R WAVE CHANGES IN PATIENTS WITH MYOCARDIAL INFARCTION DURING TREADMILL STRESS TESTING

TOSHIKATSU SADA, M.D., KUAN-MIN SU, M.D., NAOMI AMANO, M.D.
NAOKI HAYASHI, M.D., INEKO TAWARA, M.D.
AND MASAYA TAKEUCHI, M.D.

A graded multistage treadmill exercise test was performed on 78 patients with myocardial infarction (65 males and 13 females, ranging in age from 30 to 76). The amplitude of the R wave in CC₅ was measured at rest and during the periods of peak exercise. The exercise-induced R wave changes were classified into 3 groups: increased, 39.1%; no changes, 39.7%; and decreased, 22.2%. Acute attacks of myocardial infarction were more severe in the patients whose R wave decreased during exercise than in those whose R wave increased. Heart rate, blood pressure and pressure rate products were not different among the groups. However, oxygen consumption was greater in the R-increased group than in the R-decreased group. Therefore, an increase in R wave amplitude during exercise in post-myocardial infarction patients indicates a good efficiency of the myocardium and skeletal muscles.

Exercise-induced changes of the mean electrical axes of the transverse plane were significantly related to changes of the R wave amplitude in CC₅ (r = 0.50, p < 0.001). Therefore, change in the transverse plane axis is one of the important determinants of exercise-induced R wave changes.

Exercise-induced changes in R wave amplitude have recently received a great deal of attention in the diagnosis of ischemic heart disease. Some investigators¹⁻³ have reported that an increase in R wave amplitude due to exercise enhances the diagnostic sensitivity and specificity. There are also some studies⁴⁻⁵ on the relationship between exercise-induced R wave changes and hemodynamic parameters of left ventricular function. Kentala et al.⁶ have reported that the patients with an increased R wave amplitude during exercise showed a good response to physical training. However, the value of exercise-induced R wave changes in the evaluation of the physical capacity of patients with ischemic heart disease has not been studied. Therefore, we evaluated patients with myocardial infarction to compare R wave changes and physical capacity.

MATERIALS AND METHODS

Subjects: Seventy-eight patients with well-documented myocardial infarction were subjected to treadmill stress testing from June 1979 to June 1980 to evaluate their cardiac and physical functional capacities. Sixty-five of the patients were men and 13 were women, the mean age being 60.3 (range, 30 to 76). There were 51 cases of anterior infarction and 27 of inferior infarction. The interval between an acute attack and exercise testing ranged from one month to 50 months.
### TABLE 1 PATIENT CHARACTERISTICS AND RESPONSE TO EXERCISE

<table>
<thead>
<tr>
<th></th>
<th>Group I</th>
<th>Group II</th>
<th>Group III</th>
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<tbody>
<tr>
<td><strong>Age</strong></td>
<td>54.7 ± 11.6</td>
<td>63.2 ± 9.8</td>
<td>65.6 ± 5.9</td>
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<tr>
<td><strong>Peel’s prognostic index</strong></td>
<td>6.6 ± 4.5</td>
<td>8.6 ± 4.6</td>
<td>10.4 ± 5.5</td>
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<tr>
<td><strong>Intervals after infarction (months)</strong></td>
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<tr>
<td></td>
<td>15.7 ± 12.6</td>
<td>14.9 ± 13.3</td>
<td>10.5 ± 9.1</td>
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<tr>
<td><strong>Heart rate (*/min)</strong></td>
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<tr>
<td>Rest</td>
<td>75.5 ± 13.9</td>
<td>78.0 ± 10.5</td>
<td>80.7 ± 12.9</td>
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<tr>
<td>Exercise</td>
<td>130.7 ± 17.0</td>
<td>134.0 ± 15.1</td>
<td>131.6 ± 12.1</td>
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<tr>
<td><strong>Systolic blood pressure (mmHg)</strong></td>
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<tr>
<td>Rest</td>
<td>124.1 ± 16.0</td>
<td>129.6 ± 16.7</td>
<td>128.4 ± 17.7</td>
</tr>
<tr>
<td>Exercise</td>
<td>170.9 ± 19.8</td>
<td>166.3 ± 18.6</td>
<td>165.9 ± 10.8</td>
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<tr>
<td><strong>Pressure rate product (×10³)</strong></td>
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<tr>
<td>Rest</td>
<td>9.9 ± 2.0</td>
<td>10.1 ± 2.0</td>
<td>10.2 ± 2.5</td>
</tr>
<tr>
<td>Exercise</td>
<td>22.4 ± 4.0</td>
<td>21.9 ± 4.0</td>
<td>21.4 ± 2.7</td>
</tr>
<tr>
<td><strong>Oxygen consumption (METs)</strong></td>
<td>5.7 ± 1.4</td>
<td>4.6 ± 1.1</td>
<td>4.3 ± 0.9</td>
</tr>
<tr>
<td><strong>Respiratory rate (*/min)</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Exercise</td>
<td>27.8 ± 5.2</td>
<td>27.8 ± 6.2</td>
<td>30.0 ± 8.3</td>
</tr>
<tr>
<td><strong>Tidal volume (ml)</strong></td>
<td></td>
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<tr>
<td>Exercise</td>
<td>107.8 ± 27.2</td>
<td>86.8 ± 40.5</td>
<td>79.1 ± 20.1</td>
</tr>
<tr>
<td><strong>Σ Ex. Stage × Durat.</strong></td>
<td>20.7 ± 7.3</td>
<td>15.6 ± 6.8</td>
<td>11.5 ± 6.9</td>
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*Σ Ex. Stage × Durat. = sum of the products of exercise stage and duration in minutes.*

*p < 0.05, **p < 0.01

**Exercise Testing:** Every patient was given graded treadmill exercise test using a modification of Bruce’s protocol. An appropriate initial workload was chosen for each patient so that the exercising could be terminated within 10 min to avoid stopping the exercise test due to fatigue of the lower extremities. Exercising was continued in each patient until 85% of the age-predicted maximum heart rate was reached or until one of the following signs or symptoms appeared: anginal pain, dyspnea, severe fatigue, serious arrhythmia, 2 mm of horizontal or downsloping ST depression or a falling systolic blood pressure.

Electrocardiograms of bipolar leads CC2 and CC5 were recorded in a standing position before exercising and during exercising. The blood pressure was measured by a cuff-sphygmomanometer every minute throughout the procedure. The volume and contents of oxygen and carbon dioxide components of expired gases were measured continuously with an Expired Gas Analyser 1H02-4 (San-ei Instrument, Tokyo). The oxygen consumption, respiratory rate, and tidal volume were calculated at rest and during peak exercising.

The R wave amplitude was measured from the isoelectric line to the peak of the R wave in mm for an average of 10 consecutive beats in CC5. The measurement was made with the patients in a standing position before and during the period of peak exercise. Fifteen of the 78 patients were excluded from...
the test due to a QS pattern in lead CC₅ or an inadequate CC₅ recording, or because they could not exercise more than 2 min.

The unpaired t-test was used for statistical analysis.

RESULTS

We considered that any exercise-induced R wave amplitude changes of 10% or more were significant. Twenty-four patients (39.1%) showed an increasing R wave amplitude of CC₅ (Group I), 25 patients (39.7%) showed no changes (Group II), and 14 patients (22.2%) showed a decreasing R wave amplitude (Group III).

Forty of the 63 patients (63.5%) stopped the exercise test because they had reached the heart rate limit of 85% of the predicted maximum heart rate. Fourteen of the remaining 23 patients (22.2%) stopped because of ST depression, 8 (12.7%) because of fatigue or dyspnea and one (1.6%) because of angina pectoris. The distribution of the end-points in these 3 groups was not significantly different.

The clinical characteristics and test results of the 3 groups are shown in Table I. The Group I patients were younger than those of Groups II and III. Peel’s prognostic indexes⁸ of Groups I, II and III at the time of an acute attack were 6.6, 8.6 and 10.4, respectively. There was a significant difference between Group I and Group III (p < 0.05).

Heart rate, blood pressure and pressure rate product were basically similar in all 3 groups (Table II). However, the oxygen consumption (METs) of the 3 groups was 5.7, 4.6 and 4.3, respectively. The Group I patients had a significantly higher oxygen consumption than did those of Groups II and III (Table II). The sum of the products of exercise stage and exercise duration (in minutes) of Groups I, II and III was 20.7, 15.6 and 11.5, respectively. There were significant differences between Group I and Group II or III (Table II).

The mean QRS axis of the transverse plane (β) was calculated assuming that the electrical axes of CC₂ and CC₅ are perpendicular. The β at rest and at peak exercise was not significantly correlated to the exercise-induced changes of the R wave amplitude (ΔR), but the changes of β due to exercise (Δβ) were significantly correlated to ΔR (r = 0.50, p < 0.001, Fig. 1). The correlation coefficients between ΔR and Δβ were 0.60 in anterior infarction and 0.42 in inferior infarction.

The interval between the exercise test and myocardial infarction was not significantly different among the groups.

The respiratory rate was very similar among the groups. The tidal volume during peak exercise was 107.8, 86.8 and 79.1 ml, respectively. There was a significant difference between Group I and Group III. However, there were no significant correlations between the respiratory rate (or tidal volume) and the R wave amplitude, ΔR and Δβ.

DISCUSSION

Oxygen consumption (METs) of Groups I, II and III was 5.7, 4.6 and 4.3, respectively. The patients with an increasing R wave amplitude (Group I) had a greater oxygen consumption than did the patients whose R wave amplitude showed no changes (Group II) or decreased (Group III). The sums of the products of the exercise stage and duration of the 3 groups were 20.7, 15.6 and 11.5, respectively, and was larger

Fig.1. Changes of R wave amplitude and mean transverse plane QRS axis due to exercise. ΔR, Δβ = the differences of the R wave amplitude of CC₅ and the mean QRS axis of the transverse plane between peak exercise and rest, respectively. The correlation coefficients between ΔR and Δβ in all cases, anterior infarction and inferior infarction were 0.50, 0.60 and 0.42, respectively.

Japanese Circulation Journal Vol. 47, September 1983
in Group I than in Groups II and III. Therefore, the physical capacities of the 3 groups were good in the order of Groups I, II and III. The functional capacity of patients with myocardial infarction is affected by the amount of time which has elapsed after an acute attack. In this study, however, the interval after an attack was not significantly different statistically among the 3 groups, and there was no significant correlation between the interval after myocardial infarction and functional capacity. Therefore, the differences in functional capacity among the 3 groups were not due to differences in the interval after myocardial infarction.

The pressure rate product, which reflects myocardial oxygen consumption, was the same in the 3 groups. However, it was difficult to conclude that there were no differences in myocardial function because many patients (63.5%) stopped exercising when they reached 85% of the predicted maximum heart rate rather than the maximum heart rate. Group III had a significantly larger Peel's prognostic index than did Group I. Therefore, it is reasonable to assume that patients showing a decreasing R wave during exercise had a more severe myocardial infarction. Despite the same myocardial oxygen consumption, Group I patients were able to do more physical work than were Group III patients. This means that the myocardium and skeletal muscles of Group I patients had a better mechanical efficiency. Kentala et al. have reported that ischemic heart disease patients who later showed a good response to physical training exhibited an increasing R wave amplitude during exercise, and that the myocardial infarction patients, who died but not suddenly during a 6-year follow-up period, showed no changes in the R wave amplitude during exercise. They have suggested that the absence of R wave amplitude changes was related to a decreased hemodynamic capacity of the left ventricle.

It is generally agreed that the amplitude of the R wave decreases during exercise in normal subjects but there is no unanimity of opinion concerning exercise-induced R wave changes in coronary heart disease. Some investigators have reported that patients with ischemic heart disease showed an increase in the R wave amplitude in V5 and that the diagnostic sensitivity and specificity of stress testing could be significantly improved by using R wave changes. On the other hand, Wagner et al. have studied exercise-related R wave changes in coronary heart disease and reported that R wave criteria had a sensitivity of 52% and a specificity of 63%. These values were inferior to the 88% sensitivity and 72% specificity of ST segment criteria in the same group of patients. They concluded that R wave criteria were of little value for diagnostic exercise testing.

In our study, the Group I patients could work harder and consumed more oxygen than those of Groups II and III, that is, the patients with a good functional capacity showed an R wave increase. The results of exercise testing on old myocardial infarction do not support the view that the R wave increases due to exercise in coronary heart disease. Bonorisi and coworkers have reported that the R wave in V5 increases in patients with coronary heart disease. They also reported that 46% of the patients with coronary heart disease had exercise-induced R wave increases, but the frequency of R wave increases in the patients with myocardial infarction was smaller, especially in those having anterior infarctions (15%). Berman et al. have reported the usefulness of R wave criteria, but only 15% of their patients were myocardial infarction patients. However, 36% of Wagner's patients were myocardial infarction patients. We suggest that exercise-induced R wave changes are affected regardless of whether or not patients have had a previous myocardial infarction. Therefore, when the R wave criteria are used in the diagnosis of myocardial ischemia, a history of myocardial infarction must be taken into account.

The mechanism of exercise-induced changes in R wave amplitude has not been precisely elucidated. Bonorisi et al. have postulated that an increasing R wave due to exercise in ischemic heart disease is due to the Brody effect. During acute ischemia, the left ventricular end-diastolic volume increases, and an increase of intraventricular volume is reflected by an increasing R wave amplitude. However, it remains to be proved that an increasing R wave amplitude during exercise is related to this theory, which is based on a hypothetical model. Battier et al. have reported that there is no significant correlation between exercise-induced R wave changes and left ventricular volume.

Respiration becomes rapid and deep during exercise, and the respiratory effects on electrical impedance and heart position is great. We did not record an electrocardiogram during hyperventilation. The respiratory rate during the
peak exercise period was 28–30/min and the heart rate during this time was 130–134/min, and thus the interval during 10 consecutive beats corresponded to 2 respiratory cycles. We expressed the height of the R wave for an average of 10 consecutive beats when the effect of the different respiratory phases on R wave measurement could be avoided. There was no significant correlation between the respiratory rate and tidal volume during peak exercising and the R wave amplitude and the mean transverse plane electrical axis or between their changes induced by exercise (ΔR, Δβ). It is not likely that R wave changes during exercise is affected by respiration.

Watanabe et al.18 recently studied vectorcardiograms during exercise. They concluded that the etiology of R wave changes due to exercise is mainly due to shifts in the QRS vector loops rather than to changes of spatial magnitude, but the reason for these shifts is unknown. In normal subjects, the QRS loops of the transverse plane shifted to a posterior direction and the R wave amplitude in lead X decreased. In some of the patients with ischemic heart disease, the transverse plane QRS loops showed an anterior shift and the R wave in lead X increased. We calculated the mean QRS axis in the transverse plane from CC2 and CC5. Precisely speaking, the axes of the two leads were not perpendicular, but we think the mean QRS axis could be roughly determined. There was a significant positive correlation between the Δβ and the ΔR, and the mean QRS axis rotated anteriorly during exercise. The R wave in CC5 increased. The correlation coefficient between ΔR and Δβ was greater in anterior infarctions (r = 0.60) than in inferior infarctions (r = 0.42). Therefore, Δβ may be partly affected by necrosis in the anterior portion of the ventricular wall or by exercise-induced ischemia in the anterior wall, but there was no correlation between the ST depression in CC5 and the Δβ. The mechanism of QRS axis shift in the transverse plane was not clear.

REFERENCES


Japanese Circulation Journal Vol. 47, September 1983

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