REVIEW

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Rate pressure product: The key index for cardiovascular health management

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ABSTRACT

In order to prevent and treat cardiovascular illnesses, cardiovascular health management is essential. The rate pressure product (RPP) is a crucial indicator frequently used to evaluate cardiovascular health. This index, calculated by multiplying the heart rate by the systolic blood pressure, provides valuable information regarding the workload of the heart and its oxygen demand. It serves as an important indicator of cardiac function and overall cardiovascular health. Several factors can affect RPP, including physical activity, emotional stress, and the use of certain medications. Despite its significance in assessing cardiovascular health, there is ongoing debate regarding the ideal range of RPP and its interpretation. This conflicting perspective on the interpretation of RPP raises important questions about its utility in evaluating cardiovascular health and the thresholds that should be considered normal or indicative of potential health issues. Further research and consensus among experts in the field are necessary to reconcile these conflicting views and provide clearer guidelines for the clinical use of RPP as a marker of cardiovascular health. In conclusion, RPP, a key cardiovascular health management index, provides valuable insights into cardiac function and overall cardiovascular health. Thus, it is an important index for monitoring and managing cardiovascular health.

Keywords: double product, exercise, myocardial oxygen consumption, rate pressure product.
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INTRODUCTION

An index known as the rate pressure product (RPP), also called the pressure-rate product, double product, or cardiac product, is used to assess the heart's oxygen demand. It sheds light on the autonomic nervous system activity, cardiovascular health, and hemodynamic responses to exercise. The heart rate (beats per minute) was multiplied by systolic blood pressure (SBP) to obtain this figure (mmHg).[1,2]

Studies have shown that the RPP is a valuable tool in assessing cardiovascular health and predicting the risk of coronary artery disease (CAD).[3-5] In addition to its value, the RPP has also been used to monitor the effectiveness of interventions such as exercise training and the pharmacological risk of CAD.[6-8]

RPP is correlated with prognosis and left ventricular function in patients with CAD.[3,4,9] Furthermore, understanding the nuances of RPP can aid in tailoring exercise prescriptions for individuals with or without heart conditions, ensuring that the intensity and duration of physical activity align with the individual's cardiovascular capacity.[10-13]

The comprehensive understanding and application of RPP are crucial in clinical and research settings, as they provide valuable insights into cardiovascular function and support informed decision-making in managing cardiovascular health. In the subsequent sections, we will further explore the specific implications of RPP in these clinical conditions, providing comprehensive insights into its role in cardiovascular health management.

THE CORRELATION BETWEEN RATE PRESSURE PRODUCT AND MYOCARDIAL OXYGEN CONSUMPTION

Research has demonstrated a strong correlation between myocardial oxygen consumption and RPP.[13-16] The quantity of oxygen that the heart muscle consumes is known as myocardial oxygen consumption (MVO_2). The heart beats to pump oxygen-rich blood throughout the body, including the muscles. Myocardial oxygen demand is primarily determined by heart rate (HR), contractility, and wall stress, which are influenced by ventricular pressure, chamber diameter, and wall thickness.[17,18]

In 1957, Gerola et al. proposed RPP as an index of MVO_2 based on experiments involving dogs with low oxygen levels.[15] Furthermore, Katz and Feinberg defined RPP as a measure of "cardiac effort" in canine hearts and demonstrated that it was linearly correlated with oxygen consumption.[14] Kitamura et al. validated this association between oxygen consumption and human hearts.[16]

The correlation between MVO_2 and RPP allows for a non-invasive method of assessing the heart's oxygen demand in clinical practice.[6,19] The use of RPP as an indicator of myocardial oxygen demand is supported by several sources. According to Gobel et al., the RPP, also known as the Robinson index, can be used as a simple and non-invasive method of calculating blood pressure (BP) and HR, which can estimate myocardial oxygen consumption.[6,20,21] Improvements in myocardial function, such as increased efficiency and reduced oxygen uptake, can be reflected by a lower

RPP during submaximal workloads.[22-24] A lower RPP indicates improved myocardial work efficiency.

RPP represents myocardial energy expenditure and is a reliable measure of myocardial oxygen uptake.[25] The RPP is strongly correlated with MVO_2 and is a simple parameter to measure, making it a valuable tool in clinical practice. Although RPP is a reliable predictor of MVO_2 and is simple to measure, some researchers have questioned its usefulness in clinical practice due to concerns about its accuracy and applicability in certain patient populations.[26,27]

An indirect index, RPP, can evaluate angina by theoretically estimating the relative MVO_2 without requiring invasive procedures, such as cardiac catheterization.[6,28] Using RPP in the context of angina can help elucidate the mechanisms of angina in different situations.[29,30] For example, exercise in the postprandial state or after smoking a cigarette is associated with an increased RPP, indicating an increased circulatory response during physical activity, which can lead to earlier provocation of angina.[24,31-33] These indirect indices can also be instrumental in evaluating the effectiveness of therapeutic interventions for angina by observing changes in circulatory response to physical stress.

However, the indices used by the RPP to assess MVO_2 must be interpreted cautiously, particularly when a complete heart block is present, and ventricular demand (VVI) pacing is utilized. According to the research conducted by Nordlander et al., SBP, recorded wedge pressures, and MVO_2 were comparable during exercise for both ventricular synchronized (VAT) and ventricular volume index (VVI) pacing. However, energy expenditure during fixed-rate pacing was likely attributed to factors other than recorded BP and HR, such as an increased contractile state and ventricular volume.[34]

Therefore, using these indirect indices with caution is important because of their inherent limitations, such as not accounting for ventricular volume and myocardial contractile state, which are important determinants of MVO_2. Despite these limitations, indirect indices of MVO_2 remain valuable tools for the non-invasive assessment of MVO_2 and the pathophysiology of angina pectoris.[30,35,36]

RESTING RATE PRESSURE PRODUCT AND ITS SIGNIFICANCE IN CARDIOVASCULAR HEALTH

Resting RPP is an important parameter for assessing cardiovascular health. Resting RPP is the value obtained when the body is at rest and provides insight into the performance of the cardiovascular system without the influence of exercise or physical exertion. This metric is crucial for understanding the efficiency of the heart and its ability to meet the body's oxygen demand during resting periods. Resting RPP is a valuable measure in both clinical and research settings to assess cardiac function and can be particularly useful in evaluating individuals at risk for or afflicted with cardiovascular disease.[23]

The MVO_2 index is higher in older people.[35] Old age can affect resting RPP owing to several age-related physiological changes. As individuals age, there is a general trend towards increased SBP and HR. Arterial stiffening, elevated systolic blood pressure, and thickening of the left ventricular wall are associated with cardiovascular aging. These factors can collectively contribute to an increased workload on the heart, resulting in a higher RPP.[36] Furthermore, with age, there may be an increase in total peripheral resistance, which can elevate BP and, subsequently, RPP.[37] These age-related changes in cardiovascular function can lead to increased RPP in older individuals.

According to Hui et al., resting RPP is lower in men than women due to a combination of physiological and lifestyle factors. Men typically exhibit lower body fat, greater physical activity, and greater aerobic capacity than women. These factors contribute to a lower resting HR and potentially lower SBP, the two components used to calculate RPP. Additionally, the study found that resting RPP was negatively related to aerobic capacity (VO_2max) and physical activity levels, which tended to be higher in men. This suggests that men consume less oxygen during rest than women, which is reflected in a lower resting RPP.[23]

The resting RPP was significantly higher in obese women than in non-obese women, indicating a higher cardiovascular risk in obese women. According to Segan et al., resting RPP was significantly higher in patients with diabetic cardiac autonomic neuropathy than in healthy controls and patients without neuropathy, indicating a higher myocardial oxygen demand at rest. In addition, the absence of a substantial increase in RPP during periods of exercise suggests that the cardiovascular stress response was impaired in these individuals.[38]

The myocardium receives a greater quantity of blood during exercise, which places a greater demand on the heart. Enhanced myocardial blood flow and oxygen consumption during exercise can induce alterations in RPP in healthy individuals. An RPP value between 7000 and 9000 is considered safe, whereas a value exceeding 10,000 at rest signifies an elevated risk of cardiovascular disease.[39-42]

THE CORRELATION OF RATE PRESSURE PRODUCT AND SILENT MYOCARDIAL ISCHEMIA

Silent myocardial ischemia (SMI) is when the heart muscle does not receive sufficient oxygen but does not produce observable symptoms such as chest pain. This state is frequently identified through changes in electrocardiogram patterns during stress testing or ambulatory ECG monitoring.[43] To investigate the relationship between RPP and SMI, examining the impact of RPP on myocardial oxygen demand and the possible effects of silent ischemia on the heart muscle is crucial.

Research suggests a correlation between RPP and silent ischemic myocardium.[30] Gobel broadens the examination of the hemodynamic determinants of myocardial oxygen use beyond healthy individuals by including patients with ischemic heart disease. An increase in the RPP indicates latent myocardial ischemia, a marker of myocardial oxygen delivery.[6] Moreover, Deedwania et al. concluded that an increase in myocardial oxygen demand during daily life significantly contributes to the development of SMI in men with a history of chronic stable angina and confirmed CAD. This suggests that an elevation in HR and BP preceding silent ischemic events indicates that myocardial oxygen demand is involved in the pathophysiology of silent ischemia.[44]

The most important indicators of ST-segment depression in hypertension individuals were elevated SBP, HR, RPP, and pulse pressure. According to research by Uen et al., an increase in RPP is a strong predictor of ST-segment events. This suggests that myocardial oxygen demand, which is indicated by the RPP, is elevated during SMI episodes.[45]

A worse prognosis has been linked to emotional distress among patients who have suffered myocardial infarction. Workload and SMI may correlate with elevated adrenocorticotrophic hormone (ACTH), decreased cortisol, dehydroepiandrosterone sulfate (DHEAS), and cortisol:DHEAS ratio, all of which are indicators of emotional distress. A prospective study of Sympathetic activity and Ambulatory BP in Africans (SABPA) indicated that there is a dysregulated hypothalamic-pituitary-adrenal axis (HPAA) and compensatory increases in RPP among black males. This increase in RPP may serve as a defense mechanism to mitigate perfusion deficits, thus increasing the risk of ischemic heart disease.[46]

In some studies, an RPP exceeding 22,000 has been commonly associated with myocardial ischemia.[6,30] Several studies support a correlation between RPP and silent ischemic myocardium. For example, in one study, the maximal RPP values in the two groups did not exceed 15,000, indicating a lower risk of myocardial ischemia.[47] Therefore, it can be concluded that a higher RPP is associated with an increased risk of silent myocardial ischemia. However, the existing research has not sufficiently supported the correlation between RPP and SMI.

RPP can be used as an ischemic threshold in patients with chronic stable angina and CAD. In their study, Pupita et al. observed a substantial increase in RPP from 18,813 ± 3682 beats/min x mmHg during the standard modified Bruce exercise test to 20,357 ± 4227 beats/min x mmHg when a 10-minute warm-up period was implemented prior to the modified Bruce protocol. This finding implies that warm-up exercises may elevate the ischemic threshold in certain patients. Following the warm-up, the RPP at 1 mm ST segment depression increased by a minimum of 2000 beats/min × mm Hg in eight of the 16 patients.[48]

RATE PRESSURE PRODUCT IN VARIOUS TYPES OF EXERCISE

RPP plays a significant role in exercise physiology.[49] During exercise, the demand for oxygen by the working muscles and organs increases. The heart must pump more blood and increase its contraction rate to fulfill this increased demand. As a result, the HR and BP increase, leading to an increase in RPP.[50]

An increase in RPP during exercise reflects a greater workload on the heart.[31,51,52] This elevated RPP indicates a higher demand for oxygen by the myocardium, as the heart needs to supply more oxygenated blood to the body's tissues during physical activity. Monitoring changes in RPP during exercise can help evaluate the cardiovascular response to different levels of physical exertion.[24,53]

Research has shown that individuals with higher RPP during submaximal exercise may be at greater risk for cardiovascular events.[24] High-intensity aerobic training (using a bicycle ergometer, exercising at 80% of their maximum HR for 15 minutes per day, five days a week for 12 weeks) led to a significant reduction in RPP, indicating improved cardiac fitness compared to low-intensity aerobic training.[40] The effect of high- and low-intensity aerobic training on RPP was also conducted by Madhusudhan, with results showing that high-intensity aerobic training (80% HR max) led to a significant reduction in resting RPP, indicating improved cardiac fitness, compared to low-intensity aerobic training (50% HR max) in 80 sedentary men aged 18-40 years.[40]

RPP was lower during recovery after aerobic exercise training (AT) than during weight training (WT) and no treatment (NT), suggesting that aerobic exercise training may lead to a reduction in RPP, which is a measure of MVO_2 and cardiac workload. In addition, Steffen et al. found that weight management markedly reduced RPP responses with both high and low levels of physical exertion.[54] Fatigue reduction was associated with a lower RPP during submaximal walking in breast cancer survivors.[22]

Progressive resistance training (RT) significantly reduced resting SBP, mean arterial pressure, and RPP in elderly women with hypertension, suggesting that RT is an effective intervention for managing hypertension in this population. The reduction in RPP highlights the potential cardiovascular benefits of RT in reducing the stress on the heart at rest.[55] However, research by Soltani et al. added that the interval RT protocol resulted in a greater increase in RPP compared to the traditional RT protocol.[56]

Neto et al. showed that low-intensity RT combined with blood flow restriction (BFR) appears to reduce post-exercise oxygen saturation (SpO2), HR, and RPP while maintaining the perception of greater lower limb exertion.[12] On the other hand, Zheng et al. found that there were no significant differences in brachial RPP responses between the low-intensity RT session and the low-intensity RT with BFR session.[57]

The oxygen consumption of post-myocardial infarction (MI) patients during exercise differed significantly from that of healthy subjects. This suggests that caution should be exercised when prescribing exercise based on energy expenditure measured in normal subjects. Swimming resulted in significantly higher oxygen consumption. Following each activity, there is an increase in oxygen consumption and a decrease in mean RPP (a hemodynamic response measure), which indicates enhanced cellular oxygen delivery and extraction.[42]

May and Nagle analyzed changes in estimated maximal aerobic capacity (VO_2max), MRPP, and submaximal RPP (at the four-MET level) from initial to final tests in subjects with myocardial infarction or severe myocardial ischemia and compared these changes between an experimental group that underwent regular supervised aerobic exercise and a control group that did not participate in regular exercise. The conclusion related to RPP is that for the experimental group, there was a significant increase in MRPP and a significant decrease in submaximal RPP after the training period. This suggests that physiological changes in the myocardium and exercising skeletal muscles contribute to increased maximal work capacity and symptomatic improvements in individuals with CAD following regular aerobic exercise. These data indicate that the ischemic heart experiences a reduction in the disparity between oxygen demand and supply after training. This is supported by an increase in maximal RPP and a decrease in submaximal RPP, which indicate a decrease in oxygen demand.[49]

Particular attention should be paid when formulating exercise prescriptions, particularly for patients with CAD. MacMasters et al. studied the physiological responses, particularly the cardiac workload, of performing arm exercises at different pedal speeds using an isokinetic exercise machine to determine which speed was less stressful to the heart. According to the findings of this study, an arm exercise performed at a pedal speed of 60 rpm resulted in a significantly reduced RPP (heart stress) compared with exercises performed at 30 or 90 rpm while maintaining a workload of 600 kg.m/min. It is advisable to exercise caution when using low (30 rpm) and high (90 rpm) pedaling speeds in studies involving patients diagnosed with heart disease. Individuals participating in a general fitness program who prefer a higher exercise HR may benefit from these speeds.[58]

Exercise intensity variations may have various consequences on post-exercise RPP, BP, and HR responses during recuperation. Forjaz et al. investigated how young people without hypertension responded cardiovascularly after exercising on a cycle ergometer. At 30% of VO_2peak exercise, RPP was lower than baseline levels; however, activity at 50% and 80% of VO2peak did not result in a reduction of RPP after exercise.[59]

Regarding the breaking point of the RPP, when the intensity of the exercise increased, the rate of increase slowed. Kim et al. proposed that a breakpoint exists during a stepwise incremental exercise, where the RPP increases abruptly. This breakpoint appeared at a lower workload (56 watts), indicating that RPP, a measure of myocardial workload, shows a significant change at a certain point of low exercise intensity and is significantly related to the increase in HR during exercise.[53]

Multiple researchers have documented that a breakpoint in the RPP corresponding to the lactate threshold (LT) was observed in healthy subjects during incremental exercise.[58,59] According to Omiya et al., during incremental exercise, the ventilatory threshold (VT) or lactate threshold (LT) may not be as effective as the RPP breakpoint in indicating exercise intensity in individuals with heart disease. Additionally, the RPP breakpoint in cardiac patients undergoing incremental exercise may occur near the VT or LT, and the non-invasive approach to measuring the breakpoint is comparable to the intrusive one.[60] Historically, LT and VT have been utilized as indicators of the anaerobic threshold (AT).[61] AT has been utilized for various purposes, including exercise prescription and predicting endurance performance in athletes and cardiac patients.[11,62] In addition, the RPP breakpoint also allows for the prediction of AT in individuals with type 2 diabetes (T2D).[63]

According to Hargens et al., the RPP breakpoint reacts to exercise training in a manner akin to that of VT and could be a more straightforward and practical indicator of VT for exercise training.[11] Although previous research conducted by Pinkstaff et al. has shown that the present percentage of peak RPP thresholds (25,000) is useless in assessing the intensity of exertion during an exercise stress test.[64]

One of the non-pharmacological modalities for the cardiovascular system is breathing exercises.[65,66] By stimulating the vagus nerve and the principal nerve of the parasympathetic nervous system, alternate nostril breathing helps to slow the HR, reduce BP, and relax the body and mind.[67] Kalaivani et al. have shown that alternate nostril breathing exercises significantly reduce SBP, diastolic BP (DBP), HR, and RPP among patients with hypertension when practiced alongside routine treatment. Therefore, when compared to conventional methods of treating hypertension, alternating nostril breathing exercises demonstrate efficacy and cost-effectiveness as an intervention to decrease cardiac workload in individuals with hypertension.[68]

The isometric or static exercise consists of sustained muscle contraction without commensurate alteration to the length of the target muscle group. Isometric exercise training may produce greater average reductions in ambient BP than cardiorespiratory exercise, according to a recent meta-analysis.[69] Isometric exercise training, specifically isometric handgrip training, possesses the potential to serve as a beneficial supplementary therapeutic element within the comprehensive approach to hypertension management due to its broad applicability, affordability, and global accessibility. Sustaining a healthy weight and achieving even a marginal reduction in systemic arterial pressure can substantially mitigate morbidity and mortality in patients who have cardiovascular risk factors.[70] Keshari et al. discovered that non-obese individuals reported more significant increases in HR, SBP, and DBP during an isometric handgrip exercise than obese individuals. Isometric handgrip training has the potential to reduce the risk of cardiovascular complications by facilitating the early detection of autonomic dysfunction in obese individuals.[71] However, related explicitly to RPP, there was a significant increase in RPP during static exercise at altitude, indicating increased myocardial demand.[72]

RATE PRESSURE PRODUCT AS A PREDICTOR FOR CARDIOVASCULAR EVENTS

Research evidence suggests that RPP can serve as a predictor of cardiovascular (CV) events.[73] Whitman et al. demonstrated that MRPP may be a more dependable predictor of cardiovascular events and a more accurate measure of cardiac workload during exercise stress echocardiography (ESE) than age-predicted maximum HR (APMHR) alone. Those who achieved an APMHR greater than 85% but an MRPP less than 25,060 had significantly more CV events than those who achieved an MRPP greater than 25,060, regardless of the APMHR. This suggests that MRPP is a more reliable marker than APMHR for predicting future CV events.[74]

Prior research by Whitman et al.

established that patients who attain an MRPP greater than 25,000 during an exercise stress test and do not exhibit signs of myocardial ischemia do not require additional diagnostic procedures, irrespective of their functional capacity. Moreover, the prognosis for this cohort of patients is superior to that of those whose MRPP falls below 25,000.[75] Cardiovascular events in patients undergoing exercise stress testing for myocardial ischemia are significantly more prognostically significant with MRPP than with APMHR and HR reserve (HRR).[9]

Conversely, an examination of the prognostic utility of RPP revealed that it does not contribute anything beyond what is already known from SBP or pulse rate (PR) in predicting CV risk. Compared to the PR, the RPP exhibited a predictive value for CV mortality and events that were either equivalent to or diminished compared to the SBP.[76] A retrospective review of patients undergoing exercise treadmill testing (ETT) up to 30 days before coronary angiography for suspected obstructive illness revealed RPP greater than 30,000. In patients with a positive ETT, this value is important in predicting the lack of major obstructive CAD and functions as a useful tool for clinical decision-making.[77]

Elevated values of RPP during maximal exertion have been hypothesized to indicate healthy ventricular function and the absence of ischemia.[77] Villella et al. published the findings of a study conducted on 6,251 acute myocardial infarction survivors from the GISSI-2 database who were followed for six months after completing a maximal symptom-limited exercise test on a treadmill or bicycle ergometer. The results showed that limited labor capability, low RPP, and symptomatic exercise-induced ischemia were still significantly linked to a higher 6-month death rate. In addition, patients with a low shift in RPP from rest to maximal exercise (dRPP) had greater death rates than patients with a high dRPP.[78]

Without relying on conventional risk factors or exercise test responses, Rafie et al. validated that dRPP can predict prognosis more accurately than HR recovery, maximal HR, exercise capacity (METs), or SBP. In addition, Duke Treadmill Score (DTS), age, and dRPP were independent predictors of cardiovascular mortality. Elevated age and diminished dRPP were correlated with a heightened risk.[79,80]

In comparison to healthy non-pregnant women, Teli et al. found that an increase in RPP during all trimesters of pregnancy was a helpful predictor for the early diagnosis of preeclampsia and acute myocardial infarction in pregnant women. It has been suggested that monitoring RPP could be a useful tactic to enable prompt response to issues.[81] Moreover, patients who have undergone percutaneous coronary intervention (PCI) for acute coronary syndrome (ACS) and exhibit elevated RPP values are correlated with major adverse cardiovascular events (MACEs). However, the predictive value of RPP decreased when HR was accounted for. The study concluded that RPP may, therefore, reflect the prognostic value of HR in patients with ACS who are treated with PCI.[82]

The predictive value of RPP for CV events was also assessed in individuals diagnosed with diabetes mellitus. In individuals with CAD and type 2 diabetes, RPP recovery 5 min after exercise is a substantial predictor of cardiac mortality. There is an independent association between delayed RPP recovery and cardiac mortality, as well as sudden cardiac death (SCD). It was discovered to offer prognostic insights that extended beyond exercise capacity and maximal RPP.[83] Between July 1997 and January 2000, Yazdani et al. conducted a monocentric hospital-based cohort study of 3316 patients referred for coronary angiography. Pulse pressure and RPP are robust predictors of cardiovascular mortality and all-cause mortality in patients with CAD and heart failure (HF) who are at intermediate to high cardiovascular risk, according to the study's findings. RPP remained an independent predictor of cardiovascular and all-cause mortality despite adjustments for other factors. Conversely, pulse pressure did not exhibit predictive power independent of antihypertensive treatment in the study cohort.[84]

However, a retrospective cohort study by Hagel et al. found that RPP was not a predictor of adverse outcomes (cardiopulmonary resuscitation, extracorporeal cardiopulmonary resuscitation, mechanical circulatory support, unplanned surgery, heart transplantation, or death) after pediatric cardiac surgery.[85] Concerning heart transplants, Rodrigues Junior et al. showed that RPP and HR variability (HRV) were identified as predictors of quality of life in heart transplant recipients. These findings suggest that RPP and HRV could be targeted for therapeutic interventions to improve the quality of life of heart transplant recipients.[86]

Jiang et al. conducted a study to determine whether RPP could predict long-term adverse outcomes in patients with CAD who underwent PCI. According to the findings of this study, RPP was an independent predictor of long-term adverse outcomes in CAD patients who underwent PCI. A greater RPP at baseline before PCI was correlated with a greater likelihood of adverse outcomes. The predictive value of RPP for adverse clinical outcomes was greater than that of HR and BP alone. RPP can function as an innovative long-term prognostic indicator in patients with CAD who have undergone PCI.[3]

RPP may be a more powerful measure than BP or HR alone in predicting cardiovascular morbidity and mortality, and its normal limits and correlation with health outcomes should be established through a reanalysis of existing data.[73] However, this approach has several limitations that must be considered when adopting a holistic approach to cardiovascular health assessment. To address these limitations, a more holistic approach to cardiovascular health assessment is necessary. This may involve the integration of multiple biomarkers, including vascular function, cardiac structure and function, and metabolic parameters.

CONCLUSION

RPP plays a crucial role in cardiovascular event prevention by providing insight into myocardial oxygen demand, guiding exercise prescriptions, and identifying potential risks for heart disease. By monitoring and managing RPP, individuals can make informed decisions regarding their exercise and physical activity levels to optimize cardiovascular health and prevent heart disease.

While RPP is considered the product of HR and SBP, it does not incorporate factors such as diastolic blood pressure, exercise capacity, or individual variations in the cardiovascular response. In conclusion, although RPP can be a valuable tool for cardiovascular health, it is essential to consider its limitations and potential drawbacks. Healthcare professionals need to adopt a holistic approach to cardiovascular health assessment, considering a range of physiological parameters and individual variations in cardiovascular responses.

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Author Contribution

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Ethical Considerations

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