

# Recovery Heart Rate Response in Sedentary and Physically Active Young Healthy Adults of Bijapur, Karnataka, India

Haroonrashid M. Hattiwale, Shaheenkousar H. Hattiwale, Salim A. Dhundasi, Kusal K. Das\*

Department of Physiology, Al Ameen Medical College, Bijapur, Karnataka, 586108, India

**Abstract** The purpose of this study was to assess the effects of sedentary and physically active lifestyle on percent recovery heart rate in a population-based sample of healthy subjects of Bijapur, Karnataka, India. Twenty nine healthy adult males (age  $34.30 \pm 3.86$  years) with sedentary life style and equal number of age and sex matched young healthy adults with regular physical activity were selected for this study. Subjects performed submaximal exercise test by using cycle ergometer. Heart rate and blood pressure were measured before exercise (pre-exercise), immediately after exercise (peak-exercise) and 2 minutes after cessation of exercise (post-exercise) and percent recovery of heart rate and percent recovery blood pressure were calculated. Percent recovery heart rate and percent decrease of blood pressure were significantly faster in physically active subjects than sedentary individuals which may be attributed to augmented baroreflex sensitivity in physically active male subjects or greater capacity of reflex cardiovascular modulation after exercise. Results indicate that physical activity in lifestyle has a considerable impact on cardiovascular fitness.

**Keywords** Heart Rate Recovery, Sedentary, Physically Active Life Style, Blood Pressure Recovery

## 1. Introduction

Regular physical activity has long been regarded as an important component of a healthy lifestyle. Cross-sectional epidemiologic studies and controlled, experimental investigations have demonstrated that physically active adults, as compared with their sedentary counterparts, tend to develop and maintain higher levels of physical fitness. Constant-load low-volume High-intensity interval training may induce metabolic adaptations that reduce the risk for inactivity-related disorders in previously sedentary middle-aged adults[1]. During exercise, heart rate (HR) and myocardial contractility will be increased to satisfy energy demands of working muscles. Its nervous modulation is considered to be due to the vagal withdrawal at low-intensity exercise and the combination of vagal withdrawal and sympathetic activation at moderate or high-intensity exercise[2]. With the cessation of exercise, the decrease in HR immediately after exercise is mainly thought to be a function of vagal reactivation[3]. The further decrease in HR to the preexercise value also depends on the gradual withdrawal of the sympathetic system[4]. A low value for heart-rate recovery after exercise testing, which has been

previously shown to be a marker of decreased vagal activity, is a powerful and independent predictor of the risk of death[5]. Physical training was shown to increase cardiac vagal tone[6]. Training heart rate is also helpful for people who want to exercise for health benefits. Research shows that untrained individuals will begin to improve their aerobic fitness when they exercise regularly at 50% of their maximum heart rate[7]. Therefore the present study was undertaken to assess the influence of vagal modulation by evaluating percent recovery heart rate of young healthy physically active and sedentary male adult subjects of Bijapur district of Karnataka, India

## 2. Materials and Methods

Healthy adult males belonging to mean age group of  $34.30 \pm 3.86$  years, with sedentary life style (n=29) and age and sex matched young healthy adults (n= 29) belonging to mean age group of  $33.33 \pm 3.68$  (Table1), with regular physical activity (moderate-intensity aerobic/brisk walk for a minimum of 30 min on five days each week) as per guidelines of American College of Sports Medicine 2007[8] (n=29) were selected as subjects from the general population of north Karnataka, randomly for this study. No prior history of unstable cardiovascular, peripheral vascular and respiratory disease, or musculoskeletal lesions was considered before inclusion of all the subjects in this study.

\* Corresponding author:

kusaldas@yahoo.com (Kusal K. Das)

Published online at <http://journal.sapub.org/medicine>

Copyright © 2012 Scientific & Academic Publishing. All Rights Reserved

All the selected subjects were non-smokers, non-alcoholics, non-obese, non-diabetics and they were not under any medications which could affect their sub maximal aerobic fitness.

The entire protocol was explained to all the subjects and written consent was obtained from them. The experimental protocol was approved by Institutional Ethical Committee as per the ICMR guidelines 2006 on human subjects[9]. Height (cm) and weight (kg) were recorded to calculate BMI. All the subjects were asked to take rest in recumbent position for half an hour. The exercise tests were carried out in a well-ventilated room, using cycle ergometer (AFTON-ST-2488R). They were also properly instructed on how to perform the exercise test with prior demonstrations. Pre-exercise heart rate (HR) was measured before exercise by ECG. The testing protocol was comprised of an initial two-minute warm up exercise at a work load of 20 Watts, followed by a linear increase in workload every minute until the subject reached a targeted 70% of age-predicted maximum HR. The age-predicted maximum HR (HRmax) was determined as  $HR_{max} = 208 \text{ minus } (0.7 \times \text{age})$ [10]. The peak exercise HR was the highest value achieved at the termination of exercise test, after the subject has reached his predetermined HR. The post exercise HR rate was obtained 2 minutes after cessation of exercise. Electrocardiogram (ECG) was monitored by Ambulatory ECG Recorder (CARDIART-6108 BPL Electronic Company, Limited.). Relative decrease of HR immediately after exercise was calculated as a percentage of peak HR:  $(\text{peak HR} - \text{post HR}) / \text{peak HR} \times 100$ . Pre-exercise blood pressure (SBP and DBP) were measured when sitting on the cycle ergometer without pedaling by electronic blood pressure meter (EW 280W Japan). The peak exercise SBP and DBP were recorded immediately after exercise and post-exercise blood pressure were recorded 2 minutes after the cessation of exercise by using the following equation:  $(\text{peak BP} - \text{post BP}) / \text{peak BP} \times 100$ , [11,12]. All the values (mean + SD) of sedentary group and physically active group subjects were compared by student unpaired 't' test.  $p < 0.05$  has been considered as level of significance

### 3. Results & Discussion

Pre-exercise HR is significantly less in physically active subjects when compared to sedentary subjects (Table 2). The lowered resting heart rate from exercise is proposed to be due primarily to an increase in the parasympathetic activity with a minor decrease in sympathetic discharge[13, 14]. The physically active group showed a significantly faster decrease in heart rate to pre exercise value than the age matched sedentary controls. HR recovery after exercise depends on several factors: the intensity of exercise, the cardiorespiratory fitness, cardiac ANS modulation, hormones changes and baroreflex sensitivity[11]. Physical training was shown to increase cardiac vagal tone[6] and to accelerate HR recovery after exercise[15]. Recovery of the heart rate immediately after exercise is a function of vagal reactivation. A value of 12 beats per minute for the recovery of heart rate was considered abnormal. Increasing levels of physical fitness were strongly correlated with decreasing rates of abnormal values for the recovery of heart rate among men and women[5].

There was no significant difference observed between the pre-exercise SBP of both the groups but after exercise it was found to be significantly higher in physically active group than sedentary controls. Percent decrease of systolic blood pressure immediately after exercise was significantly more in physically active group than sedentary controls (Table 3). The higher peak exercise blood pressure in the in physically active group after exercise may be due to increase in left ventricular mass[16, 17] or may be due to training-induced augmentation of early diastolic filling at rest and during exercise. It may be an important adaptation to allow an increase in stroke volume at rest and an increase in stroke volume, cardiac output, and maximal oxygen consumption during exercise[18]. Percent decrease of systolic blood pressure immediately after exercise was significantly more in physically active group than sedentary controls which may be attributed to augmented baroreflex sensitivity in high-fit male subjects[19] and greater capacity of reflex cardiovascular modulation after exercise than sedentary controls[20].

**Table 1.** Physical parameters

Parameters	Age (years)	Height (cm)	Weight (kg)	BSA (m <sup>2</sup> )	BMI (kg/m <sup>2</sup> )
Sedentary group (n=29)	34.30 ± 3.86	168.46 ± 5.12	67.00 ± 12.00	1.77± 0.15	23.59± 3.70
Physically active group (n=29)	33.33 ± 3.68	167.00 ± 6.34	68.66±12.12	1.78±0.18	24.52± 3.22
Unpaired 't' test Physically active vs. Sedentary group	t=0.979, df=56, p=0.3316	t=0.965, df=56, p=0.3388	t=0.524, df=56, p=0.6023	t=0.230, df=56, p=0.8191	t=1.021, df=56, p=0.3116

All the values are mean ± SD. \*  $p < 0.05$ , \*\*  $p < 0.01$  (Significantly different from untrained controls)

**Table 2.** Recovery Heart Rate

Parameters	Pre-Exercise Heart Rate (bpm)	Peak-Exercise Heart Rate (bpm)	Post-Exercise Heart Rate (bpm)	Percent Recovery Heart Rate (bpm)
Sedentary group (n=29)	72.67± 3.70	129.96± 9.15	95.5 ±6.74	25.31± 6.85
Physically active group (n=29)	67.86 ± 7.21	132.7 ± 7.09	83.4 ± 8.75	38.12 ± 8.23
Unpaired 't' test Physically active vs. Sedentary group	t = 3.1963, df = 56, P = 0.0023*	t = 1.2747, df = 56, P = 0.2077	t = 5.8996, df = 56, P < 0.0001***	t = 6.4424, df = 56, P < 0.0001***

All the values are mean ± SD. \* p < 0.05, \*\* p < 0.01 (Significantly different from untrained controls)

**Table 3.** Blood Pressure Recovery

Parameters	Sedentary group (n=29)	Physically active group (n=29)	Unpaired 't' test Physically active vs. Sedentary group
Pre- Exercise SBP (mm Hg)	118.53± 11.07	115.46 ± 10.18	t = 0.7906, df = 28, P = 0.4358
Peak- Exercise SBP (mm Hg)	164.41 ± 8.26	172.26 ± 9.61	t = 3.3360, df = 56, p = 0.0015
Post- Exercise SBP (mm Hg)	152.47± 9.94	142.47 ± 9.49	t = 3.9185, df = 56, P = 0.0002
Percent decrease of the systolic blood pressure (SBP)	7.26±5.15	17.29±8.23	t = 5.5635, df = 56, p < 0.0001
Pre- Exercise DBP (mm Hg)	76.13 ± 8.57	77.33 ± 7.88	t = 0.5551, df = 56, P = 0.5811
Peak- Exercise DBP (mm Hg)	79.21±7.32	82.43± 8.53	t = 1.5427, df = 56, p = 0.1285
Post- Exercise DBP (mm Hg)	75.87 ± 3.74	76.86 ± 6.86	t = 0.6823, df = 56, P = 0.3227
Percent decrease of the diastolic blood pressure	4.21±2.82	5.57 ± 1.93	t = 2.1432, df = 56, p = 0.0365

All the values are mean ± SD. \* p < 0.05, \*\* p < 0.01 (Significantly different from untrained controls)

## 4. Conclusions

The present study clearly reflects a greater vagal modulation in case of physically active subjects as compared to sedentary counterparts which was found to be beneficial one as per homeostasis is concerned. Hence it may be concluded from the study that life style of an individual definitely has an impact on percent recovery heart rate which is one of the potential markers of the cardiovascular fitness.

## REFERENCES

- [1] Hood MS, Little JP, Tarnopolsky MA, Myslik F, Gibala MJ. 2011, Low-volume interval training improves muscle oxidative capacity in sedentary adults, *Med Sci Sports Exerc.*, 43(10), 1849-56.
- [2] Kluess, H.A., Wood, R.,H., Welsch, M.,A., 2000, Vagal modulation of the heart and central hemodynamics during handgrip exercise, *Am J Physiol*, 279, 1648-1652
- [3] Arai, Y., Saul, J.P., Albrecht, P., Hartley., L.H., Lilly, L.S., Cohen, R.J. Colucci, W.,S., (),1989, Modulation of cardiac autonomic activity during and immediately after exercise, *Am J Physiol*, 256, 132-141
- [4] Perini, R., Orizio, C., Comande, A., Castellano, M., Beschi, M. and Veicsteinas, A. () ., 1989, Plasma norepinephrine and heart rate dynamics during recovery from submaximal exercise in man. *Eur J Appl Physiol*, 58, 879-883.
- [5] Cole, C,R., Blackstone, E, H., Pashkow, F,J., Snader, C,E., Lauer, M,S., 1999, Heart rate recovery immediately after exercise as a predictor of mortality, *The New Engl J Med*, 341, 1351-1357.
- [6] Levy, W.C., Cerqueira, M.D., Harp, G.D., Johannessen, K.A., Abrass, I.B., Schwartz, R.S. and Stratton, J.R., 1998, Effect of endurance exercise training on heart rate variability at rest in healthy young and older men, *Am J Cardiol* , 82, 1236-1241.
- [7] Pollock, M.L., et al. 1998, American College of Sports Medicine Position Stand. The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness, and flexibility in healthy adults, *Med Sci Sports Exerc*, 30, 975-991
- [8] Haskell WL, Lee IM., Pate RR., Powell KE., Blair SN., Franklin BA., Macera CA., Heath GW., Thompson PD.,

- Bauman A., 2007 Aug, Physical activity and public health: updated recommendation for adults from the American College of Sports Medicine and the American Heart Association., *Med Sci Sports Exerc*, 39(8), 1423-34.
- [9] Indian Council of Medical Research. Ethical Guidelines for Biomedical Research on Human Subjects. New Delhi, 2006
- [10] Tanaka, H., Monahan, K.D., Seals, D.R., Age-predicted maximum heart rate revisited, *J Am Coll Cardiol* 37(1), 153-156, 2001.
- [11] Du, N., Bai, S., Oguri, K., Kato, Y., Matsumoto, I., Kawase, H., and Matsuoka T., 2005, Heart Rate Recovery After Exercise And Neural Regulation Of Heart Rate Variability In 30-40 Year Old Female Marathon Runners, *J Sports Sci Med*, 4:9-17
- [12] Dimpka, U., Ugwu, A.C., 2009, Determination of Systolic Blood Pressure Recovery Time after Exercise in Apparently Healthy, Normotensive, Nonathletic Adults and the Effects of Age, Gender and Exercise Intensity, *Int J Exerc Sci*, 2(2), 115-130,
- [13] Katona, P. G., McLean, M., Dighton, D. H., Guz, A., 1982, Sympathetic and parasympathetic cardiac control in athletes and nonathletes at rest. *J Appl Physiol*, 52, 1652-1657.
- [14] Smith, M. L., Hudson, D. L., Graitzer, H. M., Raven, P. B., 1989. Exercise training bradycardia: the role of autonomic balance. *Med Sci Sports Exerc*, 21, 40-44.
- [15] Darr, K.C., Bassett, D.R., Morgan, B.J., Thomas, D.P., 1988, Effects of age and training status on heart rate recovery after peak exercise. *Am J Physiol*, 254, H340-H343.
- [16] Whyte, G.P., George, K., Nevill, A., Shave, R., Sharma, S., and McKenna, W. J., 2004, Ventricular morphology and function athletes: a meta-analysis, *International J Sports Med*, 25, 380-383
- [17] Sung, J., Ouyang, P., Silber, H.A., Bacher, A.C., Turner, K.L., DeRegis, J.R., Hees, P.S., Shapiro, E.P., Stewart, K.J., 2003, Exercise blood pressure response is related to left ventricular mass, *J Hum Hyperten*, 17, 333-338.
- [18] Levy, W.C., Cerqueira, M.D., Abrass, I.B., Schwartz, R.S. and Stratton, J.R. 1993, Endurance exercise training augments diastolic filling at rest and during exercise in healthy young and older men, *Circulation*, 88, 116-126.
- [19] Barney, J.A., Ebert, T.J., Groban, L., Farrell, P.A., Hughes, C.V., Smith, J.J., 1988, Carotid baroreflex responsiveness in high-fit and sedentary young men. *J Appl Physiol*, 65, 2190-2194.
- [20] Laukkanen, J.A., Kurl, S., Salonen, R., Lakka, T.A., Rauramaa, R. and Salonen, J.T., 2004, Systolic blood pressure during recovery from exercise and the risk of acute myocardial infarction in middle-aged men, *Hypertension*, 44, 820-825.