Assessment of autonomic function as marker of training status: the role of heart rate recovery after exercise

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Abstract

Heart rate recovery (HRR) is the rate at which the heart rate returns to baseline after a period of exercise. HRR is a marker of autonomic function and a predictor of cardiovascular fitness in healthy subjects and in those with cardiovascular diseases. Moreover, HRR has been proposed as a marker of training status in athletes. Our aim was to perform a review of studies that evaluated HRR after exercise in trained and untrained healthy subjects and assessed its relationship with training status. Several studies suggest that the assessing of HRR after exercise may be useful to distinguish trained from untrained individuals and to establish an athlete’s state of training. However, standardization of measuring is required to compare between individuals.

Keywords: Sympathetic, Parasympathetic, Cardiovascular fitness

Introduction

The heart rate (HR) is controlled by the autonomic nervous system. From anatomical point of view the autonomic nervous system is made up of the sympathetic and parasympathetic systems that reach the heart with sympathetic and parasympathetic nerves, respectively. The initial increase in HR after the start of physical activity is determined by increased sympathetic and decreased parasympathetic activity. During exercise, cardiac output is adjusted based on metabolic demand. When the exercise stops, cardiac output is reduced by parasympathetic reactivation and sympathetic inhibition, and a
gradual return of HR to its previous resting level.

Heart rate variability (HRV) and heart rate recovery after exercise (HRR) are both markers of autonomic control of the heart. HRV is defined as the oscillation in the intervals between consecutive heart beats and spectral analysis of HRV gives an index of cardiac sympathovagal balance (Traina et al. 2010). Vagal-related HRV indices are generally associated with improved cardiorespiratory fitness and physical performance (Buchheit et al. 2011). Therefore, it seems that the relationship between endurance training and/or aerobic fitness and HRV is lost, or even reversed, within a well-trained runners (Lee and Mendoza 2011).

HRR is defined as the rate at which heart rate decreases, usually within minutes, after moderate to heavy exercise and is a consequence of parasympathetic re-activation and sympathetic withdrawal (Shetler et al. 2001). Several studies have identified HRR as a predictor of cardiovascular and all-cause mortality in healthy adults (Cole et al. 1999; Cole et al. 2000; Nishime et al. 2000), in patients with cardiovascular disease (Lahiri et al. 2008; Pitsavos et al. 2004; Watanabe et al. 2001) and with diabetes (Cheng et al. 2003). HRR has also been associated with cardiovascular fitness, aerobic fitness and endurance training (Dimpka 2009). A recent review suggests that HRR may be sensitive to detect changes in training status in athletes and could be a valuable tool to monitor and optimize training programs (Daanen et al. 2012).

Aim of our review was to analyze the rule of HRR in the assessment of autonomic function as marker of training status in athletes and less well-trained subjects.

Methods

Measure

HRR can be quantified by taking the absolute difference between the final HR at exercise completion and the HR recorded following 1- or 2-min of recovery (HRR$_{1\text{min}}$ or HRR$_{2\text{min}}$) (Buchheit & Gindre 2006; Cole et al. 1999) (Figure 1). Lamberts et al. (2004) indicate that HRR$_{1\text{min}}$ has better capacity to detect meaningful differences over time than HRR$_{2\text{min}}$ and suggest averaging the heart rate over 15 seconds (to take the 1-min value as the average over seconds 45 to 60). Alternatively, it’s possible to calculate the time constant of the HR decay obtained by fitting the post exercise HRR into a first-order exponential decay curve (HRR$_{\tau}$) (Perini et al. 1989), or to analyze the first 30-s of HRR via semi logarithmic regression analysis (T30) (Imai et al. 1994). Short-term HRR indexes (i.e. HRR$_{1\text{min}}$ and T30) could be considered as marker of cardiac parasympathetic outflow, since the initial rapid decline in HR (that is workload independent) is unaffected by sympathetic blockade but is influenced by parasympathetic blockade (Imai et al. 1994; Kannankeril et al. 2004). The second slow HR decay (that is believed to be workload dependent) (Buchheit & Gindre 2006; Imai et al. 1994) could be related to the gradual withdrawal of sympathetic activity and the clearance of stress system metabolites (Perini et al. 1989) (Figure 1).
**Figure 1.** Changes in heart rate during and following maximal exercise.
However, despite the parasympathetic origin of HRR indexes, several studies have shown a lack of association between HRR and heart rate variability (HRV) (Bucheit & Gindre 2006; Javorka et al. 2003; Lee & Mendoza 2012). This lack of association between HRR and HRV might be due to factors that interfere with parasympathetic outflow during the post-exercise recovery, such as environmental conditions (noise, light, temperature, etc.) that can exert a marked influence on HRV parameters and show a tendency to shift sympatho-vagal balance toward sympathetic predominance (Bucheit et al. 2007).

**Relationship between HRR and physiological factors**

Maximum oxygen uptake (VO$_2$max) is considered the best measure of cardiovascular fitness and is closely related to aerobic performance. Previous studies have observed faster HRR in athletes than in non-athletes. Du et al. (2005) reported that endurance training induced significant acceleration of HR recovery after exercise in female marathon runners, as results from higher aerobic capacity compared with untrained controls. Although age affects VO$_2$max and can affect HRR, Darr et al. (1988) have shown that trained subjects with high peak O$_2$ consumption, irrespective of age, demonstrated a significantly faster HRR than untrained subjects with low peak O$_2$ consumption, which was particularly marked during fast-phase recovery. Singh et al. (2008) reported that 1-min HR recovery after exercise is attenuated with age in children, and children with higher BMI and those with lower exercise endurance have slower 1-min HR recovery. In sedentary patients with type 2 diabetes, both HRR and VO$_2$max were significantly reduced (Cataldo et al. 2013), and in these subjects the positive linear correlation between HRR and VO$_2$max suggests that HRR might improve in response to training aimed to increase aerobic capacity (Cataldo et al. 2013).

**Relationship between HRR and training status**

Ota (2002) demonstrated that exercise endurance performance (all-out time, running distance, total work) is linearly related to HRR in sixty-five from 19 to 21 years old male and female students, and Otsuki et al. (2007) showed that both strength- and endurance-trained athletes have improved heart rate recovery after 8-min of steady-state exercise at 40% of maximal oxygen uptake compared to untrained controls. Sugawara et al. (2001) reported that 8 weeks of training in previously untrained men improved 30-s HRR. Two weeks of subsequent detraining maintained the improved HRR; however, by the fourth week of detraining, HRR had returned to baseline levels (Sugawara et al. 2001). Borresen & Lambert (2008) shown that endurance-trained athletes have an accelerated heart rate recovery after exercise; moreover, HRR responds to acute changes in training load: it slowed slightly after increases in training load, whereas tended to improve in subjects who decreased their training load (Borresen & Lambert 2007).

**Confounding factors**

Apart from training status, several factors can affect HRR. Antelmi et al. (2008) observed that younger individuals recovered faster than older ones from the second to the fifth minute after exercise and heart rate recovery in women was more rapid than in men. About gender, Arena et al. (2010) just found the opposite of Antelmi, while Lamberts et al. (2004) did not report gender-based differences. Zaim et al. (2010) indicate that there is a direct correlation between the peak HR obtained during a symptom-limited exercise test and the subsequent HRR measured at 1 minute. Training intensity and duration can affect the acute recovery of autonomic nervous system (ANS) balance after exercise. Seiler et al.
(2007) reported that in the highly trained endurance athlete, exercise for \( < \text{or} = 120\)-min below the first ventilatory threshold causes minimal disturbance in ANS balance. However, ANS recovery is found to be more rapid in highly trained than in trained subjects after high-intensity exercise. Lamberts & Lambert (2009) found that variation in heart rate decreased with increasing exercise intensity. Kaikkonen et al. (2008) investigated the HR recovery after exercise of different type and intensity. Increased exercise intensity resulted in lower HRR both in interval and in continuous exercise. In addition, when interval and continuous exercise were performed at a similar workload, HR recovery was lower after continuous exercise. Heffernan et al. (2006) observed that HR remained elevated to a greater extent after resistance compared to endurance exercise, and athletes engaged in intermittent sports are likely to have faster HRR during the first 20-s after maximal exercise than their counterparts trained for continuous performance (Ostojic et al. 2010).

HRR after 1 min was faster after cycle exercise compared to running; in contrast, HRR after 2- and 3-min were similar after both exercise (Maeder et al. 2009). To ensure the highest level of sensitivity in detecting meaningful changes in HRR over time, submaximal testing protocols should target exercise intensities ranging in-between 86-93% of heart rate maximum (Lamberts et al. 2011).

Our case studies
We report the example of our analysis on three healthy adult male subjects, one sedentary (SE), one endurance trained (ET), and one combined strength/endurance trained (CT), age 53, 54, and 51 respectively. Subjects performed a maximal incremental test on treadmill at the laboratory of the Sport and Exercise Sciences “DISMOT” Research Unit of the University of Palermo. HR has reached a peak of 121 bpm in SE, 139 bpm in ET, and 153 bpm in CT. HRR\(_{1\text{min}}\) and HRR\(_{2\text{min}}\) were respectively 19 and 30 bpm in SE (Figure 2), 25 and 44 bpm in ET (Figure 3), and 27 and 55 bpm in CT (Figure 4). SE had the lowest values of both HRR\(_{1\text{min}}\) and HRR\(_{2\text{min}}\), and among the trained subjects CT showed better values than ET.

Figure 2. Changes in heart rate during exercise and heart rate recovery 1 and 2 min after exercise in untrained subject.

![Figure 2](image-url)
**Figure 3.** Changes in heart rate during exercise and heart rate recovery 1 and 2 min after exercise in endurance trained subject.

**Figure 4.** Changes in heart rate during exercise and heart rate recovery 1 and 2 min after exercise in combined strength/endurance trained subject.
Conclusions

Several studies show that HRR is faster in trained than in untrained healthy subjects and is capable to quantify differences in training status of healthy individuals. These findings suggest that the assessing of HRR after exercise may be useful to distinguish trained from untrained individuals and to establish an athlete's state of training. The use of HRR recorded under standardized conditions 1 or 2 minutes after exercise may be a practical, reliable and quantifiable measure of the body’s current capacity to respond to stress with the aim to optimize training programs and to tracking an individual progression. However, the effects of confounding factors such as age, gender, type and intensity of exercise, need to be taken in account when interpreting HRR to compare between individuals.

References


