DIAGNOSTIC VALUE OF THE RATIO OF RECOVERY SYSTOLIC BLOOD PRESSURE TO PEAK EXERCISE SYSTOLIC BLOOD PRESSURE FOR THE DETECTION OF CORONARY ARTERY DISEASE

Thesis
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By

Tamer Mahmoud Salah El Din Moustafa
M.B.B.Ch.

Supervisors

Prof. Dr. Adel Mohammed Kamal El Etriby
M.D.
Ass. Prof. of Cardiology
Ain Shams University

Dr. Ihab Mohammed Atia
M.D.
Lecturer of Cardiology
Ain Shams University

Ain Shams University
1993

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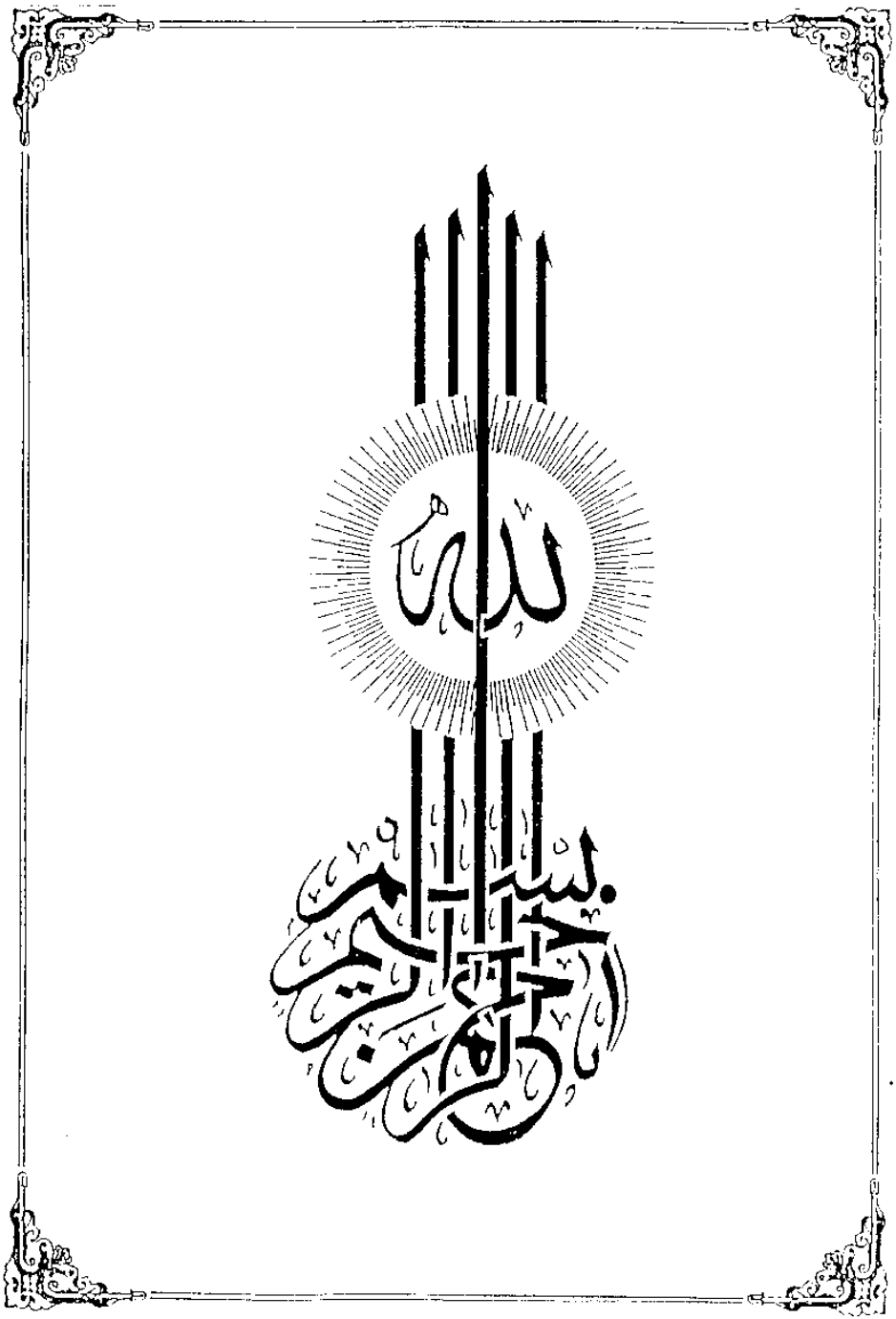

INTRODUCTION

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When pain is of questionable nature, it is difficult to predict with certainty if it is due to coronary artery disease or not, 50 - 70% of patients with typical angina and no previous infarction have normal resting ECG (Bartel et al., 1974).

A good history is usually sufficient to establish the diangosis of anginal or sympotmatic coronary insufficiency. Somtimes however the pain is atypical, the history is doubtful and the ECG recorded at rest, may be normal or equivocal.Furthermore the response to amyl nitrate may at times relieve pain that is not cardiac in origin. Under such circumstances objective confirmation of the diagonsis becomes desirable and the ECG changes seen in response to exercise may provide this confirmatory evidence (Faurtin et al 1977).

Exercise testing and ECG recording is used to expolre problems in the diagnosis of angina. It can also be used for assessment of results of therapy, evaluation of functional capacity and in studies of epidemiology of ischemic heart disease (Bartel et al, 1974).

Since the original descriptions of electrocardiographic (ECG) changes during angina pectoris (Bousfield et al 1918), numerous investigations have been conducted to assess the feasibility of increasing the sensitivity and specificity of the ECG for detecting ischemic heart disease.

Changes in the ECG - ST - segment during exercise have recieved the most attention and a wide range of

sensitivities have been demonstrated, depending on the patient population selected and other variables related to the exercise testing procedure (Fortuin N, Weiss JL - 1977).

The diagnostic usefulness of the ST segment is decreased by pharmacologic therapy, the presence of conduction abnormalities, or left ventricular hypertrophy, therfore several investigators have raised concern regarding the relative usefullness of exercise - induced ECG ST segment changes when they are applied to the diagnosis of coronary artery disease (CAD) (Redwood DR, Borer JS et al 1976).

Inanattempt to increase the sensitivity of Exercise ECG testing for detection of ischemic heart disease some Investigators have utilized changes in the R-wave and T-wave (Jansson R. et al 1980)

Greenberg et al empolyed a multivariant approach in which 21 parameters were used and found a sensitivity of 85%. Whether or not the sensitivity of exercise ECG testing can be increased consistently above 85% is doubtful.

Routine exercise stress testing usually includes measurements of the patient's blood pressure before, during, and for several minutes after exercise. The post exercise blood pressure has been recorded primarily to monitor the patient's recovery from exertion, and thus its diagnostic implications have not been studied extensively. We have

observed that some patients have systolic blood pressures (SBPs) in the recovery period that are higher than the peak exercise pressures, and that patients who exhibit this phenomenon were found to have severe CAD, as determined by coronary cineangiography (Forsell G et al 1981).

The aim of our work is to study the diagnostic value of the recovery systolic blood pressure ratio to the peak exercise systolic blood pressure and to compare it with the classical S - T segment depression in a group of patients complaining of chest pain.

PHYSIOLOGIC CARDIOVASCULAR RESPONSE TO EXERCISE TESTING

PHYSIOLOGIC CARDIOVASCULAR RESPONSE TO EXERCISE:

The response of the caridovascular system during exercise involves virtually every circulatory component, the increased sympathetic discharge during exercise is of critical importance in the regulation of heart rate, myocardial contractility and the capacitance and resistance of the vascular bed, thereby controlling the cardiac output, blood flow distribution and arterial pressure (Donald and Shepherd , 1979).

One of the major response patterns of the cardiovascular system to dynamic exercise is maintenance of an acceptable body temperature, which becomes an important consideration in the regulation of the circulation. If exercise continues until body temperature begins to rise (Wyndham et al., 1973).

The multiple changes in the cardiovascular system during exercise stress are reflected by many indirect, non invasive measurements including the following:-

1 - HEART RATE:-

This is the first cardiovascular adaptation to occur. As a general rule, the lower the resting heart rate, the better the physical condition (Scherer 1973).

During mild exercise the predominating mechanism of cardiac acceleration is vagal withdrawal, while during later stages of intense exercise it is sympathetic activity (Robinson et al 1966). Parasympathetic withdrawalclearly plays a role in humans, particularly in well trained atheletes who

will demonstrate a marked degree of bradycardia at rest (Little 1981).

The maximum predicted heart rate is age related and the peak heart rate that can be achieved decreases gradually with age (Faurtin et al 1977).

The increase in heart rate closely parallels the increase in oxygen consumption and the relationship between the maximum oxygen uptake by the body as a whole and the increase in heart rate tends to be almost linear until about 85 or 90% of maximum capacity is reached. At this point there is a slight further increase in oxygen uptake without a significant increase in heart rate. (Little 1981).

2 - Stroke volume and cardiac output:-

Stroke volume usually increases up to 30% during the early stages of exercise and then gradually levels off (Stratton et al 1983).

While cardiac ouptut may rise from 5 to 25 liters / min (Epstein et al 1967). The increased heart rate is the major factor responsible for augmentation of the cardiac output during mild to moderate exercise (Hossack K.F. 1980).

There is vasodialation of the blood vessels supplying exercising muscles during exercise. This is a multifactorial phenomenon that plays a role including autoregulation as a result of metabolic end products in working muscles, effects of circulating catecholamines and stimulation of sympathetic vasodialtor nerve fibers. Peripheral vasodilation tends to reduce afterload and therapy facilitates left ventricular

emptying (Evanset et al 1967).

Studies in humans and animals have shown that contractility is increased with exercise (Noble 1972). Norepinephrine relased by sympathetic nerve endings in the heart is probably the most important factor regulating myocardial contractility. It is likely that the Frank-starling mechansim which occurs more commonly in the supine position in normal subjects plays an important role in increasing contractility (Martin et al 1986).

4 - BLOOD PRESSURE RESPONSE:-

By increasing workloads there is gradual elevation of the normal systolic blood pressure, elevation of the systolic blood pressure during exercise is due to increased cardiac output, which results from an increase in heart rate, and is also due to an increase in peripheral vascular resistance.(Iriving et al 1977).

The rise in SBP that accompanies dynamic exercise in normotensives increases with age of the subject, as it ranges from 162 to 216 mm Hg during the peak exercise, other studies have shown that the SBP increases by approximately 60 mm Hg in men and 40 mm Hg in women. (Guyton 1979).

When exercise is extnded beyond the aerobic threshold there is accumulation of lactic acid leading to drop of the peripheral resistance as well as a decrease in Myocardial Contractility (Ellestad et al ., 1986), and so SBP levels off and often declines during peak exercise.

SBP falls in a fairly rapid manner during recovery, although a rebound temporary rise is common after exercise due to the recovery from anaerobic metabolism that has occured near peak workload, (Ellestad et al., 1986).

As regards the Diastolic blood pressure (DBP), normal subjects respond to exercise with no change or with a slight decrease of DBP (Less than 10 mm Hg.), during maximal exercise (Bruce et al 1974). This is due to a decrease in systemic vascular resistance which occurs during exercise as a result of accumulation of metabolic materials, decrease in Po_2 increase in Pco_2 and rise of temperature, excess accumulation of metabolites during the post exercise period leads to more decline of DBP. (Sheps et al 1978).

Some investigators reported that the DBP may show significant increase with exercise without any obvious cause (Franz 1986)., however the response of DBP to exercise is age dependent, as it may fall slightly in younger individuals where as in the older subjects it is likely to rise, any rise or fall of DBP in normal persons is not likely to exceed 10 mm Hg.(Fishcer et al., 1977).

The mean arterial blood pressure shows modest elevation with exercise as a rise of 20 to 25 mmHg in normal persons is seen., the SBP increase accounts for the mean arterial pressure rise since DBP remains unchanged or slightly decreases, thus the pulse pressure widened (Guyton 1981).

Myocardial oxygen demand increases during exercise by increase in SBP, heart rate and contractile state. Normally, the heart extracts 70% O_2 from each unit blood perfusing the myocardium, so that the oxygen delivery can't be greatly increased by increased circulation (Faurtin et al 1977).

Myocardial metabolism is entirely aerobic therefore in order to increase myocardial O_2 delivery, coronary blood flow must increase directly in proportion to increased myocardial oxygen demands. (Faurtin et al 1977).

Exercise is the most convenient means of stimulating the myocardium to demand maximal blood flow and is the only way of stimulating such a vigorous demand for oxygen delivery (Braunwald 1988).

In normotensive persons the increase in heart rate accounts for the greatest increment in coronary blood flow during exercise. It is not possible to measure simply either coronary blood flow or the major determinants of mycardial oxygen demand during exercise, however some indication of the degree of exercise induced increase in myocardial O2 demands and by inference blood flow, can be provided by several indices which can be measured non invasively (Braunwald 1988).

The rate pressure product (heart rate x mean aortic pressrure) - the tension - time index (the integral of left ventricular pressure during systole x heart rate) - SBP, systolic ejection period have all been shown to correlate

with measured myocardial oxygen consumption during dynamic exercise (Nelson et al 1974).

The rate pressure product shows the best correlation of previous indices, although the heart rate is almost as good. For clinical purposes the heart rate or rate pressure product are easily measured indirect indices of myocardial oxygen consumption which reflects the degree of stress imposed on the coronary circulation by exercise (Faurtin et al 1977).

PATHO-PHYSIOLOGIC RESPONSES OF THE ISCHEMIC HEART TO EXERCISE TESTING