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Evolutionary changes in left and right ventricular function in acute myocardial infarction

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To determine the evolutionary changes in right and left ventricular function in acute myocardial infarction, 3 serial gated blood pool scans were performed in 76 patients within 24 hours (24 H), at 10 days (10 D) and 3 months (3 M) following the onset of myocardial infarction. The patients were divided into 3 groups: ANT (anterior MI), INF (inferior MI without right ventricular dysfunction) and RVF (inferior MI with right ventricular dysfunction). LVEF in ANT was significantly lower than that of INF and RVF at 24 H, 10 D and 3 M. The ratio of right ventricular volume to LV volume (RVV/LVV) was compared among 3 groups. The mean values of RVV/LVV in RVF were 1.3 through 24 H and 3 M and they were significantly higher than the other two groups. The RVV/LVV in ANT and INF were around 1.0. LVEDVI in RVF was rather smaller than that of ANT and INF. LVESVI in ANT at 24 H was significantly larger than that of INF and RVF and the mean value of LVESVI in ANT were around 60 ml/M^2 from 24 H to 3 M. LVEF in ANT, RVF and INF did not increase significantly during peak exercise at 3 M. However, quantitative regional wall motion analysis revealed that regional wall motion of R2 (posterolateral wall motion) in ANT and R5 (septal wall motion) in INF decreased significantly during peak exercise. These impairments in regional wall motion might be due to the exacerbation of ischemia of non-infarcted area.

Key words: Acute myocardial infarction, Left ventricular function, Right ventricular function, Regional wall motion

INTRODUCTION

THE EVOLUTION of right and left ventricular function following acute myocardial infarction is variable—some observers have reported improvement in left ventricular or right ventricular infarction,[1–3] while others have seen no change in function.[4–6] These differences in the evolution of ventricular function may be due to the time an initial measurement is made. Radionuclide ventriculography permits determination of ventricular function at the bedside in acutely ill patients.

This study was designed to determine the evolutionary changes of right and left ventricular function in patients with acute myocardial infarction, including functional reserve at three months following the onset of myocardial infarction.

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METHODS

1. Patient population

Radionuclide ventriculography was performed in 76 patients with acute myocardial infarction within 24 hours following the onset of myocardial infarction (24 H), at 10 days (10 D) and 3 months (3 M). Acute myocardial infarction was diagnosed when at least two of the following criteria were present:

(1) A history of typical prolonged chest pain;
(2) Development of pathologic Q waves 0.04 seconds in duration in at least two electrocardiographic leads;
(3) Characteristic serial elevation of serum enzymes (creatinine phosphokinase (CK), glutamic oxaloacetic transaminase (GOT), and lactic dehydrogenase (LDH)).

Patients with cardiogenic shock or on pressors, cardiomyopathy, advanced or terminal illness and previous MI within the preceding 2 weeks were excluded from the study. The mean age was 57 ± 10 (mean $\pm$ SD) years with a range of 21–80 years. The patients were divided into 3 groups according to the location of Q waves and ST elevation in ECG tracings taken immediately after admission. Patients with pathologic Q waves in lead V1 through V4 were diagnosed as anterior MI (ANT), and those in leads II, III, aVF were diagnosed as inferior MI. Extension of Q waves to I, aVL, V5 and V6 was classified as ANT or IN_F depending on whether there were associated abnormalities in V1–4 or II, III, aV_F. Strictly posterior MI was included in inferior MI. In cases with subendocardial MI, ST depression and T wave inversion were used as a marker of location. Patients with inferior MI were subdivided into those with impaired RV free wall motion (RVF) and those without such abnormality in the 24 H study of gated blood pool scanning. Six cases in ANT and one case in RVF were diagnosed as subendocardial MI. However, no case of such category was found in INF. Table 1 shows the distribution of patients in ANT, INF and RVF groups.

Coronary anatomy was studied in 27 patients during their acute admission. Significant stenosis was defined as equal to or greater than a 50% luminal narrowing of one or more major coronary artery branches. Informed consent was obtained in all patients before the study.

2. Data acquisition

A. Radionuclide Ventriculography[7,8]

In vivo red blood cell labeling was accomplished by administering 3 mg stannous pyrophosphate (Pyrolite, New England Nuclear, N. Billerica MA) intravenously followed 20 minutes later by 20 mCi of Technetium-99m pertechnetate. Multigated blood pool images were recorded supine both in the anterior projection and in the 45 degree left anterior oblique projection (LAO), which defined the septum best with a conventional Anger Scintillation camera equipped with an all-purpose, parallel hole collimator.[9–11]

For calibration of image size for subsequent volume analysis, a lead bar pattern was placed over a technetium-99m sheet source and an image was recorded. At 3M an exercise study was also obtained. The patient was positioned with supine shoulder restraints and hand grips after the rest scan was completed. The initial resistance on the bicycle ergometer was set to 25 watts (150 kpm) and exercise was done at a steady rate of 60 revolutions per minute. The work load was increased in 25 watt increments every 3 minutes. Two minute collections of data every 3 minutes were obtained until the maximum was reached defined as the occurrence of fatigue, anginal pain, ischemic ST-T changes, significant electrical disturbance, hypotension or fatigue. The data were translated and interpreted in a standardized fashion on a Medical Data System (MDS) A2 computer. All scans were reviewed by two observers who were unaware of the clinical data.

3. Data analysis

A. (LV) Ejection Fraction

Ejection fraction was calculated from the left anterior oblique (LAO) using a variable region-of-interest counts method. Ejection fraction values were the mean of two determinations by each observer. Previous studies in our laboratory have revealed that interobserver variance (± 2 standard deviation) for ejection fraction calculation was less than 6%.[8]

B. RV Score

To define free wall motion of the right ventricle semi-quantitatively, a five-point grading scale from 4 (normal) to 0 (dyskinesis) was applied to the right venticle by viewing an endless-loop movie of the multigated scan in the LAO projection.[12]

C. (RV) Ejection Fraction

In patients with impaired RV contraction, RV ejection fraction was calculated from the LAO view using Maddahi's method.[13] Two regions-of-interest (ROI's) over the right ventricle; one in the end-systolic and the other in the end-diastolic phase of the cardiac cycle, were assigned by joystick. The end-systolic left paraventricular region was selected for background. Ejection fraction was determined by dividing the background corrected stroke counts (end-diastolic counts—end-systolic counts) by the background-corrected end-diastolic counts.

D. LV Volume[7,9–11]

LV volume calculation and wall motion analysis

were performed independently by two experienced observers and the results were averaged. LV end-diastolic volume was derived from the anterior and left anterior oblique views using a geometric biplane area-length method.[14] Details about this method were previously reported.[7,9–11]

In our laboratory, a correlation between end-diastolic volume by scan and by contrast left ventriculography was 0.84; interobserver variance was 29 ml/m^2 ($\pm$2 standard deviation), and intraobserver variance was 8 ml/m^2.[9,10] Thus, we measured absolute end-diastolic volume using a biplane geometric area-length method that we have shown to have acceptable interobserver variance when the same observers analyze serial scans in which patients are used as their own control. Although the shape of the left ventricle usually approximates a prolate ellipsoid at end-diastole, the shape at end-systole may not conform to any predictable geometry. Therefore, end-systolic volume was calculated as EDV $\times$(1-EF).

This approach avoided geometric assumptions concerning the shape of the left ventricle in end-systole. All ventricular volumes were corrected for body surface area and expressed in milliliters per square meter. The values in normal control patients appeared to be somewhat lower than those reported for contrast left ventriculography, and therefore, the absolute values using this approach should probably not be compared with values derived using other methods.

RV end-diastolic volume was calculated similarly as LVEDV by the biplane area-length method.[15] However, absolute volume of RV could not be evaluated correctly, because geometric assumptions concerning the shape of the right ventricle were made.[16] So, the ratio of RVEDV/LVEDV (RVV/LVV) was only shown as a parameter of RV enlargement.[17]

E. Wall Motion Analysis

The problem of observer variability with subjective analysis of regional wall motion is well recognized. Therefore, a quantitation technique for assessing regional wall motion was applied to this study.[18] Left ventricular end-diastolic (ED) and end-systolic (ES) perimeters were defined by a second derivative automatic edge detection algorithm. After a rectangularly shaped area enclosing the left ventricle was selected (Fig. 1), the central area of the rectangle is searched for the center of brightness (brightest 9-pixels) which is used as a fiducial marker. The ED, ES images were filtered with a Chesler filter to yield an edge enhanced scene (Fig. 1) and another set of ED, ES images were produced with a low pass filter to eliminate high frequencies. A second derivative image was approximated by forming the difference between the low pass and the Chesler filtered images.

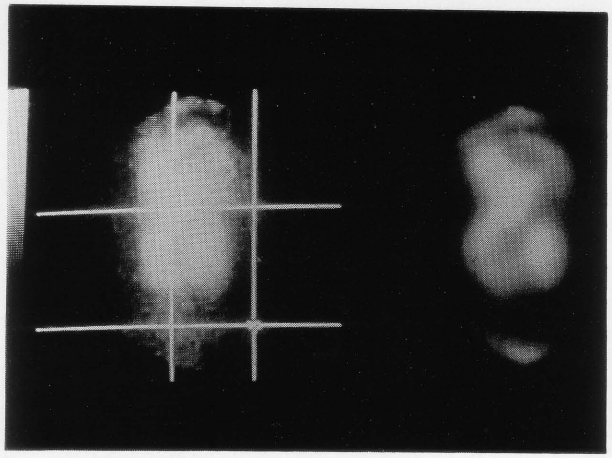

Fig. 1 Image filtering.
Left figure: Original gated blood pool scan image was shown with a rectangularly shaped area enclosing the left ventricle. Right figure: The left image was filtered with a Chesler filter to yield an edge enhanced scene.

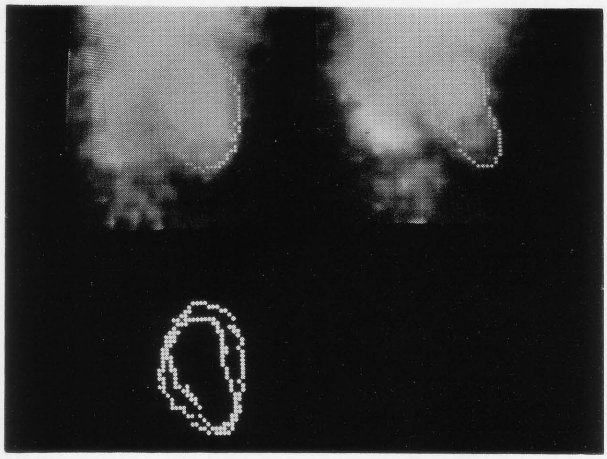

Fig. 2 Automatic edge detection.
Upper left: Automatically detected edge was superimposed on the original end-diastolic image. Upper right: Automatically detected edge was superimposed on the original end-systolic image. Lower left: End-diastolic and end-systolic edge points were superimposed on the CRT display.

Edge points were determined using a radial search procedure at 3 degree intervals (Fig. 2). The ventricular long axis was determined by edge fitting to an ellipse. Radial chords were drawn at every 10 degrees from the center of the long axis. Finally, regional wall motion as the average fractional change in 6 segments between ED and ES was computed (Fig. 3). A similar analysis was performed for the contrast ventriculograms. Overall correlation between quantitative wall motion analysis by scan and by contrast ventriculograms was r=0.75.

In eleven patients who were documented as normal by angiography, the heart was divided into

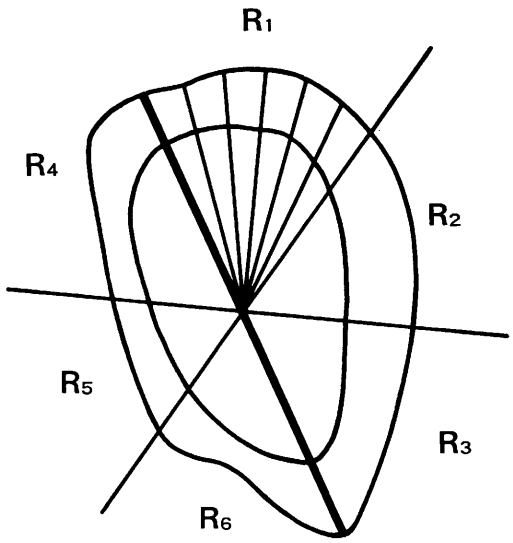

Fig. 3 Quantitative regional wall motion analysis. Radial chords were drawn at every 10 degrees from the center of long axis. Regional wall motion as the average fractional change in 6 segments between end-diastole and systole was computed. Abbreviations: R1=superior-lateral segment; R2=posterolateral segment; R3=inferolateral segment; R4=basal-septal segment; R5=apical-septal segment; R6=apical-inferior segment.

6 segments on the LAO view; R1 (superior-lateral), R2 (posterolateral), R3 (inferolateral), R4 (basal-septal), R5 (apical-septal) and R6 (apical-inferior) segments.

Results are shown in Table 2. While systolic radial shortening was of fairly uniform amplitude along the lateral segments, the septal surface had lower values compared to the lateral side ($p<0.05$).

Phantom studies were carried out using a balloon phantom which was suspended in a water tank. The edge finding algorithm was able to position the edge with an uncertainty of ± 1.7 mm (1 SD). At the values of EDV and ESV used in the phantom study of 60 and 150 cc respectively, the coefficient of variation for regional wall motion was 0.17. The precision and accuracy of the method determined by measurements on phantoms and reasonably high correlation between radionuclide and contrast ventriculograms warranted the capability of objective quantification of regional wall motion by this method.

4. Statistical Analysis

Differences between groups of patients for the parameter of global and regional ventricular function were compared by a one-way analysis of variance and the Newman-Keuls multiple comparison test. All values were expressed as mean$\pm$1 standard deviation.

RESULTS

1. Clinical and catheterization data

The summary of clinical and catheterization data was shown in Table 1. The radiologic findings of elevated left atrial pressure such as redistribution of blood flow to the upper lung zone (pulmonary venous congestion), interstitial and pulmonary edema were defined as signs of congestive heart failure. Patients in ANT had congestive heart failure more frequently than those with INF and RVF, whereas patients in INF and RVF had 1 or 2 vessel disease more frequently than ANT. Hypotension with bradycardia was present in 3 patients in RVF. Within three months following the onset of acute myocardial infarction, 4 patients in ANT were dead due to congestive heart failure (1 case), myocardial rupture (2 cases) and sudden death at home (1 case). However, only one patient in INF was dead due to cardiogenic shock and no death was reported in RVF within the same period. A total of 47 patients performed supine bicycle exercise at 3 months: 14 patients in ANT, 19 patients in INF and 14 patients in RVF.

2. RI angiographic findings at rest (Table 3)

RI sngiographic findings at rest are summarized in

Table 1 Distribution of patients

	ANT (n=27)	INF (n=27)	RVF (n=21)
Age (mean$\pm$1 SD)	58.7$\pm$8.2	54.8$\pm$11.3	59.4$\pm$8.1
Early death (within 3 months)	4	1	0
Chest X-ray film			
pulmonary edema	5	0	1
interstitial edema or pulmonary venous congestion	3	0	2
Hypotension with bradycardia	0	1	3
Ventricular tachycardia or flutter	1	2	1
Coronary anatomy studied	9	6	12
Left Main	1	0	0
3 vessel	6	1	2
2 vessel	1	3	6
1 vessel	1	2	4
CABG	2	0	2
3 month exercise studied	14	19	14

Abbreviations: ANT=anterior myocardial infarction; INF=inferior myocardial infarction without right ventricular dysfunction; RVF=inferior myocardial infarction with right ventricular dysfunction; CABG=coronary artery bypass-graft.

Table 2 Evolutionary changes in regional wall motion

	Normal (n=12)	ANT (n=23)	INF (n=25)	RVF (n=20)
R1	39±13			
24 H		27.7±14.2	33.3±14.3	28.3±10.6
10 D		25.6±12.1	34.1±17.3 †	37.4±21.4
3 M		24.4±14.3	31.7±13.1	43.0±13.5
R2	42±12			
24 H		26.3±13.7	28.1±10.9	24.9±7.0
10 D		27.1±13.5	31.3±15.5	33.6±22.0
3 M		27.7±13.6	30.7±11.9	31.5±11.3
R3	40±12			
24 H		18.1±11.3	25.1±14.6	24.0±12.2
10 D		23.2±15.6	28.9±17.4	33.1±22.3
3 M		23.8±11.9	32.3±12.8	27.5±8.4
R4	24±10			
24 H		18.4±18.6	32.6±16.6*	28.8±12.3
10 D		16.4±10.7	27.5±16.4	28.7±13.9*
3 M		20.5±13.3	27.3±11.7	30.1±9.6
R5	20±10			
24 H		16.8±18.3	27.3±14.7	24.4±10.6
10 D		16.9±17.2	27.2±16.6	22.1±12.3
3 M		19.6±14.5	24.9±10.5	16.9±10.9
R6	34±11			
24 H		20.8±11.0	27.4±15.2	23.7±13.2
10 D		21.1±15.4	28.8±17.1	29.6±22.1
3 M		21.9±13.4	30.3±11.5	18.9±14.2‡

Quantitative wall motion analysis was done on the LAO view. Values are expressed in percent as mean±1 standard deviation. They were compared by one-way analysis of variance and the Newman-Keuls multiple comparison test.

*$p<0.05$, compared with ANT value, †$p<0.01$, compared with ANT value, ‡$p<0.05$, compared with INF value.

Abbreviations: ANT=anterior myocardial infarction; INF=inferior myocardial infarction without right ventricular dysfunction; RVF=inferior myocardial infarction with right ventricular dysfunction; 24 H=values obtained from the study performed within 24 hours following the onset of myocardial infarction; 10 D=values obtained from the study performed at 10 days following the onset of myocardial infarction; 3 M=values obtained from the study performed at 3 months following the onset of myocardial infarction.

Table 3 RI angiographic findings at rest

	Anterior MI (n=27)	Inferior MI without RV dysfunction (n=27)	Inferior MI with RV dysfunction (n=21)
Age (mean±1 SD)	58.7±8.2	54.8±11.3	59.4±8.1
RV Score			
24 H	3.7±0.5	3.5±0.5	† 1.8±0.4‡
10 D	3.9±0.3	3.8±0.4	† 2.8±0.6‡
3 M	3.6±0.8	3.8±0.4	† 3.2±0.7
PSP (mmHg)			
24 H	134.5±14.7	131.0±20.4	132.3±22.1
10 D †	119.9±15.7	119.1±10.9	* 115.6±16.6
3 M	132.9±20.9	127.6±16.4	131.5±20.1
LVEF (%)			
24 H	36.9±10.2	58.6±11.1‡	56.2±11.9‡
10 D	37.2±10.3	54.5±12.4‡	57.9±13.2‡
3 M	40.7±10.2	55.7±13.4†	53.2±13.3†
RVEF (%)			
24 H	—	—	‡ 31.7±12.9
10 D	—	—	‡ 44.9±10.2
3 M	—	—	45.1±12.0
RVV/LVV			
24 H	1.0±0.2	1.0±0.2	1.3±0.4‡
10 D	0.9±0.2	1.0±0.3	1.3±0.6†
3 M	0.8±0.2	1.0±0.2	1.3±0.5*
LVEDVI (ml/m²)			
24 H	97.1±48.8	79.2±19.7	74.6±18.3
10 D	90.1±42.1	91.4±20.9	79.1±21.4
3 M	102.0±37.8	78.7±19.3	88.1±23.9
LVESVI (ml/m²)			
24 H	61.6±38.2	33.9±13.6‡	33.3±13.1‡
10 D	57.9±31.2	43.3±17.7	34.8±17.3
3 M	61.7±28.8	38.7±11.9*	43.5±19.1

Abbreviations: PSP=peak systolic blood pressure; LVEF=left ventricular ejection fraction; RVEF=right ventricular ejection fraction; RVV/LVV=right ventricular end-diastolic volume/left ventricular end-diastolic volume; LVEDVI=left ventricular end-diastolic volume index; LVESVI=left ventricular end-systolic volume index; 24 H=values obtained from the study performed within 24 hours following the onset of myocardial infarction; 10 D=values obtained from the study performed at 10 days following the onset of myocardial infarction; 3 M=values obtained from the study performed at 3 months following the onset of myocardial infarction.

*$p<0.05$, compared with Anterior MI value.

†$p<0.01$, compared with Anterior MI value.

‡$p<0.001$, compared with Anterior MI value.

Values are expressed as mean±1 standard deviation.

Table 3. In patients with inferior MI with RV dysfunction, RVEF at 24 H (31.7±12.9%) recovered significantly at 10 D (44.9±10.2%) ($p<0.001$). However, RVEF at 3 M was 45.1±12.0%. There were no significant changes in LVEF, RVV/LVV, LVEDVI and LVESVI.

LVEF in anterior MI at 24 H was significantly lower ($p<0.001$) than that of INF and RVF, and it remained significantly below the INF and RVF values at 10 D and 3 M. Inferior MI with RV dysfunction had significantly higher RVV/LVV ratios (1.3±0.4) ($p<0.001$) than that of ANT (1.0±0.02), which might be induced by enlargement of RVEDV. In addition, RVV/LVV ratios in RVF at 10 D and 3 M were also maintained at 1.3.

3. RI angiographic findings during exercise

RI angiographic findings during exercise were shown in Tables 4A and 4B. There were no significant differences in heart rate, PSP, the rate-pressure product, maximum work load and duration of exercise among ANT, INF and RVF.

LVEF in ANT (41.9±11.0%) was lower than those of INF and RVF both at rest ($p<0.05$) and during peak exercise ($p<0.05$). LVEF in INF were 54.1±9.3% at rest, and 57.2±12.4% during peak exercise. LVEF in RVF were 52.1±10.6% at rest and 55.9±13.9% during peak exercise (p=ns).

Table 4A RI angiographic findings during exercise

	ANT (n=14)	INF (n=19)	RVF (n=14)
Maximum exercised load (watt)	86.7±31.2	81.9±24.0	85.4±32.8
Duration of exercise (min)	9.1±3.3	9.8±2.9	9.1±3.6
HR			
Rest	‡ 68.4±6.4	‡ 65.7±11.1	‡ 73.4±11.1
Peak exercise	‡ 117.6±21.0	‡ 111.4±16.8	‡ 125.6±21.3
PSP (mmHg)			
Rest	‡ 133.8±18.0	‡ 127.4±17.5	‡ 140.4±21.0
Peak exercise	‡ 173.3±25.4	‡ 172.4±29.4	‡ 193.1±28.7
SBP×HR			
Rest	‡ 9,100±1,470	‡ 8,447±2,246	‡ 1,002±2,613
Peak exercise	‡ 20,500±6,530	‡ 19,531±6,397	‡ 23,898±5,699
LVEF (%)			
Rest	41.9±11.0	54.1±9.3*	52.1±10.6*
Peak exercise	42.1±15.7	57.2±12.4	55.9±13.9*

*$p<0.05$, compared with ANT.
‡$p<0.001$, compared with ANT.
Values are expressed as mean±1 standard deviation.
Abbreviations are the same as Table 1 and 3.

Table 4B Changes in regional wall motion during exercise

	ANT (n=13)	INF (n=19)	RVF (n=11)
R1			
Rest	34.6±12.2	30.7±11.3	30.9±10.2
Peak exercise	26.8±21.4	31.4±13.3	33.0±12.8
R2			
Rest	† 37.7±10.3	31.5±8.1	25.8±13.3
Peak exercise	† 28.0±11.9	29.0±12.4	29.2±13.6
R3			
Rest	24.2±13.1	31.5±8.2	20.1±15.1
Peak exercise	20.8±9.1	26.1±12.7	21.6±12.3
R4			
Rest	23.6±16.4	31.6±13.4	32.9±8.3
Peak exercise	21.1±13.1	30.8±11.0	33.5±16.8
R5			
Rest	13.7±14.8	† 32.4±8.5‡	27.3±11.3*
Peak exercise	9.7±16.0	† 18.7±9.7	24.2±13.2
R6			
Rest	17.3±11.0	29.5±10.1*	21.4±11.1
Peak exercise	14.2±10.0	26.5±11.3	18.9±14.6

*$p<0.05$, compared with ANT.
†$p<0.01$, compared with ANT.
‡$p<0.001$, compared with ANT.
Values are expressed as mean±1 standard deviation.
Abbreviations are the same as Table 1 and Fig. 3.

Regional wall motion on the LAO projection was analyzed quantitatively at 24 H, 10 D, and 3 M (Table 2). Radial chord shortening in R1 recovered significantly ($p<0.01$) from 24 H to 3 M in patients with RVF. However, no significant recovery was found in the other groups.

Regional wall motion was also analyzed quantitatively both at rest and during peak exercise at 3 M. In ANT, radial chord shortenings in R1 ($34.6\pm12.2\%$), R2 ($37.7\pm10.3\%$), R4 ($23.6\pm16.4\%$) were almost normal at rest (normal values in R1, R2 and R4 were 39 ± 13, 42 ± 12 and $24\pm10\%$), and those in R5 ($13.7\pm14.8\%$) and R6 ($17.3\pm11.0\%$) were significantly lower ($p<0.001$ and $p<0.05$, respectively) than that of INF (32.4 ± 8.5 and $29.5\pm10.1\%$, respectively). However, radial chord shortening of R2 in ANT decreased significantly ($p<0.01$) during peak exercise ($28.0\pm11.9\%$) and the value of R5 radial chord shortening during peak exercise in INF ($18.7\pm9.7\%$) was significantly low ($p<0.05$) compared to the rest value.

DISCUSSION

1. RI angiographic findings at rest

LVEF in ANT, INF and RVF did not show significant improvement as a whole. However, as reflected in the value of RV score, RVEF improved significantly from AC to 10 D. It is possible that improvement in RVEF might be related to the recovery of ischemic lesion located around the infarcted muscle in RV or early recovery of infarcted muscle in RVMI. RVV/LVV in RVF was significantly higher than those of ANT and INF from 24 H to 3 M.[17] This ratio might be increased in RVMI due to the enlargement of RV size,[19] although the size of LV was rather small in RVMI compared to ANT and INF. Incidence of hypotension in RVF was not so rare. The mechanism of hypotension might be partly due to vasovagal reflex, but the marked enlargement and impaired contraction of RV could disturb the inflow of LV across the mitral valve. Protrusion of interventricular septum into LV cavity might also decrease the size of LV. Regional wall motion of RV improved already at 10 D after the onset of MI and increased the preload of LV. However, the size of RV still remained large at 3 M and might make RVV/LVV ratio constant. So, the volume challenge to shock in RVMI might often be necessary before 10 days after onset of MI.[20,21] Thereafter, enlarged RV size and improved contraction of RV might maintain the preload of LV within a resonable range.

2. RI angiographic findings during exercise

As reported previously, the mean EF value in normal subjects increased significantly during peak exercise.[10] However, it did not increase significantly, or decrease in patients with CAD during maximal exercise. The present observations are in agreement with the results of the other authors.[22,23] Regional wall motions were already impaired at rest, septal wall motion as reflected in R4, R5 and F6 in anterior MI and posterolateral wall motion as reflected in R2 in inferior MI. In addition, posterolateral wall motion in anterior MI and septal surface wall motion in inferior MI without RVF decreased significantly during peak exercise. Such changes in wall motion might be due to induced ischemia in a noninfarcted area perfused by the other coronary arteries which might have a significant stenosis at proximal portion. In patients with inferior MI who had RV dysfunction initially, regional wall motion in apical-septal area (R6) was relatively low at rest and peak exercise. This might be explained by the fact that the extension of inferior MI to posterior septum was not so infrequent. RV volume overload as showed in increased RVV/LVV ratio in RVF and sometimes combined tricuspid regurgitation[24] might disturb the septal wall movement toward posterolateral wall during systole. Although the EF value did not change significantly during supine bicycle exercise, abnormal functional reserve in regional wall motion was detected by quantitative regional wall motion analysis.

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