Research Open

Internal Medicine Research – Open Journal
Volume 2 Issue 1

Review Article

The Usefulness of the Rate Pressure Product (RPP) for Cardiac Rehabilitation Exercise Prescription

Sherldine Tomlinson

Department of Sport and Exercise Science, Faculty of Health Science, Staffordshire University, Stoke on Trent, Staffordshire ST4 2DE, UK;
Email: srtomlinson@students.ussa.edu

Received: March 10, 2017; **Accepted:** March 30, 2017; **Published:** April 28, 2017;

Introduction

The autonomic nervous system (ANS) is an arm of the nervous system surrounded by the peripheral nervous system (PN) and the central nervous system (CNS). It is responsible for the regulation of involuntary bodily functions such as the beating of the heart to the way in which food is digested [1-4]. This system further separates into two division: the sympathetic nerves (SNs) and the parasympathetic nerves (PNs) with each carrying efferent (motor) signals to the heart and afferent responses to the brain [2,3]. In maintaining the body's homeostasis, each nerve fibre triggers internal or external stimulus. Stimulations coming from the SNs releases epinephrine and norepinephrine prepare the body for stressful or emergency situations or what is best known as the fight or flight state [2]. SNs activities increase heart rate, cardiac output, contractility, conduction velocity and blood pressure during physical stimuli such as exercise [2]. SNs also makes the palm of the hand sweat, the pupils to dilate, and causes the hair on the body to stand on end [2].

In contrast, the PNs which originate from the brain stem and the sacral portion of the spinal cord releases acetylcholine to conserve energy during normal relaxed situations [2]. The efferent outflow termed the vagus nerve operates the parasympathetic to transmit nerves fibres to the lung, heart and other organs [2, 3,]. These nerves work to lower blood pressure (BP), to slow the heart rate (HR) down and to control digestive functions [2].

The SNs and PNs work in opposite direction of each other and as such the SNs enhance automaticity, while the PNs inhabit it [5]. A good demonstration of SNs and PNs operating in opposed action would be if the heart receives a neural stimulus from the parasympathetic branch; it would slow the heart down whereas sympathetic activities would speed up the heart. There is a wide consent suggesting that any changes between the systems play a role in pathological dysfunctions of the ANS [3-5]. For example, a cascade of adverse cardiac events takes place if parasympathetic vagal tone decreases [3, 5-6]. Hypertension-related diseases, coronary heart disease, heart failure and myocardial ischemia are various heart conditions caused by chronic SNs activation [5-8]. The shifts to a more sympathetic overdrive are a catastrophe for ANS impairment. As such any treatment whether by drug action or with exercise training that tilts the autonomic balance toward greater parasympathetic dominance and less sympathetic activity significantly improve prognosis [3].

Moreover, cardiovascular autonomic functions or dysfunctions are clinically evaluated by measuring resting heart rate (RHR), heart rate (HR), BP or heart rate recovery (HRR) [6]. Whether done directly or indirectly these autonomic parametres are good indicators in determining how the heart is working during conditions like exercise or stress [6,7,5,9]. In clinical practices, more specifically in cardiac rehabilitation (CR), several autonomic parametres are used in assessing patients ANS function and their physical capacity. Unfortunately, because of the difficulties and the lack of experience to perform some tests, and the time it takes to do the test, some methods are not applicable in CR setting [4].

In looking at different nerve simulations, clinical research studies found that ANS parametres were risk markers for cardiovascular diseases [5,7,9]. A decline in heart rate variability (HRV), for example, was associated with many cardiac conditions including sudden death [9]. Indeed clinical procedures like HRV in monitoring autonomic processes are necessary with patients. They are practical to check if the ANS is operating normally or to see if a disease or disorders are attacking the system. In this paper, we briefly look at the RPP and how the usefulness of this autonomic test is to CR. The RPP has been quoted in the literature, but it is now accepted as a reliable tool for making clinical decisions for exercise prescription.

Cardiac Rehabilitation and Exercise Testing (ET)

Cardiac rehabilitation is an outpatient health programme delivered by a multidisciplinary team of health professionals (i.e. physicians, nurses, exercise physiologist, dieticians) following cardiac incidents [10-13]. The plan typically provides a multifaceted offering of health services such as low to moderate exercise training, health education, risk factor modifications, counselling and social services [10-12]. The objective of CR is to enhance secondary prevention by lessening cardiac symptoms thereby reducing cardiac mortalities and morbidities for patients with cardiovascular disease [13]. Evidence demonstrate the efficacy of CR interventions where these schemes have improved patient's quality of life (i.e. reduce depression, better risk profile, enhanced functional status) [12-14]. In one study a CR exercise-based programme was safe to improve cardiopulmonary function with patients who had preserved left ventricular ejection fraction (LVEF) and reduced LVEF [15]. In another research, the authors suggested following coronary artery bypass surgery exercise

has the potential to better the long-term prognosis and lower the need for hospital care in cardiac patients [16]. CR is indicative in supporting cardiac autonomic functions to improve the long-term health and well-being of cardiac patients and their families.

Exercise prescription in CR is a determinant on patient's ET results. Before the start of a CR programme, it is standard practice for all patients to undergo clinical assessments which include ET [11]. With a goal to boost patient's clinical outcome, ET is done by evaluating left ventricular function (LVF) using an echocardiography or with a maximal exercise test limited by symptoms [11,17-20]. Before the beginning and ending of the programme, ET is the most critical testing component in CR. It provides plenty of information about patients' functional capacity, their hemodynamic adaptation to maximal and submaximal levels of exercise HR and BP, their residual myocardial ischemia, and their cardiac arrhythmias which can be either induced or worsens with activity [20]. CR exercise testing also let us knows the amount needed to calculate patients training heart rate (THR) for the aerobic exercise [20].

The cardiopulmonary graded test or CPX is the gold standard and approved method used for CR exercise testing. Testing is conducted by treadmill walking, ergometre cycling, stepping, or performing a 12 minute timed walking test [11, 18-20]. During the CPX, patient's peak oxygen uptake ($VO_{2peak)}$, their anaerobic threshold, their VE/VCO_2 and O_2 are observed as well as other parametres such as their maximal workload, and their resting and exercised BP and HR [20]. VO_{2peak} is the most frequently analysed CPX parametre as it determines patients' functional capacity, and it is the strongest prognostic for cardiovascular disease [20]. VO_{2peak} provides information on exercise intensity with a percentage of 50% to 70% the most acceptable [18, 20]. Under the supervision of a healthcare professional patient's workload is monitored at various exercise stage [18, 20]. They are asked about the perception of exercise intensity using the well-known Borg Rating of Perceived Exertion Scale (RPE) [11, 20]. Furthermore, it is advisable the patient completes each stages of exercise [11]. However, with their discretion, the physician or cardiologist could terminate the test at a particular heart rate or at the request of the patient [11].

Following the completion of ET patients HR, BP and their total VO_{2peak} are recorded and analysed [20]. As a component of functional capacity, the VO_{2peak} decides exercise prescription and is cited as an independent predictor of all-cause mortality in patients with cardiac conditions [11, 20]. After they have been discharged from CR, studies show patient's functional capacity gets better [20]. The VO_{2peak} test appears to be a valuable clinical assessment in the planning of patients' management. If the test is not available to measure patients' fitness capacity, the one metabolic equivalent (MET) formula is applied [11, 20]. The one METs is a very simple procedure to express the energy cost of physical activities as multiples of resting metabolic rate [11]. It is a measurement of the exertion intensity of physical activity, and it is defined by the amount of oxygen consumed while sitting quietly at rest and is equal to 3.5 ml O_2 per kg body weight x min (i.e. 3.5 ml O_2/kg/min) [11, 20]. For example, a physical activity requiring an 8-MET resting metabolic rate represents a VO_2 of 28 ml $\cdot$ $kg^{-1} \cdot min^{-1}$. In calculating the absolute oxygen requirement of the activity with 8 MET the individual's body weight is multiplied by the VO_2 ($kg^{-1} \cdot min^{-1}$) (i.e. VO_2 ($kg^{-1} \cdot min^{-1}$ =28 $kg^{-1} \cdot min^{-1}$ x 70 kg = 1.960 ml $\cdot$ min^{-1}). A noted feature of the metabolic equivalent is that men and women do not produce the same values (i.e. METs= 14.7 – 0.11 x age for males and respectively 14.7 – 0.13 x age for females.) [20]. This gender difference in computation accounts for women's having lower level of work capacity [11].

As mentioned earlier, patients' exertion level in CR is estimated from the RPE scale. The scale ranges from 6 to 20, but the American Association of Cardiovascular and Pulmonary Rehabilitation (AACPR) suggest a RPE of 11 to 15 as a safe zone for patients [11]. An important characteristic of the RPE is that it works linearly with HR and with exercise intensity [11,21]. As such you can estimate the HR value of various levels of work intensity by adding a zero to each point on the PRE [21]. For instance, RPE of 6 becomes 60 and represents HR at rest, and 20 becomes 200, which may represent patients maximal HR [21]. Subsequently, you can use the RPP to know patients' maximal HR and training workload. Case in point, if a patient develops some discomfort in the chest (i.e. angina pectoris) at a given level of exercise intensity, for safety the CR health professional should advise the patient to workout at a lower intensity. In this way, it would help to keep his training HR below the threshold where he may experience physical symptoms. The RPP is complementary to the RPE while having the ability to support safe CR exercise prescription for cardiac patients who might experience mild chest pain while they are exercising.

Rate Pressure Product (RPP)

To determine the energy requirement and establish the amount of stress put on the heart during exercise, cardiac specialist or exercise physiologists use the RPP. It is an observation of myocardial oxygen consumption (MVO_2) [22-26] representing the internal myocardial workload when the heart beats while the external myocardial work is a reflection of different stages of exercise. [27]. Expressed as the product of systolic blood pressure (SBP) and resting heart rate, you can calculate the RPP by multiplying the SBP by the RHR and dividing by 100 (i.e. RPP = SBP x HR/100) [21-27]. PNs and SNs mediate both HR and SBP with SBP only affected by SN [25]. What's more, depending on the individual physical or health condition RPP score may vary. Fornitano and de Godoy suggested RPP above 30,000 mmHg bpm are good values to predict the absence of obstructive coronary artery disease in patients with positive ET [27].

Heart Rate and Blood Pressure on Exercise Training

The heart needs sufficient amount of oxygen to work properly, and if there is not enough supply, it will cause the heart to weaken (e.g. heart failure) [22]. In this case, the RRP is important in providing information on patients' myocardial oxygen consumption [20]. Blood pressure and HR is a determinant of physical fitness since they both increase during exercise, but not at the same pace [20, 24, 25]. In subjects with BP between 110 to 120 systolic and 60-80 diastolic whereas resting heart rate (RHR) is 65 to 70 beats per minute (bpm) is considered normal [26]. Under these conditions, the heart does not need to work as hard because the oxygen demand is less [26].

Conversely, in patients with BP over 140/90 mm Hg and an RHR of 85 bpm or higher the heart works harder as it requires more oxygen [26].

Typically, an increase in HR during exercise is a sign that more blood and oxygen is travelling to the working muscles, while elevated BP indicates more blood gets pumped to the heart [24, 26]. As noted, increased BP and HR do not occur at the same time. Thus, a rise in HR triggers blood vessels to widen which in turn helps to keep BP under control [24]. This situation is why healthy people can recover much faster from exercise as compared to someone with a medical condition [20, 22]. The quick recovery is also a sign that there is more parasympathetic vagal tone and less sympathetic activity, which also accounts for the reduction in HR [22].

Extensive clinical and rehabilitation studies on the impact of RPP noted its efficacy as a reliable index to assess patients' with cardiovascular conditions or related complications on myocardial oxygen consumption during their exercise. Coelho and colleague identified positive changes in MVO_2 values following training in patients with ischemic heart disease [28]. Keyhani and co-authors investigated the effects of an eight-week CR aerobic exercise programme on BP, HR, and RPP in patients with congestive heart failure (CHF) found their cardiac functional capacity improved as well as their autonomic function [29]. Still, Adams et al. compared peak RPP values with various modes of aerobic exercise after CR training discovered treadmill walking to associate with a higher score while resistance training produced a much lower number [21].

Looking at the RPP and autonomic responses of Tai Chi practitioners and non-practitioners at rest and using two different stressors: hand gripping and standing Figueroa and colleagues saw improved autonomic function (i.e. parasympathetic tone) with the Tai Chi group [27]. The Tai Chi practitioners' sympathetic outflow and RPP were also significantly lower at rest suggesting they were better efficient in myocardial oxygen use during resting and pathological stress [27]. The positive outcomes are a testament that the RPP is a reliable tool and an acceptable approach in observing patient's cardiac autonomic exercise responses that favour greater parasympathetic tone.

The magnitude and the time BP and HR changes after the cessation of exercise are not without discrepancies [28]. When compared to pre-exercise during the first hour of recovery, Somers et al. [30] found lower BP levels, whilst Pescatello et al. [30] saw a significant fall that was up to 12 hours following exercise. Equally, post-exercise HR was reported to enhance, cause no change or decrease [30]. These observations give us a hint that different exercise intensity, duration, and mode significantly influence BP and HR responses following training. Forjaz et al. [31] study recognised that exercise training at a lower intensity, does not only generate a small increase in RPP during exercise, but it also decreases post-exercise rate at rest. By doing so, this reduces myocardial oxygen consumption and lower cardiovascular risks after exercise. In regards to exercise at moderate or high intensity, RPP appears to be greater during training but it decreases below baseline following the recovery period [31]. As cited RPP varies with exercise and there is evident it has clinical implications in providing exercise prescription with those experiencing medical conditions [30].

Conclusion

In addition to standardised CR exercise tests, the RPP supplements with other ET. This autonomic measurement is efficient as gives clues and evaluates patient's physical or cardiac functional capacity, exercise tolerance and oxygen demand during CR exercise testing and training. Importantly, its utilisation offers support to CR health professionals in selecting the right exercise intensity or training method for those patients whom may show cardiac risk.

References

1. Shields RW Jr (1993) Functional anatomy of the autonomic nervous system. *J Clin Neurophysiol* 10: 2–13. [crossref]
2. Freeman JV, Dewey FE, Hadley DM, Meyers J, Froelicher VF (2006) Autonomic nervous system interaction with the cardiovascular system during exercise. *Pro ardiovasc Dis* 48: 342–362.
3. Curtis BM, O'Keefe JH Jr (2002) Autonomic tone as a cardiovascular risk factor: the dangers of chronic fight or flight. *Mayo Clin Proc* 77: 45–54. [crossref]
4. Zygmunt A, Stanczyk J (2010) Methods of evaluation of autonomic nervous system function. *Arch Med Sci* 6: 11–18. [crossref]
5. Sztajzel J (2004) Heart rate variability: a noninvasive electrocardiographic method to measure the autonomic nervous system. *Swiss Med Wkly* 134: 514–522.
6. Martin B, Papelier Y, Laursen PB, Ahmaidi S (2007) Noninvasive assessment of cardiac parasympathetic function: postexercise heart rate recovery or heart rate variability? *Am Jf Physiol Heart Circ Physiol* 293: H8–H10.
7. Ruediger H, Seibt R, Scheuch K, Krause M, Alam S (2004) Sympathetic and parasympathetic activation in heart rate variability in male hypertensive patients under mental stress. *J Hum Hypertens* 18: 307–315.
8. Palatini P, Julius S (2009) The role of cardiac autonomic function in hypertension and cardiovascular disease. *Curr Hypertens Rep* 11: 199–205. [crossref]
9. Del Pozo JM, Gevirtz RN, Scher B, Guarneri E (2004) Biofeedback treatment increases heart rate variability in patients with known coronary artery disease. *Am Heart J* 147: 545.
10. National Health and Medical Research Council: Strengthening cardiac rehabilitation and secondary prevention for Aboriginal and Torres Strait Islander Peoples: a guide for health professionals. Canberra, Australia: Australian Government; 2005.
11. Goble AJ, Worcester M (1999) Best practice guidelines for cardiac rehabilitation and secondary prevention. Victoria, Australia: Heart Research Centre - on behalf of Department of Human Services Victoria
12. Cardiac rehabilitation: a model of care for South Australia. Australia: South Australia Department of Health, Statewide Service Strategy division.
13. Cardiac rehabilitation: evidence-based best practice guidelines. New Zealand: Heart Foundation; 2002.
14. Williams MA, Ades PA, Hamm LF, Keteyian SJ, LaFontaine TP, et al. (2006) Clinical evidence for a health benefit from cardiac rehabilitation: an update. *Am Heart J* 152: 835–841. [crossref]
15. Chul K, Choi HE, Lim YJ (2016) The effect of cardiac rehabilitation exercise training on cardiopulmonary function in ischemic cardiomyopathy with reduced left ventricular ejection fraction. *Ann Rehabil Med* 40: 647–656.
16. Hedbäck B, Perk J, Hörnblad M, Ohlsson U (2001) Cardiac rehabilitation after coronary artery bypass surgery: 10-year results on mortality, morbidity and readmissions to hospital. *J Cardiovasc Risk* 8: 153–158.
17. Zoneraich S (1983) Exercise testing and cardiac rehabilitation in patients with coronary artery disease. *Bull N Y Acad Med* 59: 635–659. [crossref]
18. Pina IL, Balady GJ, Hanson P, Labovitz AJ, Madonna DW, Myers J (1995) Guidelines for Clinical Exercise Testing Laboratories A Statement for Healthcare Professionals From the Committee on Exercise and Cardiac Rehabilitation, American Heart Association. *Circulation* 91:912–21.
19. Fletcher GF, Balady GJ, Amsterdam EA, Chaitman B, Eckel R, et al. (2001) Exercise standards for testing and training a statement for healthcare professionals from the American Heart Association. *Circulation* 104: 1694–1740.
20. Niebauer J (2011) Cardiac rehabilitation manual. *Springer.*
21. Adams J, Hubbard M, McCullough-Shock T, Simms K, et al. (2010) Myocardial work during endurance training and resistance training: a daily comparison, from workout session 1 through completion of cardiac rehabilitation. In Baylor University Medical Center.
22. Rishu S, Gupta V, Walia L, Mittal N (2013) Rate pressure product predicts cardiovascular risk in type 2 diabetics with cardiac autonomic neuropathy. *Nat J Physiol, Pharma and Pharmacology* 3: 43–47
23. Miyai N, Arita M, Miyashita K, Morioka I, Shiraishi T, Nishio I (2002) Blood pressure response to heart rate during exercise test and risk of future hypertension. *Hypertension* 39:761–6.

24. Gobel FL, Norstrom LA, Nelson RR, Jorgensen CR, Wang Y (1978) The rate-pressure product as an index of myocardial oxygen consumption during exercise in patients with angina pectoris. *Circulation* 3: 549–556.
25. Figueroa MA, Demeersman RE, Manning J (2012) The autonomic and rate pressure product responses of tai chi practitioners. *N Am J Med Sci* 4: 270–275. [crossref]
26. Sembulingam P, Sembulingam K, Ilango S, Sridevi G (2015) Rate pressure product as a determinant of physical fitness in normal young adults. *IOSR-JDMS* 1: 8-12.
27. Fornitano LD, Godoy MF (2006) Increased rate-pressure product as predictor for the absence of significant obstructive coronary artery disease in patients with positive exercise test. *Arquivos brasileiros de cardiologia* 86:138–44. 28
28. Coelho EM, Monteiro F, Da Conceição JM, Cruz J, Cunha D, et al. (1982) The rate pressure product: fact of fallacy? *Angiology* 33: 685–689. [crossref]
29. Keyhani D, Kargarfard M, Sarrafzadegan N, Sadeghi M (2013) Autonomic function change following a supervised exercise program in patients with congestive heart failure. *ARYA atherosclerosis* 9:150.
30. Al-Shamma YMH, Mhous AJ, Mousawi AM (2008) Heart Rate and Blood Pressure Changes After Exercise In Normal Adolescent In Relation To Body Mass Index. *Kufa J Med* 11: 437–447
31. Forjaz CL, Matsudaira Y, Rodrigues FB, Nunes N, Negrão CE (1998) Post-exercise changes in blood pressure, heart rate and rate pressure product at different exercise intensities in normotensive humans. *Braz J Med Biol Res* 31: 1247–1255. [crossref]

Citation:
Sherldine Tomlinson (2017) The Usefulness of the Rate Pressure Product (RPP) for Cardiac Rehabilitation Exercise Prescription. *Internal Med Res Open J* Volume 2(1): 1-x