEF during exercise. In addition, all patients had abnormal LVEF responses to exercise.

In contrast, coronary artery disease patients without significant lesions proximal to the acute margin had a significant increase in RVEF and a simultaneous significant fall in LVEF. These are the first data that suggest a dissociation between the responses of the two ventricles during exercise in coronary artery disease. Moreover, there was no difference in the RVEF response to exercise between patients without significant right coronary disease and those with right coronary artery lesions distal to the acute margin. Thus, the location of the right coronary artery lesion affects the RVEF response to exercise.

In the control groups as a whole, the RVEF increased from 0.47 to 0.57; however, two subjects (nos. 1 and 3) had no change from rest to exercise. The control group was heterogeneous and consisted of three patients and eight normal volunteers. The two subjects with an abnormal RVEF response to exercise were patients who had cardiac catheterization for symptoms of atypical chest pain and may have occult myocardial disease.

The fall in RVEF during exercise in group 3 might be a consequence of both the limited right ventricular blood supply and the increase in pulmonary artery pressure that has been shown to accompany exercise-induced ischemia.16 More patients in group 3 had triple-vessel disease than in group 2, and perhaps with more left ventricular myocardium at jeopardy in these patients, an even larger increase in pulmonary artery pressure resulted in the fall in RVEF. However, even if an increase in pulmonary artery pressure contributed to the fall in RVEF in group 3 patients, an independent effect of their proximal right coronary artery lesions is also apparent, because the fall in LVEF in these patients with proximal right coronary artery disease and greater vessel involvement (group 3) was the same as that in patients without proximal right coronary artery disease and less extensive coronary artery disease (group 2).

In summary, the data from this study indicate that significant lesions of the proximal right coronary artery are associated with a fall in LVEF during exercise-induced ischemia, while RVEF does not increase. Coronary artery disease not involving the proximal right coronary artery is associated with a fall in LVEF and an increase in RVEF. These data show that there can be a dissociation of the exercise response of the right and left ventricles that is determined by the location of coronary artery lesions.

Acknowledgment

We express our appreciation to Dr. Catherine Cabot and Dr. Barry Rudin for their assistance in performing the exercise studies, to Minzer Tung for technical assistance, and to Kathleen Mulrenin for excellent secretarial work.

References


Appendix

To evaluate whether the differences in right ventricular ejection fraction were due to proximal right coronary artery disease (PR) or triple-vessel disease (TV), a multifactorial analysis of variance was performed. The factor criteria used were presence or absence of PR, presence or absence of TV, and right ventricular ejection fraction at rest and exercise, which was treated as a repeated measure. Table A 1 is a table of means. Table A 2 is the statistical table, which shows a significant effect for PR but not for TV. The interaction term of PR and TV is not significant.

This analysis supports the contention that observed effect is due to PR and not TV.

<table>
<thead>
<tr>
<th>Table A 2. Statistical Table</th>
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<td>Factors</td>
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</tr>
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*Terms of interest that test the change from rest to exercise for the classifying variables PR and TV.

Abbreviations: PR = proximal right coronary artery disease; TV = triple-vessel disease; MS = mean square; STATE = the repeated measure (right ventricular ejection fraction) at rest and exercise; unit = residual variation within subjects.

Table A 1. Table of Means

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</table>

Abbreviations: TV = triple-vessel disease; PR = proximal right coronary artery disease; R = rest; E = exercise.
Right ventricular ejection fraction during exercise in patients with coronary artery disease.
L L Johnson, D M McCarthy, R R Sciacca and P J Cannon

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