Journal of Exercise Physiologyonline
(JEPonline)

Volume 12 Number 1 February 2009

Official Research Journal of the American Society of Exercise Physiologists (ASEP)

ISSN 1097-9751

Review

POST-EXERCISE HEART RATE RECOVERY: AN INDEX OF CARDIOVASCULAR FITNESS

UCHECHUKWU DIMKPA[1]

Physiology Department/ Faculty of Basic Medical Sciences/ Ebonyi State University, Abakiliki, Ebonyi State, Nigeria

ABSTRACT

Dimkpa U. Post-exercise heart rate recovery: an index of cardiovascular fitness. ***JEP****online* 2009;12(1):19-22. Cardiovascular fitness has traditionally been assessed by such variables as resting heart rate (HR), resting blood pressure (BP), cardiac output, stroke volume (SV), maximum oxygen consumption (VO_2max), endurance capacity, HDL cholesterol, body fat, glucose-stimulated insulin, and total cholesterol. Post-exercise heart rate recovery, though a readily obtainable parameter and a powerful and independent predictor of cardiovascular and all-cause mortality in healthy adults and in those with cardiovascular diseases, is often overlooked as an indicator of cardiovascular fitness. Heart rate recovery (HRR) is mainly thought to be due to parasympathetic reactivation and has been shown to be a remarkable complement to a medical and/or physical assessment of an individual. Clinical application of HRR after exercise has been widely studied. A delayed decline of heart rate has been associated with increased risk of cardiovascular mortality, autonomic dysfunction, diabetes, endothelial dysfunction, and metabolic syndrome. Similarly, HRR is associated with some cardiovascular fitness indices such as, maximum oxygen uptake, endurance capacity and central hemodynamic variables like resting heart rate, and resting blood pressure. In conclusion, the post-exercise HRR provides information that is complementary to the traditional cardiovascular fitness indices and should be added to the list of indicators of cardiovascular fitness.

Key Words: Maximum Oxygen Uptake, Endurance Capacity, Central Hemodynamics, HDL-Cholesterol, Body Mass Index.

TABLE OF CONTENTS

INTRODUCTION

Cardiovascular fitness represents the efficiency of the heart, lungs and vascular system in delivering oxygen to the working muscles so that prolonged physical work can be maintained (4). The indicators of cardiovascular fitness have traditionally included such variables as resting heart rate (HR), resting blood pressure (BP), cardiac output, stroke volume (SV), maximum oxygen consumption (VO_2max), endurance capacity, HDL cholesterol, body fat, glucose-stimulated insulin, and total cholesterol levels. Thus a cardiovascular fit individual is expected to have a decreased resting HR, lower BP, increased cardiac output, increased SV, increase in VO_2max, increased work endurance capacity, increased HDL cholesterol, decreased total cholesterol, reduced glucose-stimulated insulin, decreased body fat and generally, increased heart function with an ability to pump more blood (5,6).

Heart rate recovery can be defined as the rate at which the HR declines from either maximal or submaximal exercise to resting levels and has been identified as a powerful and independent predictor of cardiovascular and all-cause mortality in healthy adults (7, 8, 9), in those with CVD (10, 11) and diabetes (12). It has also been reported to be a remarkable complement to the medical and physical assessment of an individual (13). HR recovery to resting levels can take one hour after light or moderate exercise (14), four hours after long-duration aerobic exercise (15), and even up to 24 hours after intense or maximal exercise (16) and has been suggested to depend on the interaction among factors like exercise intensity (14, 17), cardiac autonomic modulation, and the level of physical fitness (18, 19, 20). Investigations aiming to quantify HRR have calculated time constants by fitting HR decay data to several mathematical models (21, 22, 23). Alternatively, most investigators have simply measured the change in HR from peak exercise to 1 or 2 minute of recovery or considered the slope of the decline (7, 10, 21). At the end of the exercise a decrease of 15-20 beats per minute (bpm) in the first minute of recovery has been shown to be typical for a healthy person (9, 24). A first minute reduction of post-exercise HR less than 12 bpm if recovery is active (7) or 18 bpm if recovery is passive in the supine position (10) after a maximal exercise test, represents an unfavorable prognosis for relative risk of cardiovascular mortality in asymptomatic individuals and cardiopaths (7, 9, 10).

Over the last several years, clinical evaluation of HRR as a prognostic tool for diagnosing CVD has been the subject of interest, with little attention given to its physiological importance. Despite several facts emanating from several prognostic and few physiological investigations which suggest that HRR is a plausible index of cardiovascular health, and a remarkable complement to the medical and

physical assessment of an individual, it is often overlooked as an indicator of cardiovascular fitness. To our knowledge, limited information exists regarding the use of HRR as a cardiovascular fitness indicator. Available literatures on cardiovascular fitness have mainly included such variables as resting heart rate (HR), resting blood pressure (BP), cardiac output, stroke volume (SV), maximum oxygen consumption (VO_2max), endurance capacity, HDL cholesterol, body fat, glucose-stimulated insulin, and total cholesterol, as the major indicators of cardiovascular fitness. This review therefore focuses on the clinical, physiological and practical importance of HRR in relation to cardiovascular health and fitness and suggests the need for its inclusion as an important cardiovascular fitness indicator.

CLINICAL APPLICATIONS OF HEART RATE RECOVERY

Clinical investigations on HRR were prompted by a pharmacologic study by Imia et al (21) in which they computed HRR decay curves using beat by beat data and concluded that short and moderate term HRR curves are vagally mediated since HR decay 30 seconds and two minutes into recovery was prolonged with atropine and dual blockage. The HR decay for 2 minutes however, was more prolonged with dual blockade than with atropine alone indicating that later recovery also depends on sympathetic modulation. Later, other studies (25, 26, 27) have validated the observation by Imia et al that early HRR after dynamic exercise is mainly thought to be a function of vagal reactivation, with the sympathetic withdrawal becoming more important later in recovery. A faster HRR may therefore be a function of increase in vagal activity or reduction in sympathetic activity. Dixon et al and Du et al (28, 29) demonstrated this fact when they found that athletes, who had higher vagal activity and lower sympathetic activity, had faster HR recovery than non-athletes. Similarly, Imai et al (21) in a study involving 20 patients with chronic heart failure and 9 athletes found that vagally mediated HRR after exercise is accelerated in well trained athletes but blunted in patients with chronic heart failure. A delayed HRR is considered an abnormal HR response and a measure of autonomic dysfunction or imbalance (30) and may be a reflection of a reduction in vagal tone or an exaggerated sympathetic activation (21).

The hypothesis linking HRR to mortality arose from the work that associated the autonomic nervous system with sudden cardiac death (31). Decreased vagal activity has been associated with increased risk of death in wide spectrum of patients (31, 32). Similarly a predominance of sympathetic activity and a reduction in parasympathetic cardiac control has been found in patients with acute myocardial infarction (33). The hypothesis that HRR is an independent predictor of mortality has been tested and valdated in a number of studies in healthy adults (7, 8, 9, 34, 35) and in those with cardiovascular disease (10, 11). In these studies, a first minute reduction in post-exercise HR of less than 12 bpm if return to rest is active (7) or 18 bpm if recovery is passive in the supine position (10) represents an unfavorable prognosis for relative risk of cardiovascular mortality. Cole et al (7) studied 2428 adults, who were referred to exercise nuclear perfusion scans, and Nishime et al (9) studied 9454 asymptomatic patients without history of heart failure who were referred to symptom-limited treadmill exercise. These studies found that a drop in HR of 12 beats per minute or less at 1 minute after peak exercise was associated with a relative risk of death. Later in another study involving 5234 asymptomatic patients who underwent symptom-limited treadmill exercise, Cole et al (8) also found that abnormal HR recovery (<43 bpm at 2 min) predicted death even after adjusting for standard risk factors. Pierpont et al (22) on the other hand, demonstrated that first order decay is an inadequate model for HR recovery after maximal exercise but may be reasonable for sub-maximal levels.

Apart from being an independent predictor of mortality, HRR is also predictive of mortality after adjusting for several factors (8, 10, 36). Nonetheless these studies showed limitations in their inability to evaluate cardiovascular mortality since it is important that cardiovascular mortality in addition to all-

cause mortality must be evaluated as an outcome in any study of cardiac testing measures. Similarly, there were limited evaluation of symptomatic populations, and variations in recovery protocols and criterion for abnormality. It is also worthy of note that apart from the 'normal' reciprocal behaviour of the two autonomic nervous branches (e.g. increased parasympathetic and decrease sympathetic), another mechanism in the form of 'mutual' activation of the two ANS branches has also been demonstrated (37). Though not well investigated, this mechanism is said to oppose the reciprocal behaviour of the ANS branches in modulation of HRR.

In order to validate the use of HRR to evaluate both all-cause mortality and cardiovascular mortality Shetler et al (13) in their study demonstrated that HRR is more predictive of non-cadiovascular than cardiovascular mortality. Furthermore, few studies have tried to evaluate some symptomatic conditions. For example, Desai et al (38) in their study indicated that heart transplanted individuals, lacking both sympathetic and parasympathetic tone, have significantly slower HRR at the first minute of post-exercise when compared to apparently healthy individuals, thus demonstrating the effect of autonomic dysfunction on HRR. Post-exercise HR less than 10 bpm has also been reported in heart failure and heart transplanted individuals at the beginning of recovery stage which indicates autonomic dysfunction and related to high mortality risk (7, 9, 22). Another study (39) also evaluated patients after a recent myocardial infarction. Despite these studies, majority of previous studies have mainly focused on asymptomatics.

The study of endothelial function is a new measure that is broadening our understanding of CVD development. Inability of the endothelial cells to stimulate vasodilation properly, thus impairing the structural and functional integrity of the vasculature, is referred to as endothelial dysfunction (40). Endothelial function plays a key role in determining the clinical manifestations of established atherosclerotic lesions (41, 42); related to subclinical measures of CVD and prospectively associated with an increased risk for clinical CVD events (43) and can be observed in healthy people with risk factors for heart disease (42). Endothelial function is reported to be suppressed by increased sympathetic tone (41, 44) as opposed to decrease in parasympathetic drive (45). Similarly, studies on psychosocial stress and negative psychosocial traits have demonstrated associations between endothelium function with heightened sympathetic nervous system activity (46, 47). Another study has also suggested that similar psychosocial factors may also be linked with decreased parasympathetic nervous system (48). Heightened sympathetic nervous system activity and suppressed parasympathetic nervous system activity impair the ability of the ANS to regulate the cardiovascular system (49). Heart rate recovery has been reported to be an independent predictor of endothelial function (41), an important risk factor for cardiovascular disease (50), and delayed due to autonomic dysfunction or imbalance (41).

Other studies have also shown HRR to be a predictor of diabetes (12), and inversely associated with insulin resistance (51, 52), and other cardiovascular risk factors including, body mass index (BMI), abdominal obesity, HDL cholesterol in older men, (30, 53), triglycerides/high density lipoprotein ratio in healthy subjects (30, 54), and fasting plasma glucose in healthy and diabetic patients (55). This plethora of information no doubt underscores the clinical importance of HRR in the assessment of cardiovascular health in both asymptomatic and cardiopaths.

PHYSIOLOGICAL IMPLICATIONS OF HEART RATE RECOVERY

The physiological significance of HRR unlike the clinical application has not been fully explored by researchers. However relationships between HRR and some physiological indicators of cardiovascular fitness have been addressed by a number of literatures.

Maximum Oxygen Uptake (VO_2max) Correlates Heart Rate Recovery

Maximum oxygen consumption is the greatest amount of oxygen a person can take in and consume while performing dynamic exercise involving a large part of total muscle mass (56). It is considered the best measure of cardiovascular fitness and exercise capacity and represents the amount of oxygen transported and used in cellular metabolism (1). Previous studies (19, 29) have shown that HRR is related to VO2max. In these studies, faster HRR was observed in athletes with higher VO2max than non-athletes. Furthermore, some factors such as age, exercise habit, and cardiac vagal activity which affect VO2max are also associated with HRR. For example, age is inversely related to VO2max (1, 57), and HRR (19). Physical exercise has been shown to improve VO2max (1, 58-60), and trained athletes have been reported to present faster HRR than untrained individuals (19, 28, 29,). Goldsmith et al (61) studied 37 healthy volunteers using power spectral analysis after an incremental bicycle exercise, and found that those with higher vagal activity indicated higher VO2max. Tulppo et al (62) also in their study involving healthy males who underwent bicycle exercise test, demonstrated that poor physical fitness is associated with an impairment of cardiac vagal function during exercise. Similarly, HRR has also been shown to be mainly due to vagal activity (26, 63).

Endurance Capacity and Heart Rate Recovery

Endurance capacity is an individual's ability to perform exercise at both submaximal and maximal intensities as demonstrated either by the ability to exercise longer at a similar workload or by increasing the workload attained at a given heart rate (1). Increased endurance capacity has been shown to be one of the indices of cardiovascular fitness (4). Studies have also demonstrated that exercise endurance capacity is linearly related to HRR (64, 65). Similarly, HRR is accelerated in endurance trained athletes (66).

Heart Rate Recovery and Central Hemodynamics

Increased cardiac output, increased stroke volume, decrease in resting BP and resting HR are all evidences of cardiovascular fitness (4, 5, 6). After aerobic exercise training, it has been reported that stroke volume at rest increases due to increase in end diastolic volume; resting HR decreases due to greater venous return to the heart and increases in autonomic control; cardiac output increases primarily due to increase in stroke volume; blood flow increases through the cardiovascular system (67); and both resting blood pressure and blood pressure during exercise are reduced (68).

It is expected that with the reactivation of the parasympathetic activity and subsequent withdrawal of the sympathetic activity during recovery, HR, stroke volume, cardiac contractility will decrease resulting in decreased cardiac output. Similarly, systemic vascular resistance and BP are also expected to decline, skeletal blood flow may remain elevated above resting levels, while the skeletal muscle pumping which contributes to increases in venous return during exercise is stopped during inactive recovery. No previous study to our knowledge has reported an association of cardiac output, stroke volume, post-exercise BP decline, venous return or blood flow during recovery with HRR. A previous study (69) however reported that resting HR accounted for the changes observed in HRR after submaximal exercise, while resting systolic blood pressure and resting diastolic blood pressure are shown to be inversely correlated with HRR (70). Association of HRR with changes in some of the central hemodynamics such as resting HR and resting BP may be due to changes in autonomic nervous system modulation. Parasympathetic input from vagal tone seems to contribute largely to the maintenance of resting HR, thus a low resting HR is suggestive of higher parasympathetic activity or lower sympathetic activity (71). A decreased vagal activity at rest has been suggested as the mechanism that explains the increased risk associated with increased resting HR (72). Similarly, increased sympathetic tone and decreased parasympathetic tone at rest, hence elevated resting HR, has been found in inappropriate sinus tachycardia and postural orthostatic tachycardia syndrome,

and in chronic left ventricular dysfunction (71). Chronic activation of the sympathetic nervous system and/or limitation of the parasympathetic tone can increase the risk of cardiovascular events (31). Other studies have also suggested that well trained or physically well-fit (aerobically) individuals present with a lower resting HR, suggestive of higher parasympathetic activity (60, 73-75) and lower sympathetic activity (76). This increased parasympathetic tone which occurs with regular dynamic exercise has been demonstrated to decrease the risk of potentially lethal arrhythmias during myocardial ischemia (77). Heart rate recovery has also been demonstrated to be due to increased vagal activity (26, 63), with a delayed decrease in HRR reflecting a reduction in vagal tone. Both increased resting HR and delayed HRR have been identified to be powerful risk factors for cardiovascular and all-cause mortality (7, 9, 78, 79). Similarly, decrease in BP at rest is thought to be due to decreased sympathetic activity (68).

PRACTICAL APPLICATION OF HEART RATE RECOVERY

Heart rate recovery is a readily obtainable, relatively inexpensive and very simple diagnostic and prognostic tool that reflects the ANS and cardiac functions and which clinicians can employ in cardiac rehabilitation settings. It is known that abnormalities of the ANS are related with death risk but the way these abnormalities are measured are difficult and require sophisticated equipment and tests. As part of a regular routine exercise testing, HRR will provide the clinicians with prognosis for patients who are at risk of death and those who are at low risk. Similarly, it will provide to the exercise physiologists and physical fitness personnel, a guide for exercise prescription and help evaluate the physical fitness status of an individual. The practical application of HRR will therefore be viewed in two conditions; first in patients with normal HRR who are identified as being at a low risk and secondly in those with abnormal HRR and who are considered to be at high risk of mortality.

When an individual has a normal HRR, he is at a low risk and this will allow the clinician to manage his or her condition conservatively without sending them for further testing and projecting them to essentially risky procedures. On the other hand, an individual with abnormal HRR signifies increased risk of cardiac death related to autonomic imbalance. Studies have indicated hope of possible modifications of abnormal HRR with pharmacological therapy and exercise training.

Because of their effect on parasympathetic tone, the beneficial effects of ß-blockers have been shown in post-myocardial infarction (80), and in patients with coronary disease (81). Although other previous studies have indicated lack of effect of ß-blockers on early HRR (21) and in abnormal HRR noted in patients with congestive heart failure (82), but there remains considerable evidence of the beneficial effects of ß-blockers on HRR. Studies (83, 84) have also indicated supportive evidence that HRR can be improved or modified through exercise training. These studies however focused only on patients with abnormal HRR and also were not large enough to assess for the effects on mortality.

DIRECTION FOR FUTURE RESEARCH

Based on some of the issues raised in the foregoing paragraphs, this review therefore recommends the following as possible areas of future research:

1. Further studies employing cardiac testing specifically in predicting cardiovascular disease rather than other life-threatening disease states should be carried out, so that decisions regarding cardiovascular interventions can be made.
2. Similarly, recovery protocols and criterion for abnormality should be standardized, while more study should be done to elucidate which mechanism is most plausible for ANS modulation of HRR.

3. It is imperative to note that most of the previous prognostic studies on HRR focused mostly on the role of autonomic nervous system modulation of HRR in normal and abnormal states. We therefore recommend that since the autonomic nervous system function varies with age, level of physical fitness, and between genders, cut-off points separating normal from abnormal HRR should be determined according to gender, age, and physical fitness. This means that some adjustments are required for the differences in a subject's metabolic stress level at a workload for which normal or abnormal HRR is determined.
4. No previous study to our knowledge has reported an association of cardiac output, stroke volume, post-exercise BP, venous return or blood flow during recovery with HRR. It is therefore recommended that further studies should be done to establish whether changes in these variables are related to changes in HRR after exercise.

CONCLUSIONS

In summary, studies have identified heart rate recovery after exercise (submaximal or maximal) as a predictor of cardiovascular and all-cause mortality and its relationship to indices of cardiovascular fitness has also been demonstrated. Even though there is need for further studies with more symptomatic populations and for standardization of protocols and criteria for abnormality, available evidences significantly reveal the potential importance of post-exercise heart rate recovery as an index of cardiovascular health. The post-exercise heart rate recovery should therefore be included to the list of the cardiovascular fitness and diagnostic indicators.

Address for correspondence: Dimkpa U, MSc, Physiology Department, Faculty of Basic Medical Sciences, Ebonyi State University, Abakiliki. PMB 53, Abakiliki, Ebonyi State, Nigeria. E-mail: positivedoings@yahoo.com.

REFERENCES

1. Fletcher GF, Balady GJ, Amsterdam AE, Chaitman B, Robert E, Jerome F, et al. Exercise standards for testing and training. A statement for health care professionals from the American heart association. ***Circulation*** 2001; 104:1694-1740.

2. Blair SN, Kohl HW, Barlow CE, Paffenbarger RS, Gibbons LW, Marcera CA. Changes in physical fitness and all-cause mortality. A perspective study of healthy and unhealthy men. ***J Am Med Assoc*** 1995; 273:1093-8.

3. Centers for Disease Control. Coronary Heart disease attributable to sedentary life-style – selected states, 1998. ***J Am Med Assoc*** 1990; 264:1390-2.

4. The American Sports Medicine Institute. Cardiovascular fitness. Retrieved on 28th May, 2008 from www.asmi.org/sportsmed/performance/cardio_fitness.html .

5. BeFitLifestyle, San Diego. Training, nutrition, activities; cardio coaching. Retrieved on 28th May, 2008 from; www.befitlifestyle.com/cardiotraining.html.

6. Fitzone for Women, Health Franchise Club, USA; Personal training system; cardiovascular fitness. Retrieved on 28th May, 2008 from www.fitzones.com/members/Fitness/fitness.asp.

7. Cole CR, Blackstone EH, Pashkow FJ, Snader CE, Lauer MS. Heart rate recovery immediately after exercise as a predictor of mortality. ***N Eng J Med*** 1999; 341:1351-57.

8. Cole CR, Foody JM, Blackstone EH, Lauer MS. Heart rate recovery after submaximal exercise testing as a predictor of mortality in cardiovascularly healthy cohorts. ***Ann Intern Med*** 2000; 132:552-555.

9. Nishime EO, Cole CR, Blackstone EH, Pashkow FJ, Lauer MS. Heart rate recovery and treadmill exercise score as predictors of mortality in patients referred for exercise ECG. ***The J Am Med Assoc*** 2000; 284:1392-1398.

10. Watanabe J, Thamilarasan M, Blackstone EH, Thomas JD, Lauer MS. Heart rate recovery immediately after treadmill exercise and left ventricular systolic dysfunction as predictors of mortality; the case of stress echocardiography. ***Circulation*** 2001; 104: 1911-1916.

11. Pitsavos CH, Chrysohoou C, Panagiotakos DB, Kokkinos P, Skoumas J, Papaioannou I, et al. Exercise capacity and heart rate as predictor of coronary heart disease events in patients with heterozygous familial hypercholesterolemia. ***Atherosclerosis*** 2004; 173(2):347-352.

12. Cheng YJ, Laurer MS, Earnest CP, Church TS, Kampart JB, Gibbons LW, et al. Heart recovery following maximal exercise testing as a predictor of cardiovascular disease and all-cause mortality in men with diabetes. ***Diabetes Care*** 2003; 26:2052-2057.

13. Shetler S, Marcus R, Froelicher VF, Vora S, Kalisetti D, Prakash M, et al. Heart rate recovery: Validation and methodologic issues. ***J Am Coll Cardiol*** 2001; 38:1980-1987.

14. Terziotti P, Schena F, Gulli G, Cevese A. Post-exercise recovery of autonomic cardiovascular control: a study by spectrum and cross-spectrum analysis in humans. ***Eur J Appl Physiol*** 2001; 84:187-194.

15. Hautala A, Tulppo MP, Makikallio TH, Laukkanen R, Nissila S, Huikuri HV. Changes in cardiac autonomic regulation after prolonged maximal exercise. **Clin Physiol** 2001; 21:238-245.

16. Furlan R, Piazza S, Dell'Orto S, Gentile E, Cerutti S, Pagani M et al. Early and late effects of exercise and athletic training on neural mechanisms controlling heart rate. ***Cardivasc Res*** 1993; 27:482-488.

17. Baum K, Ebfeld D, Leyk D, Stegemann J. blood pressure and heart rate during rest-exercise and exercise-rest transitions. ***Eur J Appl Physiol*** 1992; 64:134-138.

18. Kluess HA, Wood RH, Welsch MA. Vagal modulation of the heart and central hemodynamics during handgrip exercise. ***Am J Physiol*** 2000; 279: H1648-H1652.

19. Darr KC, Bassett DR, Morgan BJ, Thomas DP. Effects of age and training status on heart rate recovery after peak exercise. ***Am J Physiol*** 1988; 254:H340-343.

20. Hatfield BD, Spalding TW, Santa Maria DL, Porges SW, Potts JT, Byrne EA, et al. Respiratory sinus arrhythmia during exercise in aerobically trained and untrained men. ***Med Sci Sports Exerc*** 1998; 30:206-214.

21. Imai K, Sato N, Hori M, Kusuoka H, Ozaki H, Yokoyama H. Vagally mediated heart rate recovery after exercise is accelerated in athletes but blunted in patient with chronic heart failure. ***J Am Coll Cardiol*** 1994; 24:1529-1535.

22. Pierpont GL, Stolpman DR, Gornick CC. Heart rate recovery as an index of parasympathetic activity. ***J Auton Nerv Syst*** 2000; 80:169-174.

23. Pavia L, Myers J, Cesare R. Recovery kinetics of oxygen uptake and heart rate in patients with coronary artery disease and heart failure. ***Chest*** 1999; 116:808-813.

24. Meszaros L. Researchers find heart rate worth a thousand words; simple test tells even healthy people their heart disease risk. WebMD Medical News. Retrieved from http://my.webmd.com/content/article/1728.61455.

25. Sears CE, Choate JK, Paterson DJ. Inhibition of nitric oxide synthase slows heart rate recovery from cholinergic activation. ***J Appl Physiol*** 1998; 84:1596-1603.

26. Arai Y, Saul JP, Albrecht P, Hartley LH, Lilly LS, Cohen RJ, et al. Modulation of cardiac autonomic activity during and immediately after exercise. ***J Appl Physiol*** 1998; 256: H132-H141.

27. Perini R, Orizo C, Comande A, Castellano M, Beschi M, Veicsteinas A. Plasma norepinepherine and heart rate dynamics during recovery from submaximal exercise in man. ***Eur J Appl Physiol*** 1989; 58:879-883.

28. Dixon EM, Kamath MV, McCartney N, Fallen EL. Neural regulation of heart rate variability in endurance athletes and sedentary controls. ***Cardiovasc Res*** 1992; 26:713-719.

29. Du N, Bai S, Oguri K, Kato Y, Matsumoto I, Kawase H, et al. Heart rate recovery after exercise and neural regulation of heart rate variability in 30-40 year old female marathon runners. ***J Sports Sci Med*** 2005; 4:9-17.

30. Brinkworth GD, Noakes M, Buckley JD, Clifton PM. Weight loss improves heart rate recovery in overweight and obese men with features of the metabolic syndrome. ***Am Heart J*** 2006; 152(4):693 el-693 e6.

31. Schwartz PJ, La Rovere MT, Vanoli E. Autonomic nervous system and sudden cardiac death. Experimental basis and clinical observations for post-myocardial infarction risk stratification. ***Circulation*** 1992; 85:177-191.

32. La Rovere MT, Pinna GD, Hohnloser SH, Marcus FI, Mortara A, Nohara R, et al. ATRAMI Investigators: Autonomic tone and reflexes after myocardial infarction. Baroreflex sensitivity and heart rate variability in the identification of patients at risk of life-threatening arrhythmias: Implications for clinical trials. ***Circulation*** 2001; 103: 2072-2077.

33. Rothschild M, Rothschild A, Pfeifer M. Temporary decrease in cardiac parasympathetic tone after acute myocardial infarction. ***Am J Cardiol*** 1988; 18:637-639.

34. Morshedi-Meibodi A, Larson MG, Levy D, O'Donnel CJ, Vasan R. Heart rate recovery after treadmill exercise testing and risk of cardiovascular disease events (The Framingham heart Study). ***Am J Cardiol*** 2002; 90:848-852.

35. Mora S, Redberg RF, Cui Y, Whiteman MK, Flaws JA, Sharrett R, et al. Ability of exercise testing to predict cardiovascular and all-cause death in asymptomatic women: a 20-year follow-up of the Lipid Research Clinics Prevalence Study. ***J Am Med Assoc*** *2003; 290:1600-1607.*

36. Diaz LA, Brunken RC, Blackstone EH, Snader CE, Lauer MS. Independent contribution of myocardial perfusion defects to exercise capacity and heart rate recovery for prediction of all-cause mortality in patients with known or suspected coronary heart disease. ***J Am Coll Cardiol*** 2001; 37:1558-1564.

37. Oida E, Moritani T, Yamori Yukio. Tone-entropy analysis on cardiac recovery after dynamic exercise. ***J Appl Physiol*** 1997; 82:1794-1801.

38. Desai MY, Pena-Almaguer E, Mannting F. Abnormal heart rate recovery after exercise as a reflection of abnormal chronotropic response. ***Am J Cardiol*** 2001; 87:1164-1169.

39. Nissinen SI, Makikallio TH, Seppanen T, Tapanainen JM, Mirja S, Tulppo MP, et al. Heart rate recovery after exercise as a predictor of mortality among survivors of acute myocardial infarction. ***Am J Cardiol*** 2003; 91:711-714.

40. Harris KF, Matthews KA. Interaction between autonomic nervous system activity and endothelial function: A model for the development of cardiovascular disease. American ***Psychosom Soc*** 2004; 66:153-164.

41. Huang P, Leu H, Chen J, Chen C, Huang C, Tuan T, et al. usefulness of attenuated heart rate recovery immediately after exercise to predict endothelial dysfunction in patients with suspected coronary artery disease. ***Am J Cardiol*** 2004; 93(1):10-13.

42. Celermajer DS, Sorensen KE, Gooch VM, Spiegelhalter DJ, Miller OI, Sullivan ID et al. Non-invasive detection of endothelial dysfunction in children and adults at risk of atherosclerosis. **Lancet** 1992; 340:1111-1115.

43. Schachinger V, Britten MB, Zeiher AM. Prognostic impact of coronary vasodilator dysfunction on adverse long term outcome of coronary heart disease. ***Circulation*** 2000; 101:1899-1906.

44. Oyake Y, Ohtsuka S, Yamazaki A, Yamaguchi I. Increased sympathetic nerve activity relates to vascular endothelial dysfunction in patients with essential hypertension. Circ J 2003; 67:229

45. Gulli G, Cemin R, Pancera P, Menegatti G, Vassanelli C, Cevese A. Evidence of parasympathetic impairment in some patients with cardiac syndrome X. Cardiovascular Research 2001; 52:208-216.

46. Burns JW, Friedman R, Katkin ES. Anger expression, hostility, anxiety, and patterns of cardiac reactivity to stress. ***Behav Med*** 1992; 18:71-78.

47. Cacioppo JT, Malarkey WB, Kiecolt-Glaser JK, Uchino BN, Sgoutas-Emch SA, Sheridan JF et al. heterogeinity in neuroendocrine and immune responses to brief psychological stressors as a function of autonomic cardiac activation. ***Psychosom Med*** 1995; 57:154-164.

48. Lucini D, Norbiato G, Clerici M, Pagani M. Hemodynamic and autonomic adjustments to real life stress conditions in humans. ***Hypertension*** 2002; 39:184-188.

49. Decker JM, Crow RS, Folsom AR, Hannan PJ, Liao D, Swenne CA, et al. Low heart rate variability in a 2-minute rhythm strip predicts risk of coronary heart disease and mortality from several causes: the ARIC Study. Atherosclerosis Risk In Communities. ***Circulation*** 2000; 102: 1239-1244.

50. Grundy SM, Benjamin IJ, Burke GL, Chait A, Eckel RH, Howard BV, et al. Diabetes and cardiovascular disease. A statement for healthcare professionals from the American Heart Association. ***Circulation*** 1999; 100:1134-1146.

51. McLaughlin T, Abbasi F, Cheal K, Chu J, Lamendola C, Raeven G. Use of metabolic markers to identify overweight individuals who are insulin resistant. ***Ann Intern Med*** 2003; 139:10:802-809.

52. Reaven G. Metabolic syndrome: pathophysiology and implication for management of cardiovascular disease. ***Circulation*** 2002; 106:286-288.

53. Lind L, Andren B. heart rate recovery after exercise is related to the insulin resistance syndrome and heart rate variability in elderly men. ***Am Heart J*** 2002; 144(4):666-672.

54. Shishehbor MH, Hoogwerf BJ, Lauer MS. Association of triglycerides-to-HDL cholesterol ratio with heart rate recovery. ***Diabetes Care*** 2004; 27:936-941.

55. Panzer C, Lauer MS, Brieke A, Blackstone E, Hoogwerf B. Association of fasting plasma glucose with heart rate recovery in healthy adults: a population based study. ***Diabetes*** 2002; 51: 803-807.

56. Cohn JN, ed. Quantitative exercise testing for the cardiac patient; the value of monitoring gas exchange: introduction. ***Circulation*** 1987; 76(suppl VI); I-1-VI-2.

57. Fleg JL, Morell CH, Bos AG, Brant LJ, Talbot LA, Wright JG, et al. Accelerated longitudinal decline of aerobic capacity in healthy older adults. ***Circulation*** 2005; 112:674-682.

58. McGuirre DK, Levine BD, Williamson JW, Snell PG, Blomqvist CG, Saltin B, et al. A 30-year follow-up of the Dallas Bed Rest and Training Study. The effect of age on the cardiovascular response to exercise. ***Circulation*** 2001; 104:1358-1366.

59. Seals D, Esler M. Human ageing and the sympathoadrenal system. ***J Physiol*** 2000; 528:3:407-417.

60. Blomqvist CG, Saltin B. Cardiovascular adaptations to physical training. ***Ann Rev Physiol*** 1983; 45:169-189.

61. Goldsmith RL, Bigger JT, Bloomfield DM, Steinman RC. Physical fitness as a determinant of vagal modulation. ***Med Sci Sports Exerc*** 1997; 29(6):812-817.

62. Tulppo MP, Makkallio TH, Seppanen T, Laukkanen RT, Huikuri HV. Vagal modulation of heart rate during exercise; effects of age and physical fitness. ***Am J Physiol*** 1998; 274(2):H424-429.

63. Javorka M, Zila I, Ballharek T, Javorka K. heart rate recovery after exercise; relations to heart rate variability and complexity. ***Braz J Med Biol Res*** 2002; 35:991-1000.

64. Ota Y. Evaluation of the whole body endurance capacity by treadmill all-out run. ***Bull Fukuoka Univ Edu*** 2002; 51:71-77.

65. Singh T, Rhodes J, Gauvreau K. Determination of heart rate recovery following exercise in children. ***Med Sci Sports Exerc*** 2008; 40(4):601-605.

66. Otsuki T, Maeda S, Iemitsu M, Saito Y, Tanimura Y, Sugawara J, et al. Post exercise heart rate recovery accelerates in strength trained athletes. ***Med Sci Sports Exerc*** 2007; 39(2):365-370.

67. 123HelpMe.com Free Essays. The effect of aerobic exercise on the cardiorespiratory system. Retrieved on 28th May, 2008 from www.123helpme.com/view.asp?id=148639.

68. Staheim-Smith A, Fitch GK. Exercise, blood flow and blood pressure. In: **Understanding human anatomy and physiology.** West Publishing Company, Minneapolis 1993. pp.634.

69. Mahon AD, Anderson CS, Hipp MJ, Hunt KA. Heart rate recovery from submaximal exercise in boys and girls. ***Med Sci Sports Exerc*** 2003; 35(12); 2093-2097.

70. Jidon S, Yoon-Ho C, Jeong BP. Metabolic syndrome is associated with delayed heart rate recovery after exercise. ***J Korean Med Sci*** 2006; 21:621-626.

71. Freeman JV, Dewey FE, Hadley DM, Myers J, Froelicher VF. Autonomic nervous system interaction with the cardiovascular system during exercise. ***Progr Cardiovasc Dis***. 2006; 48:342-362.

72. Jouven X, Empana J, Schwartz PJ, Desnos M, Courbon D, Ducimetiere P. Heart rate profile during exercise as a predictor of sudden death. ***N Engl J Med*** 2005; 352:1951-1958.

73. Aubert AE, Beckers F, Ramaekers D. Short term heart rate variability in young athletes. ***J Cardiol*** 2001; 37:S85-88.

74. Spalding TW, Jeffers LS, Porges SW, Hatfield BD. Vagal and cardiac reactivity to psychological stressors in trained and untrained men. ***Med Sci Sports Exerc*** 2000; 32:581-591.

75. Shin K, Minamitani H, Onishi S, Yamaaki H, Lee M. Autonomic differences between athletes and non-athletes: spectral analysis approach. ***Med Sci Sports*** **Exerc** 1995; 18:583-586.

76. Chacon-Mikahil MPT, Forti VAM, Catai AM, Szrajer JS, Golfetti R, Martins LEB, et al. Cardiorespiratory adaptations induced by aerobic training in middle-aged men: the importance of a decrease in sympathetic stimulation for the contribution of dynamic exercise tachycardia. ***Brazillian J Med Biol Res*** 1998; 31:705-712.

77. Hull SSJ, Vanoli E, Adamson PB, Verrier RL, Foreman RD, Schwartz PJ. Exercise training confers anticipatory protection from sudden death during acute myocardial ischemia. ***Circulation*** 1994; 89:548-552.

78. Goldberg RJ, Larson M, Levy D. Factors associated with survival to 75 years of age in middle-aged men and women: the Framingham Study. ***Arch Intern Med*** 1996; 156:505-509.

79. Palatini P. Need for a revision of the normal limits of resting heart rate. **Hypertension** 1999; 33:622-625.

80. Lampert R, Ickovics JR, Viscoli CJ, Horwitz RI, Lee FA. Effects of propranolol on recovery of heart rate variability following acute myocardial infarction and relation to outcome in the Beta Blocker Heart Trial. ***Am J Cardiol*** 2003; 91:137-142.

81. Pavia L, Myers J, Rusconi C. Effect of beta blockade on heart rate and VO2 kinetics during recovery in patients with coronary disease. ***Heart Drug*** 2002; 2:69-74.

82. Racine N, Blachet M, Ducharme A, Marquis J, Boucher J, Juneau M, et al. Decreased heart rate recovery after exercise in patients with congestive heart failure: effect of ß-blocker therapy. ***J Card Fail*** 2003; 9:296-302.

83. Tiukinhoy S, Beohar N, Hsie M. improvement in heart rate recovery after cardiac rehabilitation. ***J Cardiopulm Rehabil*** 2003; 23:84-87.

84. Kligfield P, Cormick A, Chai A, Jacobson A, Feuerstadt P, Hao SC. Effect of age and gender on heart rate recovery after submaximal exercise during cardiac rehabilitation in patients with angina pectoris, recent acute myocardial infarction or coronary bypass surgery. ***Am J Cardiol*** 2003; 92:600-603.