Significance of ST segment depression appearing in recovery phase of stress testing

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Introduction

Introduction: ST segment depression is a marker of ischaemia on surface ECG. The other non-ischemic causes of repolarisation abnormality are mechanical lesions that place a greater burden on left ventricular dynamics and oxygen requirements include such abnormalities as mitral or aortic valvular dysfunction, pulmonary hypertension, pericardial constrictions, and left ventricular hypertrophy. Relative coronary insufficiency is probably also the responsible mechanism in patients with left ventricular hypertrophy. Patients with increased left ventricular mass, even in the absence of standard ECG voltage criteria for this diagnosis, may have false-positive ECG exercise responses.

A wide variety of miscellaneous situations has also been associated with falsely positive ST responses to exercise. These include digitalis administration, hypokalemia, normal postprandial changes, hyperventilation, postural changes, vasoregulatory abnormalities, mitral valve prolapse, pectus excavatum, and intraventricular conduction defect including left bundle branch block and Wolff-Parkinson-White syndrome. There is no common mechanism for ST shifts in these diverse situations.

ST segment depression during exercise: ST-segment depression, the commonest manifestation of exercise-induced ischemia, reflects subendocardial hypoperfusion, but does not have any localization value. It is said to be significant when it is horizontal (or downsloping), measures more than one millimeter 80 ms after the J point.

Approximately 3 to 24% (average 13%, primarily dependent on age) of asymptomatic subjects will have ST-segment depression during exercise. This abnormality is considered a hallmark of left ventricular ischemia resulting from coronary artery disease if other cardiac, metabolic or iatrogenic causes can be excluded.

In symptomatic populations in which a relatively high prevalence of coronary artery disease is expected, the presence of ST-segment depression (>1 mm) during exercise has a high (about 90%) predictive value. In asymptomatic populations, however, in which the prevalence of coronary artery disease is likely to be very small, the predictive value of ST-segment depression during exercise falls to 37-55%. Even though the predictive value is poor, it has been shown that this asymptomatic group with ischemic ST changes during exercise has a 10 to 14-fold increased chance of developing a serious coronary event within three to five years compared to asymptomatic individuals without ST-segment depression during exercise.

To be of diagnostic value, the ST-segment depression should be:
1. at least 1 mm;
2. 80 ms after the J point;
3. in two concordant leads;
4. horizontal or downsloping.
5. It has even more value if it:
   - persists or worsens during recovery;
   - is accompanied by chest pain or by arrhythmia, especially ventricular.

Mechanism of ST Depression: During exercise, progressive ischemia results in changing endocardial action potentials during both diastole and systole. Less negative endocardial cell resting membrane potential leads to current flow across the ischemic boundary during diastole, leading to elevation of the TQ segment on the ECG. Lower endocardial plateau voltage leads to current flow during systole, leading to ST-segment depression. These combined diastolic
and systolic effects of subendocardial ischemia produce ST-segment depression.10

**ST segment depression during recovery phase:**

While the diagnostic and prognostic value of ST segment depression occurring during the active phase of exercise test is well recognised, only a few studies have investigated the clinical significance of ST segment depression in recovery phase of exercise testing. Indeed, there are no indications about how to consider this finding in the international guidelines for exercise stress testing.

G A Lanza et al5 has shown that diagnostic and prognostic power of recovery-only ST segment depression is largely similar to that of ST segment depression induced during the active phase of the test; the diagnostic power of recovery-only ST segment depression for significant CAD was similar to that of exercise-induced ST segment depression. Significant coronary artery stenoses were found in 85% of patients with ST depression during exercise and 78% of patients with ST depression during recovery phase. Moreover, although severe CAD (three vessel or left main disease) was more prevalent in the group of patients with exercise-induced ST segment depression, it was also found in a clinically relevant proportion of patients with recovery-only ST segment depression (18%).

Casella G et al6 has shown that in stable patients with old Q wave AMI, “recovery only” ST depression is rare, but does represent a true sign of ischemia. It could be associated with indirect indexes of worse ventricular function. The prognostical power of “recovery only” ST depression is mild, although similar to that of “exercise only” ST depression. Moreover, the presence of ST depression not only during exercise but also during the recovery phase identifies patients with more severe prognosis. Therefore, the inclusion of findings from the recovery phase in the analysis of the exercise test could increase the predictive power of the test itself.

The prevalence of ST segment depression limited to the recovery phase of exercise test ranges widely among the few studies published in the literature. The variability can probably be explained mainly by differences in the characteristics of the study cohorts and methods applied for positivity of ST segment depression.

In conclusion, in patients with a suspected or documented history of CAD, ST segment depression in recovery phase should be carefully assessed when evaluating the results of exercise stress testing in patients with suspected or documented CAD.

The relatively frequent occurrence of ST segment depression, however, highlights the importance of having an appropriate recovery phase, which should be prolonged to at least five minutes.

Computerized analysis of the HR-adjusted ST depression pattern during the exercise phase, integrated with the HR-adjusted ST depression pattern during the recovery phase after exercise, can significantly improve the diagnostic performance and clinical utility of the exercise ECG test for the detection of coronary artery disease.

The predictability of coronary disease in asymptomatic subjects may be enhanced by analysis of the duration as well as the appearance of ST segment depression.

The relevance and relationship of exercise-induced ST segment and assessment of CAD is well established. The appearance of exercise-induced delayed ST segment depression during recovery phase of stress testing still need rationalization with respect to therapeutic validation.

**References**