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**Editorial Comments**

**Improvements in Exercise Electrocardiography**

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n 1908, Einthoven\textsuperscript{1} published the first example of normal electrocardiographic responses to exercise. In 1928, Feil and Siegel\textsuperscript{2} observed abnormal downsloping ST depression and T wave inversion in three of four patients experiencing anginal attacks (presumably after climbing stairs to the physician’s office; H. Hellerstein, personal communication). In 1932, Goldhammer and Scherf\textsuperscript{3} associated this response with coronary insufficiency, and in 1941, Master and Jaffee\textsuperscript{4} added postexercise electrocardiographic (ECG) monitoring to the previously established two-step test. In 1965, Blomqvist\textsuperscript{5} made important observations on changes in computerized Frank lead ECG recordings, and in 1966, Bruce...and colleagues\textsuperscript{6} evaluated variance of computer-averaged and computer-analyzed components of the ECG. Hornsten and Bruce,\textsuperscript{7} in 1969, compared computerized bipolar precordial and Frank lead responses in healthy young and middle-aged men with those of patients who had coronary heart disease. Also in 1969, Bruce and McDonough\textsuperscript{8} reported quantitative differences in computerized ST responses during exercise and hysteresis variations in recovery and plotted ST displacement versus heart rate as stress-strain diagrams. Since then, innumerable clinical and research studies have described the value and limitations of exercise electrocardiography, one example being published in 1984.\textsuperscript{9} Common problems regarding the value of exercise electrocardiography include “false-positive” responses in healthy persons, especially in women, and “false-negative” responses in patients with coronary heart disease.

A new and interesting insight into the diagnostic value of exercise electrocardiography was the prediction of severity of coronary artery disease with the slope of the submaximal ST segment/heart rate relation that Elamin and colleagues\textsuperscript{10} published in 1980. Despite initial skepticism, subsequent publications from these investigators in Leeds, England, in 1982 and 1984 have strengthened the evidence for diagnostic value and have prompted others to adopt the improved method that incorporates exertional ST responses. Among the latter group are two important contributions. In 1986, Okin, Amiesen, and Kligfield\textsuperscript{11} emphasized the value of modifying the conventional multistage Bruce protocol for treadmill exercise testing to provide smaller increments in heart rate with increasing workloads. This modification provided more computerized data points (every 2 minutes) to better determine the greatest changes in the ST-segment/heart rate slope. The mean increment in heart rate by their modified protocol was only 10 beats/min rather than 27 beats by the conventional Bruce protocol, yet overall heart rate and systolic pressure changes were similar. They established both the accuracy of computer measurements of the ST segment at 60 msec after the J point and the reproducibility of these measurements.

Another study by Kligfield, Amiesen, and Okin,\textsuperscript{12} reported in this issue of *Circulation*, compares the results obtained in 150 patients who did not have coronary artery disease, some of whom had nonanginal pain syndromes, with 150 patients who had acceptable symptoms or signs of coronary artery disease, 100 of whom had undergone evaluation by coronary angiography. With this experimental design for selection of four groups, nearly the entire spectrum of ischemic responses was evaluated. The sensitivity for detecting exertional myocardial ischemia in the patients was only 68% by the usual ECG criterion of $-1$ mm of ST depression. The sensitivity was increased to 95% when the highest ST segment/heart rate slope observed in any of 10 ECG leads exceeded 2.4 $\mu$V/beat/min. Among the 100 clinically normal and the 50 patients with nonanginal chest pain, specificity for exclusion of coronary disease was 84% and 82%, respectively, by standard test criterion and 95% and 90%, respectively, by the ST segment length/heart rate slope criterion. Thus, these additional, careful studies from the laboratory of Kligfield and colleagues provide independent confirmation of their improved method for interpretation of ECG responses for clinical diagnosis. Clinically useful, additional comments have just been published by Kligfield.\textsuperscript{13}

I would suggest, however, two other modifications: 1) expression of the ST displacement/heart...
rate relation as a negative value because few coronary patients have ST elevation and 2) consideration of additional information during the patient’s recovery to complete the time course and hysteresis of the stress-strain relation. The latter may be particularly important in applying to the concept of ST segment depression as a sensitive marker of a true ischemic response that reflects an imbalance between hemodynamic demand and coronary supply.

Finally, because the previous comments relate to diagnosis by improved techniques, two other recent observations should be mentioned that relate the prognostic value of the ST segment response to exercise and that reflect the continuing value of conventional analyses. Among 117 (6.8%) of 1,718 healthy men less than 60 years of age who had no conventional risk factor for coronary heart disease but who had ST depression (according to the standard criterion of −1 mm) with maximal exercise, no coronary events occurred in 5 years, making the prognostic sensitivity of the ST response to exercise zero. Conversely, in 44 (1.1%) of 4,105 healthy men with one or more conventional risk factors and two or more abnormal responses to symptom-limited exercise, high risk was identified with a poor prognosis for primary coronary events and for survival from all causes of death. Of interest, the most frequent abnormal response to exercise was failure to attain 90% of age-predicted maximal heart rate. The prevalence of this response was 88%, whereas prevalence of ST depression in these high-risk men was only 44%.

With the conventional criterion for ST displacement, equaling or exceeding either −1 mm depression or +1 mm elevation during exercise and recovery, there were no significant differences in coronary heart disease event-free survival and survival from all causes of death in 2,982 patients who were not operated on from the Coronary Artery Surgery Study. These patients had ST depression during exercise only or during exercise and recovery, or had ST segment elevation only during exercise. However, among 68 (2.2%) of the patients who had ST elevation during exercise and recovery, there was a significantly lower survival rate. Thus, prolonged exertional ST elevation is a noninvasive marker of poor ventricular function and poor prognosis. Additional research will be needed to assess the prognostic value of ST segment/heart rate slope criteria in such patients.

References

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