

Cardiac Stress (Treadmill) Test in the Prediction and Diagnosis of Heart Disease in Hypertensive Persons

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ABSTRACT

Heart disease is a major cause of mortality and morbidity in hypertensive persons. The medical data leads used in cardiac stress test is useful in detection of cardiovascular dysfunction. This test is used for evaluation of heart disease and risk grading with inadequate blood supply by exercise or stress and increased cardiac demands in hypertensive patients. This research was conducted to evaluate cardiac stress test in hypertensive patients with chest pain, diabetes, smoking and pre-family records of heart disease. The result showed that inheritance of hypertension is general and use of tobacco increases risk, and there is need of precaution for persons with hypertensive family records. The observation indicated TMT as predicting factor for heart problems with less expensive diagnostic feature.

INTRODUCTION

The cardiac stress test is diagnosis for heart dysfunction with ECG record during an induced event of increased cardiac demand. This test is used for detection of coronary artery disease (CAD) and patients are monitored for risk stratification as blood supply may inadequate when cardiac demands increased by exercise or stress. Stress testing is less invasive and less expensive than cardiac catheterization and it detects patho-physiologic abnormalities of circulation. It can define the functional significance of abnormalities in anatomy of coronary artery identified with coronary angiography during catheterization.

This test compares the coronary circulation while the patient is at rest and during maximum physical exertion, showing any abnormal blood flow to the heart's myocardium. The results can be interpreted as a reflection on the general physical condition of the test patient. The test can be used for diagnose ischemic heart disease, and for patient prognosis after myocardial infarction (heart attack). Abnormal hemodynamic responses to cardiac stress test may indicate an increased risk of coronary events influenced by exercise duration and variation in blood pressure and heart rate.

METHODS AND MATERIALS

The cardiac stress test is done with heart stimulation, either by exercise on a treadmill, pedaling a stationary exercise bicycle ergometer (Medline Plus, 2013) or with intravenous pharmacological stimulation, with the patient connected to an electrocardiogram. The level of mechanical stress is

progressively increased by adjusting the difficulty and speed. The attending physician examines the symptoms and blood pressure response. An exercise stress test may provide more information about exercise tolerance than a pharmacologic stress test (Weissman *et al.*, 2004).

This test was performed on three patients with inherited hypertension, alcoholic and pre-symptoms of coronary artery disease. The prognostic variables are ST-segment depression, exercise tolerance time, blood pressure variation, chronotropic incompetence, heart rate recovery and ventricular ectopy.

RESULTS AND DISCUSSIONS

The test was performed with a low speed (1.7 miles/hour) with 10% incline of treadmill set, and both parameters were increased simultaneously after 3 minutes duration. The test continues for 27 minutes or until the patient quits or develops symptoms of ischemia or an arrhythmia. The average time was 8 to 10 minutes for age-group 45-55. The longer time on treadmill reflects functional capacity and low oxygen uptake (Table 1, Figure1). The duration of exercise is a good prognostic indicator that must be included in risk scores for exercise treadmill testing (Mark *et al.*, 1987; Prakash *et al.*, 2004).

We measured blood pressure with sphygmomanometer. The blood pressure measurement during exercise is variable also due to background noise from the treadmill machine. The measurement revealed lower value than actual blood pressure of patient and this error increased with exercise intensity. Exercise hypotension is well defined as systolic blood pressure of patient that lower during exercise than while standing at rest prior to exercise (Dubach *et al.*, 1988). In same manner, exercise hypertension is defined as a rise in systolic blood pressure during exercise, usually between 190 and 220 mmHg (Tzemos *et al.*, 2002). There is also evidence that exercise hypertension predicts future arterial hypertension in people with normal resting blood pressure (Dlin *et al.*, 1983). There is controversy about exercise hypertension as association with cardiovascular events during a mean period of 7.7 years (Dlin *et al.*, 1983), while in other study predicted that hypertensive response to exercise had a lower prevalence of severe angiographic coronary disease and a lower risk of death over the next 2 years compared with other patients with hypertension (Table2). The recovery period of hypertensive response has been shown to be a marker of hidden coronary artery disease (Amon *et al.*, 1984), but has not consistently been associated with an adverse prognosis (Ellis *et al.*, 2004).

The chronotropic incompetence is failure of increase in heart rate expected during exercise and also decreases as soon as exercise stops. There is a different criterion based on resting heart rate, exercise protocol, age and medication of patient. The predicted chronotropic response can be calculated as: $(\text{peak heart rate} - \text{resting heart rate}) + (220 - \text{age} - \text{resting heart rate})$. The difference between peak heart rate and resting heart rate is the heart rate reserve. The chronotropic incompetence is defined as less than 80% of the predicted value and as less than 62% for patients using beta-blockers (Kligfield *et al.*, 2006).

The heart rate recovery is defined as normal heart rate over several minutes to hours, with the most marked reduction in just after exercise, and impaired recovery predicts cardiovascular events even in population without coronary problems (Jouven *et al.*, 2005). This may be noted that several variables influence heart rate including activity or position after exercise. The difference in successive heart cycle also predicts cardiovascular death during next 5 years(Figure3).

The patient with sustained ventricular tachycardia or ventricular fibrillation or rare left ventricular dysfunction during or after exercise may give rise cardiovascular impairment in future, however, ventricular tachycardia and arrhythmogenic ventricular dysplasia (cardiomyopathy) may not be considered as heart dysfunction without heart problem in healthy persons. This is more common than single ventricular premature contractions, couplets, or short episodes of non-sustained ventricular tachycardia occur during or soon after cardiac stress test than abovementioned sustained type. The prognostic significance of these ectopies is controversial. Beckerman *et al.* (2005) reviewed that ventricular ectopy during exercise testing or recovery was associated with an increased death rate in 13 out of 22 papers in which 15 studies including patient with symptomatic coronary artery disease and normal persons in other 7 studies without symptoms.

Jouven *et al.* (2000) found that among 6,101 asymptomatic male without clinically evident cardiovascular disease 2.3% had frequent premature ventricular contractions (defined as $\geq 10\%$ of all ventricular beats) and 4.4% had ECG changes during exercise that indicated ischemia. The higher risk (RR= 2.67) of cardiovascular death in next 23 years was associated with frequent premature contractions in ventricle.

Frolkis *et al.* (2003) evaluated treadmill testing and found a low prevalence of frequent ventricular ectopy (3% during exercise, 2% after exercise and 2% both during exercise and after exercise). The 5-year mortality rate was higher in patients with ventricular ectopy vs those without ectopy and after adjusting for confounding variables, only frequent ectopy in recovery, and not during exercise, was associated with an increased death rate (Figure 4).

The association between ventricular ectopy after exercise, ischemia and left ventricular function are unclear.

CONCLUSIONS

This is uncertain to manage patients with an abnormal hemodynamic activity in the absence of ischemia, but such response due to poor health or autonomic nervous system dysfunction can be managed with interventions. The exercise training may increase functional capacity is associated with low mortality (Blair *et al.*, 1995), and coronary artery bypass surgery can abolish exercise-induced hypotension (Thomson *et al.*, 1975).

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Table 1. Cardiac stress test for patients with variable symptoms.

Variable	Patient with Hypertension	Patient with inherited Hypertension	Patient with Diabetes

Duration (minutes, Bruce protocol)	12	8	4
Chest Pain during exercise	No	No	No
Limiting symptom	Fatigue	Mild fatigue	Dyspnea
Resting blood pressure (mmHg)	130/80	140/90	150/100
Peak exercise blood pressure (mmHg)	200/60	210/70	120/60
Resting heart rate (beats/minute)	70	92	62
Peak exercise heart rate (beats/minute)	150	170	130
Heart rate 1 minute recovery (beats/minute)	128	160	142
Exercise ST-Segment depression (mm)	1.5	2.3	1.3

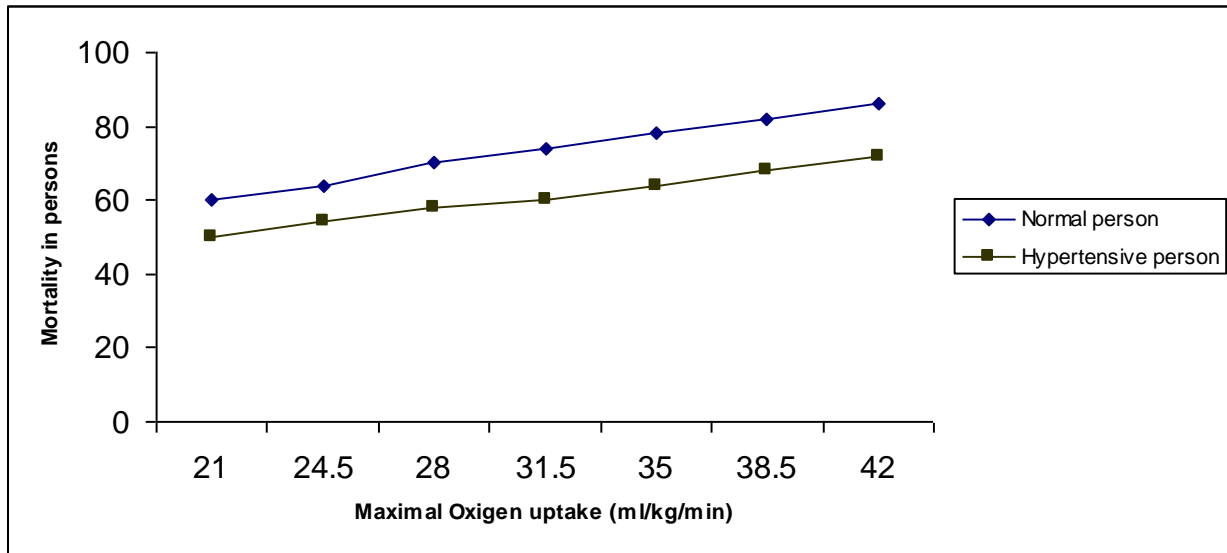


Figure1. Fluctuation in maximal Oxygen uptake and mortality in hypertensive persons.

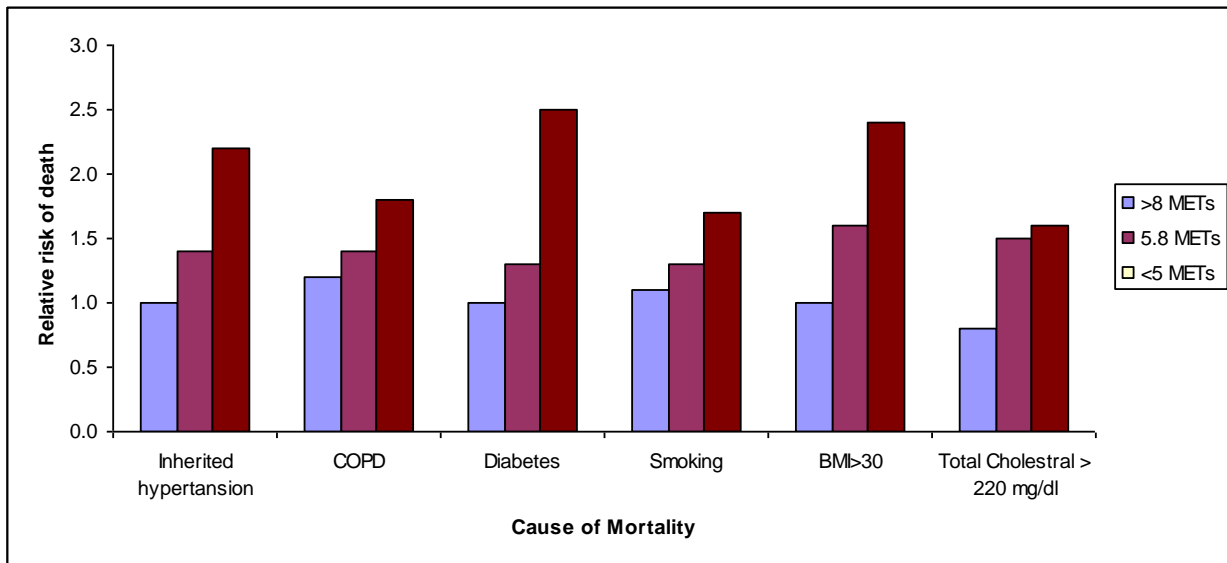


Figure2. Relation between mortality factors with variable METs and risk of death.

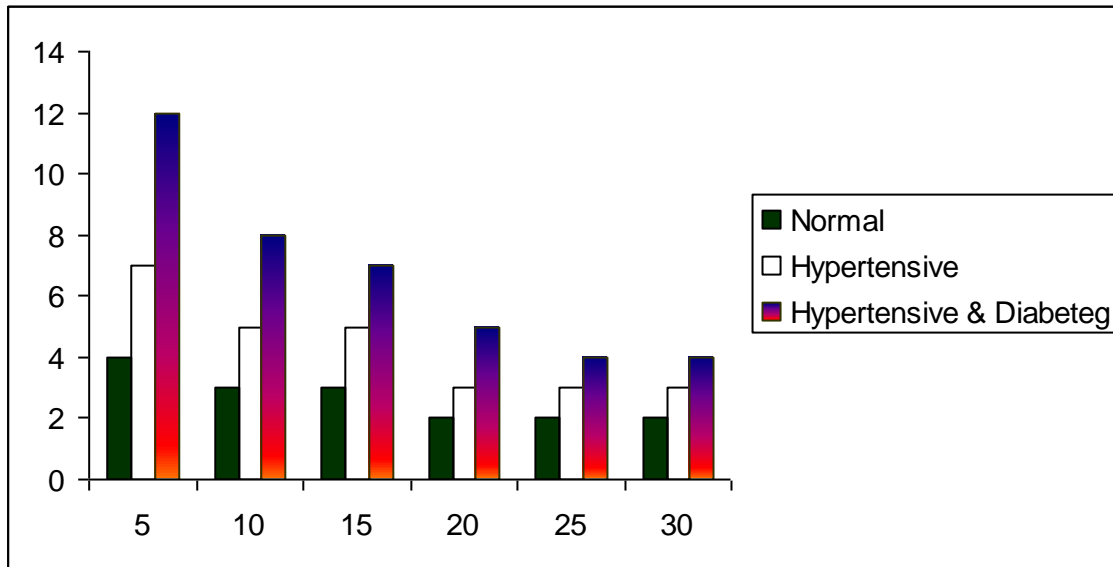


Figure3. Expected life-span and percentage occurrence of death in persons(X=Life-span and Y= % death).

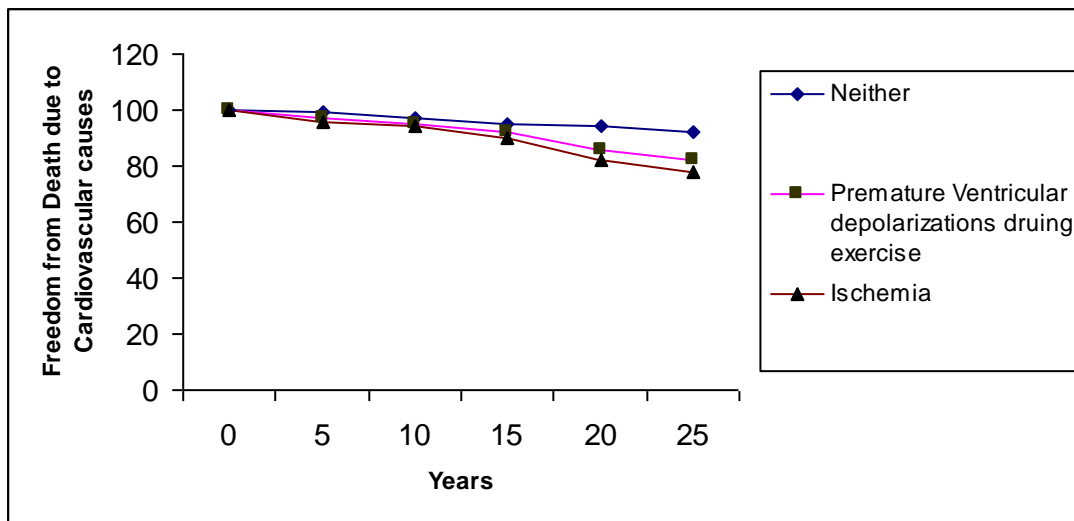


Figure4. Expected survival and freedom from death with various symptoms.