

EVALUATION OF CHEST PAIN IN THE MIDDLE AGED BY RATE PRESSURE PRODUCT ANALYSIS - A RETROSPECTIVE STUDY

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ABSTRACT

Background: Coronary artery disease (CAD) results from imbalance between oxygen supply and demand of the heart. Myocardial oxygen consumption (MVO_2) is a good indicator of the response of the coronary circulation to increased myocardial oxygen demand. Direct measurement of MVO_2 is difficult in routine clinical practice but it can be easily calculated by indirect methods like Rate Pressure Product (RPP) analysis. The internal myocardial work performed is represented by RPP and external myocardial work performed is generally expressed as stages of exercise.

Objective of the study: The objective of this study was to study rate pressure product in middle aged with non specific chest pain to isotonic exercise.

Materials and Methods: This is a retrospective cross sectional study carried out in the Department of Medicine, JSS Hospital, Mysore. The study group comprised of 43 patients in the age group of 40-50 years with a history of nonspecific chest pain but normotensive, nondiabetic and with normal resting ECG. Standard Bruce protocol was followed. The patients were divided into two groups - Group A (35 patients with typical angina) and Group B (8 patients with atypical angina). Statistical analysis was done using SPSS version 16.

Results: Mean RPP was decreased in patients with atypical angina (Group B) compared to patients with typical angina (Group A) ($p < 0.05$).

Conclusion: Angina is precipitated by an increase in work of the myocardium to a critical level that is essentially fixed in each patient. This increase in myocardial work is measured by RPP. Most normal individuals develop a RPP of 20 to 35 mm Hg \times beats/min $\times 10^{-3}$.

Keywords: Angina, Myocardial oxygen consumption, Rate Pressure Product, Treadmill test.

INTRODUCTION

Coronary artery disease (CAD) is known to be one of the major causes of morbidity and mortality in the Western population especially after the fifth decade of life. Over the past few years, CAD is increasingly diagnosed even in Asian countries, that too among the lesser demographic age group. Some reasons for this rise in disease burden maybe sedentary lifestyle, rapid urbanisation with the inevitable changes in food habits, increasing prevalence of obesity with associated risks of developing Hypertension, Diabetes Mellitus etc. Hence, non invasive methods to diagnose CAD in asymptomatic but high risk individuals, within the constraints of a basic clinical set-up, would be extremely useful in a country like India.

The contractile state of the heart, intra myocardial tension and heart rate are the major factors that determine myocardial oxygen consumption¹. The intra myocardial tension or wall tension is determined by the intra ventricular volume and the intra ventricular pressure, which can be estimated by Arterial Blood Pressure measurements.

Basal metabolism of the myocardium, activation energy and external work of the heart are all important but minor determinants of myocardial oxygen consumption.

The oxygen supply to the myocardium is determined by the volume and distribution of coronary blood flow, in addition to the difference in oxygen concentration between arterial and venous vasculature of the coronary circulation. In a healthy heart, this supply is greater than the oxygen demand of the heart. However, in ischemic hearts, there is a reversal of this relationship which means that the oxygen supply cannot keep up with the oxygen demand for a certain workload. This inadequacy results in ventricular dysfunction which causes pain, abnormalities in ECG and metabolic abnormalities.

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So, the primary pathology in CAD is impaired coronary circulation but the accelerated cell death in the aging myocardium leading to depletion of functional myocytes that decreases the contractile performance² also contributes.

Direct measurement of MVO_2 is difficult in routine clinical practice but it can be easily calculated by indirect methods like Stroke Work, Pick's Principle, Tension Time Index and Rate Pressure Product (RPP)³.

Rate pressure product (RPP) is an easily measurable index that correlates well with myocardial oxygen consumption (MVO_2). It defines the response of coronary circulation to the metabolic demands of the myocardium. RPP is the product of heart rate and systolic blood pressure.

$$RPP = \frac{\text{Systolic Blood Pressure (SBP)} \times \text{Heart Rate (HR)}^4}{1000}$$

It is a good index of MVO_2 in patients with ischaemic heart disease⁵. Rate pressure product is also called *Robinson index*⁶. The internal myocardial work performed is represented by RPP and external myocardial work performed is generally expressed as stages of exercise⁷.

MATERIALS AND METHODS

This is a retrospective cross sectional study conducted in the Treadmill Unit, Department of Medicine, JSS Hospital, Mysore. Approval from the institutional Ethical committee was obtained prior to the commencement of the study.

The source of data collection were those individuals presenting with a history of non-specific chest pain at the OPD of the Department of Medicine, JSS Hospital. The purpose of this study and the protocol were clearly explained to them. Simple random sampling was made among those individuals willing to participate in this study who also fulfilled the criteria for inclusion. Written informed consent was obtained from them.

Before exercise testing, a structured interview and chart review yielded data on symptoms, medications, coronary risk factors, prior cardiac events and a number of cardiac and non cardiac diagnoses.

Resting hypertension was defined as a resting Systolic blood pressure ≥ 140 mm Hg or a resting Diastolic blood pressure of ≥ 90 mm Hg or treatment with

antihypertensive medications⁷. Assessment of diabetes was based on history of medication usage, patient's symptoms, past investigations and family history.

The study group comprised of forty three patients who were between the ages 40-50 years of whom 30 were males and 13 females.

The inclusion criteria were:

- No previous history of Hypertension or Diabetes Mellitus
- Normal Electrocardiogram (ECG) at rest.

The exclusion criteria were:

- Evidence of recent myocardial infarction
- History of angina pectoris of recent onset
- A pronounced change in the severity or frequency of chest pain in a known case of angina

The study subjects were divided into two groups based on the quality of chest pain manifested.

Group A consisted of those patients with typical angina – 35 in number

Group B consisted of those patients with atypical angina - 8 in number.

Exercise Testing using treadmill was carried out according to standard Bruce protocol^{8,9}. To facilitate estimation of exercise capacity, leaning on handrails during exercise was discouraged. During each stage of exercise, data on symptoms, rhythm, heart rate, blood pressure (by indirect arm-cuff sphygmomanometer), estimated work load in metabolic equivalents (METs) and ST segments were collected and entered on-line.

Estimated functional capacity in METs was estimated from standard published tables⁹. Based on the protocol and total time completed in the final stage, a MET is the measure of oxygen consumption representing basal resting metabolic needs and equals 3.5 ml/kg/min. An ischaemic response was considered to be present if there was ≥ 1 mm of horizontal or down sloping ST-segment depression of 80 ms after the J-point or if there was ≥ 1 mm of additional ST-segment elevation in leads without pathologic Q waves. If a patient had more than one treadmill exercise echocardiogram performed during the study period, only the first one was considered for analysis.

RPP was computed as follows:

$$RPP = SBP \text{ (in mm Hg)} \times HR \text{ (in beats/min)} \times 10^{-3}$$

The RPP obtained at maximal exercise is called PRPP. The exercise test is said to be maximal when the subject appears to give true maximal effort i.e. effort done to the point of bodily exhaustion or when other clinical end points are reached. Exercise test was terminated in the subjects if the target heart rate was achieved or they complained of fatigue. It was also discontinued if there were abnormal changes like decrease in SBP of 10 mm Hg along with evidence of ischaemia, abnormal ECG pattern like ST segment displacement, appearance of arrhythmias or if the subject complained of chest pain.

Data was represented as Mean \pm Standard Deviation (SD). Analysis was done by independent 't' test using SPSS (version 16). p value < 0.05 was considered as statistically significant.

RESULTS

The characteristics of the two study groups and the number of subjects in each group is as shown by Table I.

Table I: Group Characteristics

Group	Chest Pain Characteristics	Number of Patients
Group A	With typical angina	35
Group B	With atypical angina	08

Table II shows changes in RPP in both the groups with exercise. The RPP of Group A was more than that of Group B both at baseline and after exercise. The rise in the RPP after exercise in Group A as compared to Group B was statistically significant (p < 0.05)

Table II: Comparison of Rate Pressure Product before and after exercise in patients of both groups

RPP (in mm Hg beats/min $\times 10^{-3}$)	Group	Mean \pm SD	p value
Before exercise	A	10.13 \pm 2.0	0.736
	B	9.86 \pm 2.4	
After exercise	A	22.43 \pm 3.2	0.017 **
	B	19.16 \pm 4.4	

** p < 0.01 is Highly Significant

DISCUSSION

Heart rate (HR) and Systolic Blood Pressure (SBP) are the most important variables determining changes in myocardial oxygen consumption between rest and exercise¹⁰. HR, SBP and RPP increases with increased workload on the heart, to provide adequate blood supply to the active myocardium during exercise.

As reported in earlier studies, there was significant increase in SBP, HR and RPP with exercise, due to increased sympathetic discharge^{7,11}. RPP increased progressively with exercise. This increase in RPP was significantly more in Group A as compared to group B indicating better coronary perfusion in Group A. The less increase in RPP in Group B suggests significant compromise in coronary perfusion in such patients with decreased left ventricular function.

Angina is precipitated by increased work of the myocardium to a critical value that is essentially fixed in each patient which is measured by RPP. Most normal individuals develop a RPP of 20 to 35 mm Hg \times beats/min $\times 10^{-3}$. Even in many patients with significant ischemic heart disease, RPP values exceeding 35 mm Hg \times beats/min $\times 10^{-3}$ are unusual¹². In the present study, RPP was 22 and 19 mm Hg \times beats/min $\times 10^{-3}$ in those patients with typical angina & with atypical angina respectively.

The peak RPP is an accurate reflection of the myocardial O₂ demand and workload. Reaching a high RPP without symptoms or evidence of severe ischemia suggests adequate left ventricular function and the low value of RPP suggests significant limitation of coronary perfusion and decreased left ventricular function leading to angina.

Although Rate Pressure Product does not predict regional myocardial demand and supply relationships, examination of the individual components (heart rate and systolic blood pressure) is useful in management of ischaemic heart disease. An increase in blood pressure without a change in heart rate appears to be better for myocardial oxygenation than an increase in heart rate along with the increase in blood pressure¹³.

Our findings are important in suggesting that apparently healthy subjects with low mean RPP response to exercise

are at higher risk for early subclinical atherosclerosis and myocardial ischaemia.

CONCLUSION

The Rate Pressure Product has been shown to be predictive of the risk of CAD in healthy populations even after adjusting for age, physical fitness and standard cardiovascular risk factors.

Our findings in this study are important as it suggests that apparently healthy subjects with low mean RPP response to exercise are at higher risk for early onset of subclinical atherosclerosis and myocardial ischaemia. Hence Rate Pressure Product is an useful tool for early detection of the coronary heart disease.

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