Predicting an acute myocardial infarction (AMI), within the context of physicians’ or epidemiologists’ realms, is an important task. Accordingly, the recovery period after an exercise stress test has been subjected recently to scrutiny, with very fruitful results. Traditionally, the time after completion of an exercise stress test has been merely devoted to observing the patients for clinical symptoms, without much thought given to a possible diagnostic utility of information generated during the recovery period. However, in the past few years, diagnostically important data from the recovery period have been forthcoming. An early example was the observation that in >15% of patients subjected to exercise testing, electrocardiographic (ECG) ST-segment depression occurred only during recovery, a finding with a predictive value of significant coronary artery disease not different from that associated with ECG changes occurring during exercise. Also, recent work has documented a relationship between postexercise blunted drop of heart rate and subsequent morbidity and mortality.

In the same context, delayed decrease of the systolic blood pressure (SBP) during the recovery period after exercise has been linked recently to an increased risk of coronary heart disease, stroke, and emergence of hypertension. According to these new ideas, SBP and heart rate, at various designated time points during recovery, become new “risk factors,” and it is the task of researchers and users to validate their incremental value above the “established” indices of risk.

In this issue of Hypertension, Laukannen et al report on an important role of SBP at 2’ of recovery for predicting future AMI, beyond that of the resting SBP. Characterization of their representative cohort of Finnish middle-aged men was careful, and it was based on 26 traditional risk factors and other important differentiators. Measurements of 10 exercise testing variables were used, and the average follow-up exceeded 13 years. A set of 12 clinical and exercise predictors of AMI, in addition to the SBP at 2’ of recovery, were analyzed. SBP at 2’ of recovery, treated as a continuous variable, as a classified in tertiles one, and as a difference from the resting SBP, was directly related to future risk of AMI and cardiovascular death after adjusting for 14 and 6 other risk predictors.

The findings of the study reflect an ongoing activation of the sympathetic nervous system precipitated by exercise testing and attenuated vagal reactivation, as postulated previously for the changes in the heart rate and emergence of ventricular ectopy. Also, it appears that functional and structural abnormalities operating in concert conspire to induce this postexercise-attenuated decline in SBP at recovery. A very reassuring element is that aerobic exercise training can probably improve the vascular response to exercise of atherosclerosis patients.

The strong points of the study are the prospective design, inclusion of the 72.2% of the original study cohort, the complete patient follow-up, the appropriate characterization of the study subjects, inclusion of many parameters from before, during, and after stress testing, and the thorough review of the relevant literature. The authors were careful in pointing out the differences in using a cycle ergometer, in keeping with the prevailing practices of many European studies, rather than a treadmill for the exercise test. They cite as advantages the ease of reliably measuring blood pressures, and they are fair by referring to the controversy whether the heart rate pressure product is similar or lower with their method compared with that found with treadmill testing. The authors view as an advantage may be a limitation for other environments, such as the United States, where the use of the treadmill-based exercise testing is almost universal. As for the authors’ speculation that the mode of exercise may not influence the response in SBP at recovery, this remains to be shown. The same applies to the SBP with recovery “cool-down” protocols consisting of walking, lying, or sitting.

Without detracting from the value of the study, the inclusion of only men, of a certain age bracket, and of a specific nationality with a high prevalence of atherosclerotic cardiovascular disease could be considered “limitations” of the study, using the term narrowly and in the sense that the study findings may have less generalizability than if the study were less restrictive in its recruitment. Furthermore, some opportunity was missed (and this includes a real limitation) by noninclusion of data on the 2’ heart rate recovery. Such information was not available by design, but it would have been very useful for the multivariate analysis because it could demonstrate the real incremental role of heart rate and SBP at recovery as future predictors of AMI. It is conceivable that if 1 of these 2 is available, the other (which one?) is not needed. This may have practical implications because heart rate is automatically acquired by continuing the ECG monitoring after exercise cessation.
What needs to be done in the future is work similar to that reported herein, using treadmill exercise testing and using as many blood pressure and heart rate variables as possible. Because much literature has been generated with data obtained at 30’ to 5’ of recovery, it would be reasonable to include such information beyond that from the 2’ recovery time mark. Information obtained before and after the 2’ of recovery may yield different prognostic insights with different sensitivity and specificity. The trend in exploring the recovery period is expected to continue at an accelerating rate. However, what should drive this enterprise is the quest for incremental information. The positive correlation among SBP at 2’ of recovery and peak SBP during exercise, SBP at rest, and even the resting diastolic blood pressure under-score the significance of this.

The study of Laukannen et al provides some justification for performing an exercise test in subjects with risk factors but not overt cardiovascular disease, something traditionally not recommended. However, to assess change in the risk longitudinally of different cohorts of patients, responses of the SBP and heart rate at recovery may need to be studied in repeat exercise tests. The feasibility of en masse clinical application of such recommendation may be called to question. Perhaps adopting a “poor man’s” approach may contribute to the wide acceptance of evaluating the postexercise recovery period: checking SBP and heart rate at 1’ or 2’ after a patient walks the same number of flights of stairs in the same time interval. This will provide an analog to the “6’ exercise test” used in congestive heart failure patients.

References
Blunted Decline in Systolic Blood Pressure After Exercise Predicts Future Acute Myocardial Infarction
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