Journal of Exercise Physiology**online**

**December 2012**

**Volume 15 Number 6**



**JEPonline**

**Editor-in-Chief**

Tommy Boone, PhD, MBA

**Review Board**

Todd Astorino, PhD

Julien Baker, PhD

Steve Brock, PhD

Lance Dalleck, PhD

Eric Goulet, PhD

Robert Gotshall, PhD

Alexander Hutchison, PhD

M. Knight-Maloney, PhD

Len Kravitz, PhD

James Laskin, PhD

Yit Aun Lim, PhD

Lonnie Lowery, PhD

Derek Marks, PhD

Cristine Mermier, PhD

Robert Robergs, PhD

Chantal Vella, PhD

Dale Wagner, PhD

Frank Wyatt, PhD

Ben Zhou, PhD

Official Research Journal of the American Society of Exercise Physiologists

ISSN 1097-9751

Official Research Journal of the American Society of Exercise Physiologists

ISSN 1097-9751

### **Post-Exercise Cardiac Full Vagal Reactivation: Initial Proposal and Influence of Physical Training**

Tiago Peçanha de Oliveira, Jorge Roberto Perrout de Lima

1Laboratory of Motor Assessment, Faculty of Physical Education and Sports, Juiz de Fora Federal University, Minas Gerais, Brazil

##### ABSTRACT

**Oliveira TP, Lima JRP**. Post-Exercise Cardiac Full Vagal Reactivation: Initial Proposal and Influence of Physical Training. **JEPonline**2012;15(6):103-111. The optimal autonomic recovery after physical exercise has an important impact on the health status and in sports training. There is no easy-to-apply method to assess the body's ability to fully recover the autonomic balance after the exercise stress. The purpose of this study was to make an initial proposal of a method to evaluate the time needed for restoration of cardiac vagal modulation after exercise and investigate the influence of physical training on this time. Cardiac vagal modulation was evaluated through the analysis of RMSSD at rest and during 60 min after a submaximal exercise (50% VO2 max) on a cycle ergometer in healthy young males (n = 18). Then, the time required individually for the post-exercise RMSSD to reach its resting values (full vagal reactivation time, FVR) and the influence of the level of physical training on this time. The t-test for independent measurements was performed to compare the FVR time between the low and the high physical training groups (P<0.05). On average, 17 min (± 14 min) was required for FVR after submaximal exercise. Individuals with higher levels of physical training had faster FVR (9.5 ± 7.6 min) than their counterparts with lower levels of physical training (24.8 ± 17.1 min). The method proposed by this study is efficient and promising. Also, the positive effects of physical training on post-exercise FVR support the practice of regular exercise as an efficient strategy to improve the cardiac autonomic recovery after exercise.

**Key Words**: Autonomic Nervous System, Parasympathetic Nervous System, Physical Exercise, Heart Rate Control, Heart Rate

**INTRODUCTION**

Physical exercise produces an acute body stress, with substantial effects on cardiac autonomic control (23). The increase in heart rate (HR) that occurs during physical exercise is the outcome of a coordinated neural adjustment system that produces reflexly a decrease in cardiac vagal activity and an increase in cardiac sympathetic activity (17). This autonomic imbalance produced by exercise creates an environment conducive to the emergence of cardiovascular arrhythmias that often persists for some time during post-exercise recovery (1,19,23).

Given the cardioprotective role attributed to cardiac vagal modulation (4,18), it is believed that the individual remains under the risks of physical exercise while cardiac vagal modulation is returning to its resting condition. Thus, one can say that the shorter the time required to completing post-exercise vagal reactivation, the better the individual's clinical condition. It is reasonable then that strategies promoting the acceleration of this response are welcome.

Physical exercise is considered a non-drug therapy with a positive impact on the cardiovascular system and cardiac autonomic modulation (21,26). Studies have shown that exercise training also enhances the cardiac vagal reactivation (5,6). However, most of these studies evaluated only the first moments of cardiac autonomic recovery, disregarding the time of post-exercise full vagal reactivation (FVR), whose clinical significance was defined previously. This is probably due to the lack of methods for FVR assessment.

The purpose of this study was to make an initial proposal of a method to study the time required to achieve the full post-exercise cardiac full vagal reactivation, and also to verify the role of physical training at this time.

###### METHODS

**Subjects**

Eighteen healthy males participated in the study (age = 22.1 ± 2 yrs; BMI = 24.1 ± 2 kg·m-2). All subjects were instructed not to drink alcohol or caffeine-containing beverages, and also not to do any physical exercise in the 24 hrs preceding the tests. The use of any drug with a cardiovascular action was an exclusion criterion. The subjects were fully informed of the procedures to be undertaken, and provided written informed consent about their participation in the study. The procedures were adopted with respect to the international norms of experimentation on human subjects (Helsinki Declaration, 1975) and were approved by the ethics committee of the institution.

**Experimental Protocol**

The experiment was conducted in two sessions, on non-consecutive days. In order to avoid influence of circadian rhythm in the measured variables, all tests were performed in the morning (8-12 am).

***Day 1:*** On the first day, height and body mass, level of physical activity, and maximal aerobic capacity of the subjects were assessed. The level of physical activity was assessed through the Baecke Questionnaire of Habitual Physical Activity – BQHPA (3). Only the systematic practice of physical exercises/sports was considered, that is, the physical training through calculation of the gross score BQHPA question 9, regarding the practice of sports/physical exercises. In order to assess the subjects’ maximal aerobic capacity, they performed an incremental maximal exercise test, on an electromagnetically-braked cycle ergometer, with initial power of 100 W, with 25 W increases each minute until maximum voluntary exhaustion was reached. Analysis of the expired gases was continuously performed with the VO2000 metabolic analyzer (MedGraphics, USA), which was calibrated manually before each test. From the exercise test, VO2 max was identified. The exercise test was considered maximal under two of the following three conditions: (a) plateau in the VO2 curve in spite of increased load; (b) respiratory ratio >1.1; and (c) at least 90% of the maximal age-predicted HR.

***Day 2:*** On the second day, the subjects underwent resting RR interval (RRi) recording, performed the submaximal exercise and then, realized the recording of post-exercise recovery RRi. The Polar S810i monitor (sampling frequency: 1000 Hz), was used to measure the RRi at rest and at post-exercise recovery (25). Initially, the subjects remained resting in the supine position for 15 min. Then, they performed the submaximal exercise on the cycle ergometer (50% VO2 max, 30 min) at constant cadence of 60 revolutions per minute (RPM). After exercise cessation, the subjects quickly proceeded to lie down on a stretcher where they rested and remained during the 60-min recovery period.

**Procedures**

***Data Transmission, Signal Processing and HRV Analysis***

After RRi recording by the monitor, the data were transmitted to a computer, through an interface with an infrared device and the Polar Precision Performance software. The data were subsequently sent to Kubios HRV (v. 2.0) to HRV analysis. The 5-min mean of root mean square of successive differences in RRi (RMSSD index) was used to analyze HRV at rest and during whole recovery (Figure 1). Although studies on HRV generally control for the respiratory rate, we chose not to do so in order to not interfere with the return of the HRV to baseline levels.

***Identification of Full Vagal Reactivation***

For FVR identification, the last 5 min of resting iRR recording were divided into 10 windows of 30 sec each. The RMSSD index was then calculated for each window (RMSSD30s). The mean, standard deviation, and coefficient of variation (%CV) of the RMSSD30s values of the formed windows were then calculated. A mean %CV of 19% was observed among the RMSSD30s at rest. From this variation, a tolerance range was determined, which, when reached by recovery RMSSD values, would determine the FVR (Figure 1).



 Figure 1

**Statistical Analyses**

The data were presented as mean ± standard deviation. Because the Shapiro-Wilk’s test showed that the RMSSD index had a skewed distribution, the logarithmic transformation of this index (InRMSSD) was performed. The median was used for group definition according to the physical training. The low physical training group (LPTG) was the one whose physical training score was lower than the calculated median and the high physical training group (HPTG) was the one whose physical training score was higher than the calculated median. In order to compare FVR between the groups, student’s t-test for independent measurements was performed (α = 0.05).

RESULTS

Table 1 shows the characterization of the sample. The groups did not significantly differ as for age, weight, or VO2 max.

**Table 1. Sample Characterization.**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Group** | **N** | **Age** **(yrs)** | **Weight** **(kg)** | **HR max**(beats·min-1) | **VO2 max** **(mL·kg-1·min-1)** | **PT (score)** |
| LPTG | 9 | 22.1 ± 2 |  74.4 ± 10.7 | 188.1 ± 9.2 | 46.3 ± 3.6 | 2.1 ± 1 |
| HPTG | 9 | 22.0 ± 3 | 76.4 ± 5.1 | 187.0 ± 6.7 | 47.4 ± 2.8 |  6.5 ± 2\* |

LPTG = low physical training group; HPTG = high physical training group; HR max = maximum heart rate; PT = physical training score (question 9 - Baecke’s questionnaire); (\*) P<0.05 (‘between groups’ comparison).

**Exercise**

HR values during exercise were 143.0 ± 8.1 and 137.8 ± 10.9 beats·min-1 for the LPTG and HPTG, respectively. There was no statistically significant difference between these values. Exercise HR corresponded to a mean relative intensity of 75% of the HR max.

**Influence of Physical Training on the Post-Exercise Full Vagal Reactivation**

Figure 2 shows the behavior of RMSSD at rest and during post-exercise recovery period in LPTG and HPTG groups. The FVR-method proposed by this study proved to be easy-to-apply and could be performed in all subjects (Figure 3). Figure 4 shows the mean FVR values (± standard deviation) of all individuals and in each formed group. Both groups reached the FVR in <1 hr (17.1 ± 14.8 min). Yet, whereas in the LPTG, the FVR occurred after 25 min (±17.1), only 9 min (±7.6) was necessary for FVR to be reached in the HPTG.

Figure 2



Figure 3



Figure 4

DISCUSSION

This study made an initial proposal of a method to identify the required time for the post-exercise FVR and also tested the effect of physical training on this time. In this sense, the FRV-method proved to be easy-to-apply and sensitive to the effects of physical training.

Several methods have been proposed to study post-exercise full vagal reactivation. The most widely known are: (a) HRR, the difference between HR at the peak of exercise and HR after 60 sec of recovery (2,9,20,29); (b) HRRt, calculated by the exponential-adjusted HR falling time constant (27); and (c) T30, which represents the negative reciprocal of the inclination of the regression curve of the natural logarithms of the HR corresponding to each R-R interval from the 10th to the 40th sec of recovery (16). In common between these indices, the fact they consider only the first minutes of recovery. Despite the potential clinical importance of the FVR study, there is no easy-to-apply index that mirrors the ability of the human subject to fully recover from exercise-induced cardiovascular autonomic stress.

Post-exercise FVR involves the restoration of many regulatory mechanisms. Among these, the following should be highlighted: (a) cessation of inhibitory vagal stimuli from the motor cortex (central command) (8); (b) cessation of mechanoreceptor action (7); and (c) the gradual reduction of the stimulation of skeletal muscle metaboreceptors (10). Besides these factors, it should be noted also the normalization of blood pH concentrations and of the partial pressure of arterial O2 and CO2, which deactivates the aortic and carotid chemoreceptors (14) are involved in post-exercise FVR. Another factor that should be highlighted is the restoration of body temperature, which possibly also has relation with the cardiac FVR (13).

This study found positive influence of physical training on FVR. This finding is consistent with previous studies (5,6), which indicated that physical training influenced cardiac autonomic recovery. We observed that after moderate-intensity exercise, both groups had FVR in less than 1 hr. However, whereas the HPTG was fully recovered 9 min after exercise cessation, it took the LPTG 24 min to achieve FVR. Besides being statistically significant, these differences also have high magnitude, as the division of the difference of mean groups results by the pooled-standard deviations results in an effect size (Cohen d) of 1.3 (i.e., large effect). Although the methods of the present study do not allow for greater understanding of the causes underlying this response, it is speculated that the metabolites removal capacity (known to be enhanced by physical training), may have contributed to the faster FVR in the trained (22).

Other factors that may also be enhanced in trained subjects and may have influenced the results are increased baroreflex sensitivity (30), lower chemoreflex activation (15), and better post-exercise body temperature cooling system (11). Future studies should investigate the behavior of these mechanisms during the post-exercise period, and the relation of each to the FVR responses. However, regardless of the mechanisms underlying the findings, the results of this study endorse FVR-method as promising, since even in a homogenous group of young and healthy subjects, whose only difference was the physical training level, the method was able to distinguish such groups.

**Full Vagal Reactivation Method Proposition: Next Steps**

For the FVR-method validation, it is necessary that a pharmacological blockade study confirms the theoretical assumptions of this study and, therefore, the ability of the method to assess the post-exercise FVR. It is also necessary to investigate the reproducibility of the method and its usefulness in studies involving clinical populations so that finally a possible prognostic value can also be investigated. Furthermore, factors such as exercise intensity, duration and type; co-morbidities and cardiovascular impairment; age; lifestyle; drug use; fatigue and overtraining levels; among others, may influence FVR, being amenable to investigation.

**Study Limitations**

The fact is there are no analyses of the HRV in the frequency domain of the post-exercise autonomic recovery could be considered a limitation of this study. HRV analysis in the frequency domain allows for greater understanding of the individual roles of the loops of the autonomic nervous system in post-exercise recovery (28). However, the use of these indices assumes the steady condition of the data, typical of analyses made at rest. The use of linear methods for obtaining HRV values in situations of HR variation, such as exercise and recovery, could lead to inconsistent results that hamper the reading of the results. Furthermore, a study by Ng et al. (24) suggests that analyses in the time domain are preferable to those in the frequency domain during recovery. According to this study, there is a high correlation between the two analyses, although the former is less prone to the mathematical anomalies. The choice of RMSSD is justified because this is a validated index of post-exercise full vagal reactivation (12).

### **CONCLUSIONS**

The FVR method proposed by this study is efficient, easy-to-apply, and presents a potential practical application, since it was sensitive to the effects of physical training. The positive effects of physical training on post-exercise FVR endorse the practice of regular exercise as an efficient strategy to improve the cardiac autonomic recovery after exercise.

Address for correspondence: Oliveira, TP, Faculty of Physical Education and Sports, Laboratory of

Motor Assessment, Campus Universitário, Martelos, Juiz de Fora – MG, Brazil, 36036-900. Phone:

(32) 2102-3287; Email: tiago\_faefid@yahoo.com.br

**REFERENCES**

1. Albert CM, Mittleman MA, Chae CU, Lee IM, Hennekens CH, Manson JE. Triggering of sudden death from cardiac causes by vigorous exertion. ***N Engl J Med.*** 2000;343:1355-1361.
2. Antelmi I, Chuang EY, Grupi CJ, Latorre Mdo R, Mansur AJ. Heart rate recovery after treadmill electrocardiographic exercise stress test and 24-hour heart rate variability in healthy individuals. ***Arq Bras Cardiol.*** 2008;90:380-385.
3. Baecke JA, Burema J, Frijters JE. A short questionnaire for the measurement of habitual physical activity in epidemiological studies. ***Am J Clin Nutr.*** 1982;36:936-942.
4. Buch AN, Coote JH, Townend JN. Mortality, cardiac vagal control and physical training--what's the link? ***Exp Physiol.*** 2002;87:423-435.
5. Buchheit M, Gindre C. Cardiac parasympathetic regulation: respective associations with cardiorespiratory fitness and training load. ***Am J Physiol Heart Circ Physiol.*** 2006; 291:H451-H458.
6. Buchheit M, Millet GP, Parisy A, Pourchez S, Laursen PB, Ahmaidi S. Supramaximal training and post-exercise parasympathetic reactivation in adolescents. ***Med Sci Sports Exerc.*** 2008; 40:362-371.
7. Bull RK, Davies CT, Lind AR, White MJ. The human pressor response during and following voluntary and evoked isometric contraction with occluded local blood supply. ***J Physiol.*** 1989;411:63-70.
8. Carter R, Watenpaugh DE, Wasmund WL, Wasmund SL, Smith ML. Muscle pump and central command during recovery from exercise in humans. ***J Appl Physiol.*** 1999; 87:1463-1469.
9. Cole CR, Blackstone EH, Pashkow FJ, Snader CE, Lauer MS. Heart-rate recovery immediately after exercise as a predictor of mortality. ***N Engl J Med.*** 1999;341:1351-1357.
10. Coote JH. Recovery of heart rate following intense dynamic exercise. ***Exp Physiol.*** 2010; 95:431-440.
11. Davies CT, Barnes C, Sargeant AJ. Body temperature in exercise. Effects of acclimatization to heat and habituation to work. ***Int Z Angew Physiol.*** 1971;30:10-19.
12. Goldberger JJ, Le FK, Lahiri M, Kannankeril PJ, Ng J, Kadish AH. Assessment of parasympathetic reactivation after exercise. ***Am J Physiol Heart Circ Physiol.*** 2006; 290:H2446-4452.
13. Gonzalez-Alonso J. Human thermoregulation and the cardiovascular system (Thermoregulation). ***Exp Physiol.*** 2012 Jan 6.
14. Gujic M, Laude D, Houssiere A, Beloka S, Argacha JF, Adamopoulos D, et al. Differential effects of metaboreceptor and chemoreceptor activation on sympathetic and cardiac baroreflex control following exercise in hypoxia in human. ***J Physiol.*** 2007;585:165-174.
15. Harthmann AD, De Angelis K, Costa LP, Senador D, Schaan BD, Krieger EM, et al. Exercise training improves arterial baro- and chemoreflex in control and diabetic rats. ***Auton Neurosci.*** 2007;133:115-1120.
16. Imai K, Sato H, Hori M, Kusuoka H, Ozaki H, Yokoyama H, et al. Vagally mediated heart rate recovery after exercise is accelerated in athletes but blunted in patients with chronic heart failure. ***J Am Coll Cardiol.*** 1994;24:1529-1535.
17. Kaufman MP, Hayes SG. The exercise pressor reflex. ***Clin Auton Res.*** 2002;12:429-439.
18. Kent KM, Smith ER, Redwood DR, Epstein SE. Electrical stability of acutely ischemic myocardium. Influences of heart rate and vagal stimulation. ***Circulation.*** 1973;47:291-298.
19. Maron BJ. The paradox of exercise. ***N Engl J Med.*** 2000;343:1409-1411.
20. Mattioli GM, Araujo CG. Association between initial and final transient heart rate responses in exercise testing. ***Arq Bras Cardiol.*** 2009;93:141-146.
21. Melanson EL. Resting heart rate variability in men varying in habitual physical activity. ***Med Sci Sports Exerc.*** 2000;32:1894-1901.
22. Messonnier L, Freund H, Denis C, Féasson L, Lacour JR. Effects of Training on Lactate Kinetics Parameters and their Influence on Short High-Intensity Exercise Performance. ***Int J Sports Med.*** 2006;27:60-66.
23. Mittleman MA, Siscovick DS. Physical exertion as a trigger of myocardial infarction and sudden cardiac death. ***Cardiol Clin.*** 1996;14:263-270.
24. Ng J, Sundaram S, Kadish AH, Goldberger JJ. Autonomic effects on the spectral analysis of heart rate variability after exercise. ***Am J Physiol Heart Circ Physiol.*** 2009;297:H1421-4218.
25. Nunan D, Donovan G, Jakovljevic DG, Hodges LD, Sandercock GR, Brodie DA. Validity and reliability of short-term heart-rate variability from the Polar S810. ***Med Sci Sports Exerc*.** 2009; 41:243-250.
26. Paffenbarger RS, Jr. Contributions of epidemiology to exercise science and cardiovascular health. ***Med Sci Sports Exerc.*** 1988;20:426-438.
27. Perini R, Orizio C, Comande A, Castellano M, Beschi M, Veicsteinas A. Plasma norepinephrine and heart rate dynamics during recovery from submaximal exercise in man. ***Eur J Appl Physiol Occup Physiol.*** 1989;58:879-883.
28. Task-Force. Heart rate variability. Standards of measurement, physiological interpretation, and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. ***Eur Heart J.*** 1996;17:354-381.
29. Trevizani GA, Benchimol-Barbosa PR, Nadal J. Effects of age and aerobic fitness on heart rate recovery in adult men. ***Arq Bras Cardiol.*** 2012 July 26.
30. Ueno LM, Moritani T. Effects of long-term exercise training on cardiac autonomic nervous activities and baroreflex sensitivity. ***Eur J Appl Physiol.*** 2003;89:109-114.

# Disclaimer

The opinions expressed in **JEPonline** are those of the authors and are not attributable to **JEPonline**, the editorial staff or the ASEP organization.