

## TITLE PAGE

### HEART RATE RECOVERY NORMALITY DATA RECORDED IN RESPONSE TO A MAXIMAL EXERCISE TEST IN PHYSICALLY ACTIVE MEN

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## ABSTRACT

**Background.** Despite a growing clinical interest in determining the heart rate recovery (HRR) response to exercise, the limits of a normal HRR have not yet been well established. **Purpose.** This study was designed to examine HRR following a controlled maximal exercise test in healthy, physically active adult men. **Methods.** The subjects recruited (n=789) performed a maximal stress test on a treadmill. HRR indices were calculated by subtracting the 1<sup>st</sup> and 3<sup>rd</sup> minute heart rates during recovery from the maximal heart rate obtained during stress testing and designated these as HRR-1 and HRR-3, respectively. The relative change in HRR was determined as the decrease in HR produced at the time points 1 min and 3 min after exercise as a percentage of the peak HR ( $\%HRR-1/HR_{peak}$  and  $\%HRR-3/HR_{peak}$ , respectively). Percentile values of HRR-1 and HRR-3 were generated for the study population. **Results.** Mean HRR-1 and HRR-3 were  $15.24 \pm 8.36$  bpm and  $64.58 \pm 12.17$  bpm, respectively and  $\%HRR-1/HR_{peak}$  and  $\%HRR-3/HR_{peak}$  were  $8.60 \pm 4.70\%$  and  $36.35 \pm 6.79\%$ , respectively. Significant correlation was detected between Peak  $VO_2$  and HRR-3 ( $r=0.36$ ;  $p<0.001$ ) or  $\%HRR-3/HR_{peak}$  ( $r=0.23$ ;  $p<0.001$ ). **Conclusions.** Our study provides normality data for heart rate recovery following a maximal ergometry test obtained in a large population of physically active men.

Keywords: heart rate recovery, physical fitness, ergometry test, health.

## ABBREVIATIONS

BMI body mass index  
HR heart rate  
HRR heart rate recovery  
HRrest heart rate at rest  
Peak  $VO_2$  peak oxygen uptake  
RER respiratory exchange ratio  
 $VO_2$  oxygen uptake  
 $VO_{2max}$  maximal oxygen uptake

## **INTRODUCTION**

Heart rate recovery (HRR) is a predictive variable used to assess physiological stress, the physical condition of an individual (Chorbajian 1971), to monitor the response of an athlete to training, and for training prescription (Borresen and Lambert 2007). Although early studies examining the HRR response to intense physical exercise date back to 1931 (Boas 1931), clear reference values for a normal HRR response to a standardized maximal test performed under controlled conditions are still lacking.

A faster HRR response to exercise has been reported in individuals with a greater aerobic capacity (Hirsh et al. 2006; Goldberg and Shephard 1980; Kirby and Hartung 1980; Sidney and Shephard 1978; Brown and Brown 2007; Borresen and Lambert 2008; Darr et al. 1988) as well as in subjects trained in aerobic resistance exercise (Bosquet et al. 2007; Goldsmith et al. 2000; Huikuri et al. 1999; Parekh and Lee 2005; Esco et al. 2010; Savin et al. 1982; Cole et al. 1999; Borresen and Lambert 2007; Bunc et al. 1988; Short and Sedlock 1997; Buchheit and Gindre 2006; Buchheit et al. 2007; Cooke and Carter 2005; Goldberger et al. 2006; Seiler et al. 2007; Martinmaki and Rusko 2008). This suggests that post-exercise HRR is a marker of training-induced changes in autonomic control (Borresen and Lambert 2008) that take the form of faster vagal system reactivation and/or faster sympathetic tone withdrawal.

In adults, normality values have been defined for HRR one minute after finishing a standard exercise stress test: <12 bpm for active recovery or <18 bpm for passive recovery (Nishime et al. 2000). However, as far as we are aware, the field of exercise physiology lacks studies examining the normality of HRR in large populations of healthy, physically active subjects in response to a controlled maximal exercise test. The purpose of this study was to examine HRR following a controlled maximal exercise test in healthy, physically active adult men.

## **METHODS**

### *Subjects*

Seven hundred and eighty nine men were recruited from a Sports Club. The subjects visited our center for a medical examination. Subject training varied widely but the normal training schedule of all subjects involved running, as at least 3 sessions per week over the past 6 months or more.

Individuals were excluded if they had a diagnosed disease, were under cardioactive medication or showed an abnormal cardiovascular or respiratory response during the stress test.

Participation in the study was voluntary in agreement with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants. The study protocol was approved by the Human Ethics Committee of the Universidad Complutense.

### *Exercise stress test*

All the subjects undertook a maximal exercise test of 8-12 min duration on a treadmill (h/p/Cosmos quasar, Nussdorf-Traunstein, Germany). Starting treadmill speed was 8-12 km/h, depending on the subject's training level, and increased by 0.5 km/h every minute until exhaustion. The slope was set at 1% throughout the test. Test protocols were individualized based on a pre-test interview to estimate each subject's exercise capacity. Subjects were encouraged to exercise until muscle fatigue. The test termination criteria followed AHA/ACC guidelines (Gibbons et al. 2002).

During the test, gas-exchange data were collected using a breath-by-breath metabolic system (ZAN 600 USB CPX, Messgeräte Gmbit, Germany). Maximal oxygen uptake ( $\text{VO}_2\text{max}$ ) was determined when two of the following conditions were met: a plateau in  $\text{VO}_2$  despite an increased treadmill speed; respiratory exchange ratio (RER)  $\geq 1.10$ ; heart rate within 10 beats of the age-predicted maximum ( $220 - \text{age}$ ); or volitional fatigue (Esco et al. 2010). During the test, heart rate (HR) was continuously monitored by 12-lead electrocardiography.

Before the test (24 h), subjects refrained from consuming caffeine or other stimulants and from intense physical exercise. They were also instructed not to have a large meal in the 4 h leading up to the test. All tests were performed between 3 pm and 9 pm. To ensure that the environment was appropriately controlled, the laboratory was kept as quiet as possible during all procedures. During trials, the testing room was kept at  $21 \pm 2^\circ\text{C}$  and 45-55% relative humidity (Ostojic et al. 2010).

### *Heart rate recovery (HRR)*

The HR recovery period after exercise (3 min) was divided into two stages: (1) the first 2 min were undertaken at a treadmill speed of 4 km/h and slope 0%, and (2) during the third minute of passive recovery, the subject stood still on the treadmill with arms at the sides.

Immediately after completing the stress test, the subject's mouthpiece was removed while the ECG was continued during recovery to record HR at 1 and 3 min of recovery. Heart rates were taken from the computerized reports on which the HR was the average of the last five RR intervals to prevent any false result due to sinus arrhythmias.

HRR indices were calculated by subtracting 1<sup>st</sup> and 3<sup>rd</sup> minute heart rate on recovery period from the maximal heart rate obtained during stress testing and designated as HRR-1 and HRR-3, respectively. (Tulumen et al. 2011; Cole et al. 1999). The relative change in HRR was determined as the decrease produced in HR at 1 min and 3 min after finishing exercise expressed as a percentage of the peak HR ( $\% \text{HRR-1}/\text{HR}_{\text{peak}}$  and  $\% \text{HRR-3}/\text{HR}_{\text{peak}}$ , respectively). We also calculated the relative decrease in HR at 1 min and 3 min post-exercise as a percentage of the increase produced from resting HR to peak HR ( $\% \text{HRR-1}/(\text{HR}_{\text{peak}} - \text{HR}_{\text{rest}})$  and  $\% \text{HRR-3}/(\text{HR}_{\text{peak}} - \text{HR}_{\text{rest}})$ , respectively).

### *Percentiles*

Percentiles (5<sup>th</sup> to 95<sup>th</sup>) of the different heart rate indices were generated for the study population.

### *Statistical analysis*

The normality of the data distribution was determined using the Shapiro-Wilk test. Only the data related to HRR-1 showed a non-normal distribution. Continuous variables are provided as the mean  $\pm$  standard deviation. The relationship between heart rate recovery and VO<sub>2</sub>max was assessed by calculating Spearman and Pearson correlation coefficients. Significance was set at  $p < 0.05$ . All statistical tests were performed using commercially available software (SPSS, Version 19.0, Inc., Chicago, IL, USA).

## **RESULTS**

### *Study population*

The demographic characteristics of the subjects (n=789) were: age 38.50 $\pm$ 8.64 years (20-65 years), height 176.5 $\pm$ 6.8 cm, weight 77.43 $\pm$ 10.98 kg and BMI 24.82 $\pm$ 3.10 kg/m<sup>2</sup>.

### *Chronotropic response and heart rate recovery*

The mean values obtained for the subjects' chronotropic response and heart rate recovery are provided in Table 1.

*Table 1. Subjects' chronotropic responses and heart rate recovery. Data are expressed as mean  $\pm$  SD.*

Mean HRR-1 and HRR-3 were 15.24 $\pm$ 8.36 bpm and 64.58 $\pm$ 12.17 bpm, respectively, corresponding to mean HR values of 162.60 $\pm$ 13.88 and 113.27 $\pm$ 14.63 bpm recorded at these time points; %HRR-1/HR<sub>peak</sub> and %HRR-3/HR<sub>peak</sub> were 8.60 $\pm$ 4.70% and 36.35 $\pm$ 6.79%, respectively; and %HRR-1/(HR<sub>peak</sub>-HR<sub>rest</sub>) and %HRR-3/(HR<sub>peak</sub>-HR<sub>rest</sub>) were 12.84 $\pm$ 6.97% and 54.08 $\pm$ 9.38% respectively.

### *Percentiles*

Percentiles 5<sup>th</sup> to 95<sup>th</sup> for the HRR indices are graphically represented in Figure 1.

**Fig.1** Percentiles (5<sup>th</sup>, 25<sup>th</sup>, 75<sup>th</sup>, 95<sup>th</sup>) for the HRR indices

### *Relationship between heart rate recovery and Peak VO<sub>2</sub>*

Peak VO<sub>2</sub> showed significant correlation with HRR-3 ( $r=0.36$ ;  $p<0.001$ ) and %HRR-3/HR<sub>peak</sub> ( $r=0.23$ ;  $p<0.001$ ), but no correlations were observed with the rest of the HR

recovery indices ( $p>0.05$ ). **Fig.2 and Fig.3** shows HRR-3 and %HRR-3/HR<sub>peak</sub> plotted against Peak VO<sub>2</sub>, respectively.

*Fig.2 Correlation between HRR-3 (bpm) and Peak VO<sub>2</sub> (ml/kg/min)*

*Fig.3 Correlation between %HRR-3/HR<sub>peak</sub> (%) and Peak VO<sub>2</sub>*

## DISCUSSION

The main objective of our study was to provide normality data for heart rate recovery after a maximal exercise test in a wide age range (20-65 years) of physically active men. As far as we are aware, this is the first report to provide normality HRR data for a large population of male subjects that will be useful as reference data for graded exercise testing under gas exchange monitoring in health physically active individuals. These data will be especially useful considering that in healthy subjects heart rate recovery seems to show good intrasubject reproducibility and excellent retest reliability up until the fifth minute of recovery (Tulumen et al. 2011).

A rapid HR recovery post moderate to intense exercise has been used as an indicator of good cardiovascular function (Chorbajian 1971). In effect, we detected significant correlation between Peak VO<sub>2</sub> (ml/kg/min) and HRR-3 ( $r=0.36$ ;  $p<0.001$ ) or %HRR-3/HR<sub>peak</sub> ( $r=0.23$ ;  $p<0.001$ ), confirming the effect of aerobic capacity on the HRR rate noted by other authors (Heffernan et al. 2007; Darr et al. 1988; Hirsh et al. 2006; Myers et al. 2007; Carnethon et al. 2005). Collectively these data indicate that cardiorespiratory fitness is especially linked to HRR at 3 min post-exercise and shows poor correlation with the HR recovery produced 1 min after cessation of exercise.

To interpret the HRR data obtained at 1 minute versus 3 minutes post-exercise, we should take into account the fact that early HR recovery (<1min) can be mainly attributed to vagal tone reactivation (Imai et al. 1994; Perini et al. 1989; Coote 2010), and later recovery (>2min) is likely attributed to a drop in sympathetic activity and to humoral factors such as catecholamines (Perini et al. 1989; Kannankeril et al. 2004; Gibbons et al. 2002; Nilsson et al. 2007; Huikuri et al. 1999; Jouven et al. 2005; Hart et al. 2006).

Having established that HR recovery in the first minute after exercise cessation is not related to the intensity of exercise or VO<sub>2</sub>max, it would appear that central mechanisms such as the release of inhibiting signals from the motor cortex to the parasympathetic center, along with exercise cessation are mostly responsible for the early HR recovery response, as also proposed by others (Baraldi et al. 1991). The role played by regular physical activity in this early response remains unclear.

In contrast, the later stage of HR recovery (3 min in our study) does seem to be related to a reduction in sympathetic activity induced by the reduced activation of peripheral muscle mechanoreceptors and chemoreceptors once the exercise stimulus has been removed. An improved aerobic capacity would thus be linked to an earlier recovery of intracellular homeostasis and this will translate to reduced sympathetic impulses from the peripheral muscle receptors. This hypothesis is supported by the correlation observed here between the variables related to aerobic capacity and a faster HR

recovery at 3 minutes post-exercise. Thus, HRR-3 in response to a maximal stress test could be an indicator of an individual's aerobic capacity.

We propose that rather than absolute HR indices (HRR-1 and HRR-3), it is perhaps best to consider normal HRR as a % of the peak HR recorded during the graded cardiopulmonary test, considering that this was a maximal exercise test. Given that our subjects were all physically active and stress tests were performed at the individual's maximal intensity, we feel it preferable to express HRR relative to  $HR_{peak}$  as a measure of HR recovery applicable to all subjects regardless of age.

Moreover, the expression of HRR at both 1 and 3 minutes relative to the increase in HR produced in the stress test from resting HR ( $\%HRR/(HR_{peak}-HR_{rest})$ ) is also useful but has the limitation that the true value of  $HR_{rest}$  is difficult to measure in a clinical setting. This difficulty may be attributed to individual emotional factors associated with performing a graded cardiopulmonary test.

Although passive recovery leads to improved HRR following exercise compared to active recovery (Takahashi et al. 2005), we adopted a mixed recovery protocol during which recovery was active in the first two minutes and passive in the third minute. Similar recovery protocols have been used by others (Maeder et al. 2009; Darr et al. 1988). This is because stress tests in athletes are maximal and it is better not to abruptly stop exercise after reaching a point of exhaustion.

Previous studies have shown that a physical training program used in sedentary subjects can improve post-exercise HRR (Sugawara et al. 2001; Martinmaki and Rusko 2008; Seiler et al. 2007). Heart rate recovery was also found to improve in cyclists with increasing training intensity (Lamberts et al. 2009), suggesting that HRR may be a sensitive monitoring tool to track changes over time in performance parameters in already well-trained cyclists. Perhaps genetics could markedly influence the training response in terms of HRR (Singh et al. 1999; Singh et al. 2002; Hagberg et al. 2001). This could partly explain the large interindividual variation observed in the indices that reflect this response (Hautala et al. 2006).

Two main limitations apply to our study. We had no data on subjects' training characteristics, so we were unable to relate it with the HRR. Moreover, our results are only referred to men and treadmill

In conclusion, this study provides normality data for heart rate recovery following a maximal ergometry stress test in a large population of physically active men. These data suggest that heart rate recovery 3 minutes after exercise cessation is related more to the aerobic capacity of the subject than recovery at 1 minute post-exercise.

## **ETHICAL STANDARDS AND CONFLICT OF INTEREST**

The study protocol was approved by the review board of the Universidad Complutense de Madrid and comply with the current laws of Spain

The authors declare that they have no conflicts of interest

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## TABLES

*Table 1. Subjects' chronotropic responses and heart rate recovery. Data are expressed as mean  $\pm$  SD.*

<b>Variable</b>	<b>Mean <math>\pm</math> SD</b>
Exercise time (min)	10.46 $\pm$ 1.90
Resting heart rate (bpm)	57.96 $\pm$ 10.32
HR peak (bpm)	177.85 $\pm$ 11.17
Percentage-predicted heart rate (%)	98.05 $\pm$ 5.30
Heart rate at first minute of recovery (bpm)	162.60 $\pm$ 13.88
HRR-1 (bpm)	15.24 $\pm$ 8.36
%HRR-1/HRpeak (%)	8.60 $\pm$ 4.70
%HRR-1/(HRpeak-HRrest) (%)	12.84 $\pm$ 6.97
Heart rate at third minute of recovery (bpm)	113.27 $\pm$ 14.63
HRR-3 (bpm)	64.58 $\pm$ 12.17
%HRR-3/HRpeak (%)	36.35 $\pm$ 6.79
%HRR-3/(HRpeak-HRrest) (%)	54.08 $\pm$ 9.38
Peak VO <sub>2</sub> (ml/kg/min)	49.83 $\pm$ 9.77

*HR: heart rate, HRR-1: heart rate peak minus heart rate at min 1 of recovery, HR<sub>peak</sub>: heart rate peak, HRR-3: heart rate peak minus heart rate at min 3 of recovery, VO<sub>2</sub>: oxygen consumption*

## LEYENDS

**Fig.1** Percentiles (5<sup>th</sup>, 25<sup>th</sup>, 75<sup>th</sup>, 95<sup>th</sup>) for the HRR indices

*HRR-1: heart rate peak minus heart rate at min 1 of recovery, HR<sub>peak</sub>: heart rate peak, HRR-3: heart rate peak minus heart rate at min 3 of recovery; HR<sub>rest</sub>: heart rate at rest*

**Fig.2** Correlation between HRR-3 (bpm) and Peak VO<sub>2</sub> (ml/kg/min)

*HRR-3: heart rate peak minus heart rate at min 3 of recovery; VO<sub>2</sub>: oxygen consumption. Higher values of Peak VO<sub>2</sub> are significantly related to higher levels of HRR-3 (p<0.001, r=0.36)*

**Fig.3** Correlation between %HRR-3/HR<sub>peak</sub> (%) and Peak VO<sub>2</sub>

*HRR-3: heart rate peak minus heart rate at min 3 of recovery; HR<sub>peak</sub>: heart rate peak; VO<sub>2</sub>: oxygen consumption. Higher values of Peak VO<sub>2</sub> are significantly related to higher levels of %HRR-3/HR<sub>peak</sub> (p<0.001, r=0.23)*