# A New Look at Exercise Treadmill Stress Testing

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This *Heartbeat* will take a new look at graded exercise treadmill testing (ETT), a cornerstone of diagnostic procedures for coronary artery disease (CAD), which was introduced by Robert Bruce five decades ago. Traditionally, interpretation of ETT has been based almost exclusively on the observation of ST-segment depression, the hallmark of exercise induced myocardial ischemia. At a time when radionuclide and echocardiographic imaging have become increasingly popular in cardiovascular diagnosis, this *Heartbeat* will point out other ways in which ETT alone can be extremely useful. A list of diagnostic and prognostic variables that should be observed during exercise and recovery is outlined below in Table 1.

**Table 1.**

| **Diagnostic and Prognostic Variables during Exercise or Recovery.** |
|---|
| **Observations during exercise** |
| *Maximal exercise capacity* |
| ST-segment depression |
| ST-segment elevation |
| Angina Pectoris |
| Inadequate blood-pressure response |
| Inadequate heart-rate response |
| Ventricular arrhythmia |
| **Observations during recovery** |
| ST-segment depression |
| *Delayed slowing of heart rate* |
| *Ventricular arrhythmia* |

## Survival of the Fittest

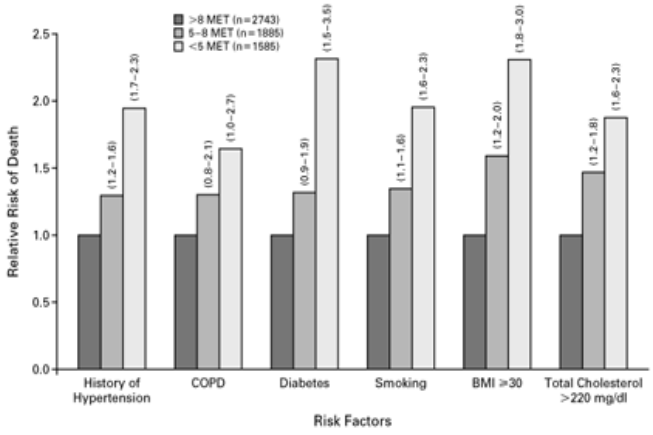
Nearly 150 years after Charles Darwin's published his theory of evolution, more evidence regarding the relation between fitness and survival was provided in a study by Myers et al.[1] More than 6200 consecutive male patients who underwent ETT were followed for six years. The results demonstrate that exercise capacity is a strong predictor of death—that is, *the survival rate was lower as exercise capacity decreased* (P-0.001). Exercise capacity (cardiorespiratory fitness or physical fitness) is a set of attributes that enables a person to perform physical activity. It is determined in part by habitual physical exercise but is influenced by other factors such as age, sex, genes, and medical status.

In this study peak exercise capacity was a stronger predictor of death than established risk factors such as hypertension, smoking, and diabetes, as well as other exercise test variables, including ST segment depression, peak heart rate or development of arrhythmias during exercise. Being more fit is analogous to being younger and having the ability to "take a hit" and keep on going.

A perfect example of this is seen in a very recently published study that demonstrated functional status to be a significant predictor of CABG outcome and when compared with age it was also found to be a more reliable predictor of CABG outcome.[2] Poorer functional status is also one of the probable explanations of why women historically have poorer outcomes post CABG. They as a rule have poorer preoperative functional status compared to men of equal age and this was also supported by this study.

Myers' data also confirm the protective role of fitness even in the presence of other risk factors. The *risk of death from any cause in subjects whose exercise capacity was less than 5 METS (peak energy expenditure in metabolic equivalents)—completing a first stage of the Bruce ETT protocol—was roughly double that of subjects whose exercise capacity was more than 8 METS—completing the third stage of the Bruce ETT protocol* (Figure 1).

**Figure 1. Relative Risks of Death from Any cause Among Subjects with Various Risk Factors Who Achieved an Exercise Capacity of < 5 METS Compared to Those Who Achieved an Exercise Capacity of > 8 METS.**




*Clinical implication:*

There is a significant link between survival and fitness. *Patients with and without cardiovascular disease (CVD) who are less fit (less active) can improve their survival if they increase their level of fitness or physical activity.* This data should compel clinicians to go beyond the identification of risk to the initiation of interventions, such as an exercise prescription of moderately intense physical activity for at least 30 minutes on most–and preferably all—days of the week, in order to decrease risk, especially in those with low levels of fitness. In terms of reducing mortality from any cause, improving exercise tolerance warrants as much clinical attention as other major risk factors from physicians who treat patients with or at high-risk for CVD.

## Mortality and Heart Rate Recovery After Exercise

During a graded ETT, heart rate (HR) progressively increases, due to an increase of sympathetic activity and a concomitant decrease in parasympathetic (vagal) activity. After cessation of exercise, these changes in autonomic activity are reversed, and HR decreases, i.e. a fall in HR after exercise is mediated by vagal activity. The rate at which HR declines after exercise is a reflection of physical fitness—the more rapid the decline, the higher the level of fitness. Increased vagal activity has been associated with reduced risk for death.

A Cleveland Clinic study of 2428 patients found that an *abnormal HR recovery (12 beats per minute or less after stopping exercise*—reflecting an inadequate reactivation of vagal tone—was an independent predictor of death with a quadrupling of the risk of death over the next six years.[3] These conclusions were made after adjustments for numerous variables, including history of CAD, and use of cardiovascular medications.

*Clinical implication*:

HR recovery, presumably mediated through the autonomic nervous system is an easily calculated risk factor that can provide independent prognostic information. *Careful monitoring of HR recovery post-exercise adds substantially to the value of ETT and can be used to identify higher risk and trigger more aggressive intervention* (invasive) in patients with intermediate risk stress test results or stimulating patients to better comply with risk factor reduction strategies and exercise programs to improve fitness (non-invasive).

## Ventricular Ectopy after Exercise Predicts Increased Mortality

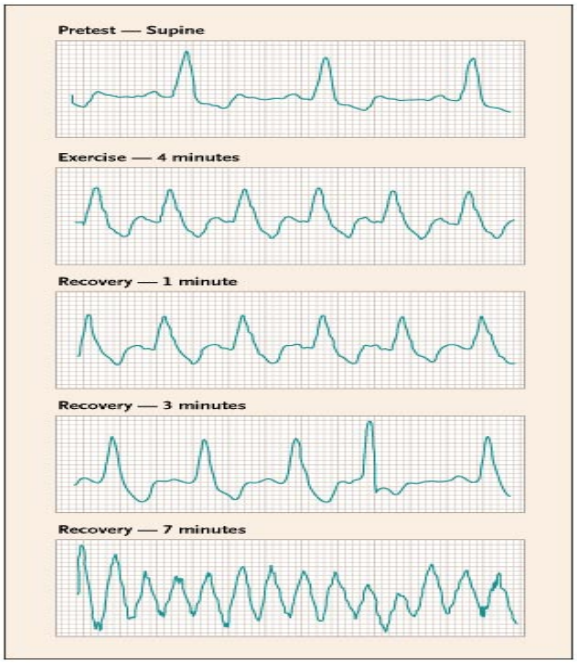
Horizontal or down-sloping ST segment depression of 1 mm or more is the hallmark of exercise-induced myocardial ischemia. Marked ST segment depression, especially at low levels of exercise, is a prognosticator of more severe disease. ST segment elevation in leads without Q waves is also indicative of severe ischemia. Other important markers include angina pectoris, ventricular arrhythmias and chronotropic incompetence (inadequate or depressed HR or blood pressure response).

Observations of ST segment depression that first appear post-exercise or significantly worsen post-exercise, have at least similar—possibly worse—prognostic implications as those that occur during exercise. This information, along with HR recovery post-exercise, stresses the importance of continuing to monitor the patient and the ECG during recovery.

A new study from the Cleveland Clinic group further supports these observations.[4] The authors concluded that the appearance of high-grade ventricular arrhythmias during the recovery period post-exercise predicted subsequent mortality better than did ventricular arrhythmias during exercise. The association between death and ectopy after exercise

was noted among patients with normal systolic function and those with reduced systolic function.
As with delayed slowing of HR, the study group suggests that the ventricular arrhythmias during recovery may be due to inadequate vagal reactivation, since vagal stimulation is known to suppress ventricular arrhythmias. An ECG from one of their patients demonstrates the association of these two findings. The patient had both delayed HR recovery and high-grade ventricular arrhythmia in recovery and then the development of fatal ventricular fibrillation (Figure 2.)

**Figure 2. ECG Tracing from a patient before, during and after exercise**




*Clinical Implication*:

Frequent ventricular ectopy during recovery was found to be an independent predictor of an increased risk of death. No such association was found with frequent ventricular ectopy during exercise. Accordingly, comprehensive risk-factor assessment and aggressive management of the risk identified would be recommended in patients with this finding.

Ventricular ectopy post-exercise is common in patients with asymptomatic left ventricular dysfunction. Accordingly, echocardiography to evaluate for LV dysfunction is also recommended since proven treatment would be of clinical benefit to decrease risk.

## Conclusion:

The important clinical implication from this *Heartbeat* is that ETT should include careful cardiac monitoring post-exercise during the recovery period. Information from observations during exercise and the recovery period post-exercise are useful for risk stratification and subsequent management, with *emphasis on regular exercise for all.*

**Hot off the press from the ACC meeting in Chicago (4-3-03):** *hs*-CRP, an inflammatory marker associated with high-risk CAD is decreased with regular physical exercise—an added bonus!

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*Heartbeats* can be found @ www.sjhg.salu.net under Patient Education—From Your Physician
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[1] Myers J et al. Exercise capacity and mortality among men referred for exercise testing. *N Engl J Med* 2002; 346: 793-801.
[2] Mayer C et al. Self reported functional status as a predictor of coronary bypass graft surgery outcome in elderly patients. *Canadian J Cardio* February 2003; 19: 140-44.
[3] Cole C R et al. Heart-rate recovery immediately after exercise as a predictor of mortality. *N Engl J Med* 1999; 341: 1351-57.
[4] Frolkis J P et al. Frequent ventricular ectopy after exercise as a predictor of death. *N Engl J Med* February 27 2003; 348: 781-90.