Post Exercise P Wave Changes; Correlation with ST-T

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The original purpose of the exercise test was to evaluate clinically equivocal cases of angina pectoris by observing ST-T changes occurring in left ventricular leads during or after exercise. This method was then used to diagnose asymptomatic cases of coronary artery disease. In recent years, it was further extended to evaluate the cardiac reserve in the rehabilitation of post myocardial infarction and to determine how much physical activity was to be allowed.

The purpose of the exercise tests differed from time to time and in different attempts, but many investigators emphasized that the ST-T changes in V₄ to V₆ was evidence of left ventricular ischemia caused by coronary artery disease. The author does not question the importance of ST-T changes occurring coronary artery disease, however, we must also realize that the observation of ST-T does not always give satisfactory answers. For example, those who already showed ST-T changes in electrocardiograms at rest, as in the cases of left ventricular hypertrophy or left bundle-branch block, tended to show no further alteration after exercise, despite the fact that the patients might have angina pectoris. Furthermore, pure mitral stenosis without any evidence of coronary artery disease often showed dramatic ST-T changes after exercise which was difficult to differentiate from coronary artery disease. In addition, ST-T changes observed in mitral stenosis may disappear with the appropriate use of cardiac glucosides. It was therefore assumed that changes in ST-T were due to left ventricular ischemia caused by left ventricular failure and not by coronary artery disease.

The observation of the P wave in V₁ in cases of congestive heart failure has been known to correlate with the hemodynamic state of the left ventricle and reflects left atrial pressure. It is believed that this P wave in V₁ may be a useful finding in the exercise tests to determine the mechanical functional capacity of the left ventricle.

Two hundred twenty-four patients were examined and a total of 450 exercise electrocardiograms were recorded. Cases with acute myocardial infarction, clinically apparent congestive heart failure, and any arrhythmia which might interfere with the pattern of the P waves were excluded. All patients were in good condition to carry out the two-step test at 30 trips in 1-1/2 minutes. A routine electrocardiogram at rest and exercise electrocardiogram using V₁ and V₅ or V₆ were taken immediately after, 1 minute, 3 minutes, 5 minutes and 7 minutes after exercise. In some cases the observation was made after 9 minutes until the changes returned to the control levels.

The patients were divided into 4 groups according to the results

Group I: This group consisted of 42 patients who were hospital employees or non-cardiac patients without any evidence of cardiac diseases. The P wave and ST-T were within normal limits at resting electrocardiogram and showed no changes after exercise (14.1%).

Group II: This group consisted of only 8 patients with coronary artery disease who demonstrated either normal or slightly biphasic P waves in V₁ at resting electrocardiogram and showed no alteration of the P waves after exercise. But the normal or abnormal ST-T became abnormal or more marked after exercise (3.2%).

Group III: This group consisted of 68 patients and a total of 149 tracings were recorded. The majority of the cases were of coronary artery

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disease with or without hypertension. The P waves in this group were either normal or biphasic at rest which became abnormal or more biphasic after exercise. It should be noted that the ST-T was either normal or abnormal at rest and did not show any demonstrable change after exercise. It must be emphasized that these cases (43.8%) might have been overlooked as either normal or having good cardiac reserve if one only observed the ST-T changes.

Group IV: This group consisted of 82 patients similar to those of group III. A total of 132 tracings was taken and showed both P waves and ST-T changes after exercise (38.9%).

On a total of 79 patients, serial tracings were recorded over a 12 month period. It is very interesting to note that the majority of those patients showing P wave changes became less biphasic after administration of cardiac glucosides despite the absence of clinically recognizable congestive heart failure. It should also be pointed out that the improvement of the P wave with cardiac glucosides was associated with an improvement of ST-T. It is therefore hypothesiz-
ed that the ST-T changes caused by ventricular ischemia may be due to subclinical heart failure and that it is not necessarily due to primary ventricular ischemia of coronary artery disease as previously assumed. This may explain some of the anginal cases which often become asymptomatic with an administration of either cardiac glucosides or diuretics.

The amelioration of the symptoms without electrocardiographic evidence of any improvement has been the subject for many discussion about the efficacy of using coronary dilators. The present study offers some new ideas about this topic. Some coronary dilators may be effective on anginal pain through neurohumoral action, but the ST-T show no improvement unless the coronary dilator increases cardiac contractility or cardiac output.

Although this is a preliminary report, the method is non-invasive and reproducible. It seems therefore that the observation of the P wave in V1 is clinically useful for the evaluation of cardiac function.

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