Culprit Vessel Localization on Stress ECG Testing

Exercise stress electrocardiographic testing (GXT) remains the test of choice for coronary artery disease (CAD) in most patients (1,2). However, it lacks the ability to localize the culprit vessel compared with the more costly tests, such as stress imaging. Given the historical focus on ST-segment changes, it is not surprising that a majority of the suboptimal localizing performance has been related to looking at ST-segment changes alone. Exercise-induced ST-segment elevation in segments without old myocardial infarction localizes the culprit vessel (2) but is an uncommon finding. Non-ST-segment changes might add important localizing information (e.g., the presence of LAHB during exercise [3] or an interatrial block [4]), but these are mostly case reports and not part of an extensive database of exercise testing. We found a heretofore undescribed heart rate pattern during GXT—a sudden and transient slowing of heart rate in the immediate recovery period—and hypothesized that

Figure 1. Inducible Ischemia

An example of a patient with inducible ischemia showing adequate heart rate response at peak exercise (A), which changed to sudden slowing in the immediate recovery period (B), which then spontaneously normalized in the post-exercise recovery period (C). An angiogram later showed a significant lesion in the artery subending the sinus node.
this unusual exercise-induced change in heart rate could signal ischemia of the sinus node and thus localize the culprit vessel.

A total of 9,536 stress electrocardiography (ECG) tests (Bruce protocol) were performed over 15 years in the stress ECG testing laboratory at Central Railway Headquarters Hospital, Mumbai, India. In addition to the usual criteria for a positive stress test, we also prospectively evaluated for the sudden and transient slowing of heart rate during the immediate recovery period (Fig. 1) in all of these patients. Of the 4,497 patients (47%) evaluated for chest pain, 2,129 (47%) tests were positive for ischemia based on standard criteria. In addition, 32 of the total patients with positive tests (1.5%) showed an atypical heart rate response. All of these patients underwent coronary angiography, and all had >90% proximal stenosis before origin of the sinoatrial (SA) nodal artery (right coronary artery in 66% and left circumflex coronary artery in 34%); 14 patients had 1-vessel disease, 11 patients had 2-vessel disease, and 7 patients had 3-vessel disease. None of the patients without a positive stress test or those tested for a non-CAD indication had this type of heart rate response.

We had hypothesized that this atypical heart rate response could have been due to ischemia of SA nodal artery, and our findings support this. The origin of the SA nodal artery follows a distribution that is similar to that seen in our angiographic findings. We believe that this is the first report prospectively describing this phenomenon, and confirmation in a large clinical database suggests that this finding is a reliable parameter to localize the culprit vessel. There is one case reported in literature (5) of syncope with bradycardia during ergometric testing, and angiography revealed obstruction of the SA nodal artery arising from the left circumflex coronary artery. However, that response is different from what we have observed, and a detailed search of the literature did not reveal any report finding sudden and transient slowing of heart rate during the immediate recovery period on stress ECG testing. We conclude that sudden and transient slowing of the heart rate in the immediate recovery period and a positive result on stress ECG testing are a unique, novel marker that strongly suggests ischemia and localizes the culprit vessel to the right and left circumflex coronary arteries.

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REFERENCES


Molecular Imaging of Native HDL in Human Coronary Plaques by Color Fluorescent Angioscopy

We recently discovered that Fast Green dye (FG), which is used clinically in the field of ophthalmology (1), elicits a brown