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Research article

Heart rate recovery after submaximal exercise in four different recovery protocols in male athletes and non-athletes

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Abstract

The effects of different recovery protocols on heart rate recovery (HRR) trend through fitted heart rate (HR) decay curves were assessed. Twenty one trained male athletes and 19 sedentary male students performed a submaximal cycle exercise test on four occasions followed by 5 min: 1) inactive recovery in the upright seated position, 2) active (cycling) recovery in the upright seated position, 3) supine position, and 4) supine position with elevated legs. The HRR was assessed as the difference between the peak exercise HR and the HR recorded following 60 seconds of recovery (HRR_{60}). Additionally the time constant decay was obtained by fitting the 5 minute post-exercise HRR into a first-order exponential curve. Within-subject differences of HRR_{60} for all recovery protocols in both groups were significant ($p < 0.001$) except for the two supine positions ($p > 0.05$). Values of HRR_{60} were larger in the group of athletes for all conditions ($p < 0.001$). The time constant of HR decay showed within-subject differences for all recovery conditions in both groups ($p < 0.01$) except for the two supine positions ($p > 0.05$). Between group difference was found for active recovery in the seated position and the supine position with elevated legs ($p < 0.05$). We conclude that the supine position with or without elevated legs accelerated HRR compared with the two seated positions. Active recovery in the seated upright position was associated with slower HRR compared with inactive recovery in the same position. The HRR in athletes was accelerated in the supine position with elevated legs and with active recovery in the seated position compared with non-athletes.

Key words: Heart rate recovery, autonomic activity, active recovery, physical activity.

Introduction

In order to return to a pre-exercise value following exercise, heart rate (HR) is mediated by changes in the autonomic nervous system but the underlying mechanisms governing these changes are not well understood (Imai et al., 1994). An initial exponential drop in HR is a result of rapid restoration of vagal tone after the cessation of exercise (Imai et al., 1994; Perini et al., 1993). Further decrease in HR is governed by progressive weakening of the sympathetic influence (Niewiadomski et al., 2007; Perini et al., 1993). To quantify parasympathetic reactivation after exercise, indices such as HR recovery (HRR) and heart rate variability (HRV) have been used (Barak et al., 2010; Buchheit et al., 2007; Buchheit and Gindre 2006; Goldberger et al., 2006; Otsuki et al., 2007; Pierpont et al., 2000; Pierpont and Voth, 2004).

The effects of different body postures on post-exercise heart rate recovery have partly been described by Takahashi who showed accelerated HRR in the supine compared with the upright sitting position (Takahashi et al., 2000). A more recent in depth analysis by Buchheit and colleagues examined the effects of four body postures (standing, sitting, supine and supine with elevated legs) on post-exercise parasympathetic reactivation (Buchheit et al., 2009). They showed poor HRR values in the standing upright position and no improvement in the supine position with elevated legs as opposed to plain supine position (Buchheit et al., 2009).

The effects of the upright sitting position with active recovery on HRR have not yet been examined. In the upright position blood from the central venous system is shifted to the lower extremities, eliciting an increase in the sympathetic mediated vasomotor activity for the preservation of arterial blood pressure (Hainsworth, 2000). After exercise cessation, the presence of the muscle pump in active recovery would prevent greater pooling of blood in the lower extremities and a decrease in ventricular filling, leading to greater parasympathetic reactivation and faster HRR (Carter et al., 1999). On the other hand the ongoing influence of central command might favour sympathetic activity leading to a somewhat slower HRR compared with inactive recovery (Carter et al., 1999).

In previous studies, HRR was described as the absolute difference between the final HR at exercise completion and the HR recorded following 60 seconds of recovery (HRR_{60}) or the time constant decay obtained by fitting the 5 minute post-exercise HRR into a first-order exponential curve (T) (Buchheit and Gindre, 2006; Pierpont et al., 2000; Pierpont and Voth, 2004). These indices describe the initial phase of HRR and they appear to be due to parasympathetic reactivation (Freeman et al., 2006; Buchheit and Gindre, 2006). T was found to be slower for the upright position than for the supine and supine with elevated legs, but no differences were observed between the two latter conditions (Buchheit et al., 2009).

In the present study the effects of four recovery conditions on HRR patterns were compared by fitting HR decay curves. Whether active recovery has a substantial influence on HRR in the seated position was also observed. Previous research has suggested faster HRR after

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exercise in well trained athletes and fit individuals (Buchheit and Gindre, 2006; Carnethon et al., 2005; Darr et al., 1988; Hagberg et al., 1980). In the present study, the HRR in athletes and sedentary subjects in relation to different recovery protocols was investigated.

Methods

Participants

Twenty four well trained male athletes and 19 sedentary male students gave written informed consent to take part in the study. The International Physical Activity Questionnaire (IPAQ) was used to confirm the physical activity level of the participants. The IPAQ proposes a categorical scoring protocol determining three levels of physical activity: low, moderate and high level (Craig et al., 2003). The group of athletes included participants with high physical activity scores who were professionally involved in various organized high and vigorous intensity activities (e.g. football, basketball, handball). The group of non-athletes included participants with low level physical activity scores who were not engaged in any organised physical activity for the last six months before the start of the investigation. All participants were in self-reported good health, without medications and had no medical history of cardiovascular diseases. They also underwent a general physical examination to exclude any acute diseases and ailments of the cardiorespiratory and locomotor system. Throughout the investigation orthostatic hypotension was observed in three athletes by the end of the fourth minute of recovery and none of the non-athletes. Their results were excluded from further computing for they were not suitable for exponential modeling. To assess individual differences in peak heart rate (HRpeak) a 30 second all-out Wingate test was used and peak heart rate was measured. In highly cooperative participants, peak HR during Wingate test might reach up to 94% of maximal HR obtained in aerobic cycling tasks (Hebestreit et al., 1993). All procedures conformed to the Declaration of Helsinki and were approved by the local ethics committee.

Experimental design

Measurements were undertaken in a quiet room, air temperature ranging from 22 to 24 ºC, between 9 and 12 h. Subjects were instructed to perform no strenuous exercise and have a solid night's rest the day before the testing.

Participants were asked to visit the exercise laboratory on five different occasions. On the first day peak power and peak HR were estimated by the Wingate anaerobic test and during the subsequent four visits the HRR measurements were performed. Participants were randomly allocated to one of four equally sized groups relating to the order in which each group performed measurements using different recovery protocols (inactive recovery in the upright seated position, active recovery in the upright seated position, supine position, and supine position with elevated legs).

In the upright seated position, the participants' feet were placed on a platform in front of the pedals while both legs were flexed at the knee at about 90º. This way most of the body weight was concentrated on the seat. The arms were placed over the thighs. In this position, participants reported that they felt comfortable and relaxed. In the seated position with active recovery, after exercise cessation participants continued to ride the cycle ergometer but without any workload to ensure muscle pump activity and venous return. Pedalling rate was set at 20 turns per minute. In the supine body position, participants were asked to lie facing upwards, flat on the bed which was located next to the cycle ergometer. And finally, in the supine position with elevated legs the lower legs were placed horizontally on a pillowed platform placed at the end of the bed while the thighs were vertical to the upper body. In this position both legs were flexed in the knee at approximately 90º.

Resting heart rate was obtained in the same position as the corresponding recovery protocol. Participants underwent a five minute submaximal cycling (Wattbike cycle ergometer, Wattbike Ltd, Nottingham, UK) with an intensity of 80% of individual peak HR values. Even with great inter-individual differences in HR response, loads at the level of 80% HRpeak are below lactate threshold and avoid primarily anaerobic work yet evoke a substantial cardiac autonomic response (Gladwell et al., 2010; Hofmann et al., 2001). Upon cessation of exercise a 5-minute recovery period in the appropriate recovery condition followed. Participants managed to move into the required body position in less than 5 seconds.

Assessment of post-exercise heart rate recovery

Digital ECG (VNS-Spektr, Neurosoft, Ivanovo, Russian Federation) was recorded during rest, exercise and recovery. A sampling rate of 1000 Hz was chosen and recordings were transferred to a PC via a USB interface. The epochs gained from the V5 lead were saved in a computer for further analysis. All R-R intervals were edited by visual inspection to exclude all the undesirable or ectopic beats. They were deleted together with the post extra systolic beat and replaced automatically with interpolated adjacent R-R interval values.

HRR was assessed during the 5 minute period following submaximal cycle exercise by the following methods: (1) the absolute difference between the peak HR at exercise completion and the HR recorded following 60 seconds of recovery (HRR_{60}) and (2) the time constant decay obtained by fitting the 5 minute post-exercise HRR into a first-order exponential curve (Pierpont et al., 2000, Pierpont and Voth, 2004, Buchheit and Gindre, 2006).

The resultant heart rates vs. time data were modelled with an iterative technique using MatLab (The Math Works Inc, Natick, MA, USA) to fit the following equation:

$$HR = HR_0 + HR_\Delta e^{(-t/T)}$$

where: HR=heart rate, HR_0=stabilized heart rate following exercise, HR_Δ=maximal heart rate – HR_0, t= time (s), T= decay constant

Data analysis

The distribution of each variable was examined with the Lilliefors normality test. Homogeneity of variance was verified by the Levene`s test. Heart rate recovery measures (HRR_{60}, T and HR_0) were analyzed using a two-factor repeated measure ANOVA, with one within-factor (´position´; SeatInact, SeatAct, Sup, SupElev) and one

between-factor (´activity level´; athletes, non-athletes). If a significant interaction was identified, Bonferroni´s post hoc test was used to further delineate the main effects of the recovery positions and activity levels. All statistical analyses were carried out using MatLab 6 software (The Math Works Inc, Natick, MA, USA) and the Statistica 8.0 software package (Statistica, StatSoft®, Tulsa, USA)

Results

Demographic characteristics of the participants are given in Table 1. Athletes engaged in organized high intensity physical activity for 8.8±2.4 years developed a higher peak power during Wingate anaerobic test than non-athletes (Table 1). Mean HRR_{60} values in each recovery condition for both groups are presented in Figure 1. We found a significant ´position´ ($p < 0.001$) and ´activity level´ effect ($p < 0.001$), without a ´position x activity level´ interaction ($p = 0.642$). Post hoc analyses showed significant within-subject differences for all recovery conditions in both groups ($p < 0.01$) except for the two supine positions ($p > 0.05$). Values of HRR_{60} were larger in the group of athletes for all conditions ($p < 0.001$).

Table 1. Demographic characteristics of participants. Data are means (±SD).

	Athletes	Non-athletes
Height (m)	1.83 (.05)	1.81 (.06)
Body mass (kg)	76.9 (7.0)	81.4 (9.2) *
Age (years)	19.9 (1.0)	20.5 (.6)
Activity (years)	8.8 (2.4)	0
Peak power (W)	996.2 (96.4)	768.6 (84.5) ***
HRpeak (beats·min^{-1})	186.2 (4.3)	183.1 (3.1) *

*p < 0.05, *** p < 0.001

Heart rate and heart rate recovery indices for each recovery condition in athletes and non-athletes are presented in Table 2 as means ± SD. For the time constant of HR decay (T) there was a significant ´position´ ($p < 0.001$) and ´activity level´ effect ($p < 0.001$), without a ´position x activity level´ interaction ($p = 0.18$). Post hoc analyses showed within-subject differences for all recovery conditions in both groups ($p < 0.01$) except for the two supine positions ($p > 0.05$). Between group difference was found for active recovery in the seated position and the supine position with elevated legs ($p < 0.05$), athletes demonstrating faster HRR as opposed to sedentary participants.

For the mean HR_0 values there was a significant ´position´ ($p < 0.001$) and ´activity level´ effect ($p < 0.001$), without a ´position x activity level´ interaction ($p = 0.651$). Post hoc analyses revealed no significant differences either between the two seated positions or between the two supine positions ($p > 0.05$). For athletes HR_0 was significantly lower in both supine positions than in the upright seated positions ($p < 0.01$). For non-athletes HR_0 in both supine positions was lower only compared with the inactive recovery in the upright seated positions ($p < 0.01$). The values of HR_0 were lower for the group of athletes in all recovery conditions ($p < 0.01$).

We found no correlation between peak power output and HRR parameters ($r < 0.3$ for all positions).

Discussion

The aim of the present study was to compare the effects of four recovery protocols (inactive recovery in the upright seated position, active recovery in the upright seated position, supine position, and supine position with elevated legs) on HRR through HRR_{60} and to discriminate between the corresponding fitted HR decay curves. Additionally we investigated the HRR in athletes vs. non-athletes in relation to different recovery protocols. The results from the present study showed that the supine position with or without elevated legs accelerated HRR more than the two seated conditions. Active recovery in

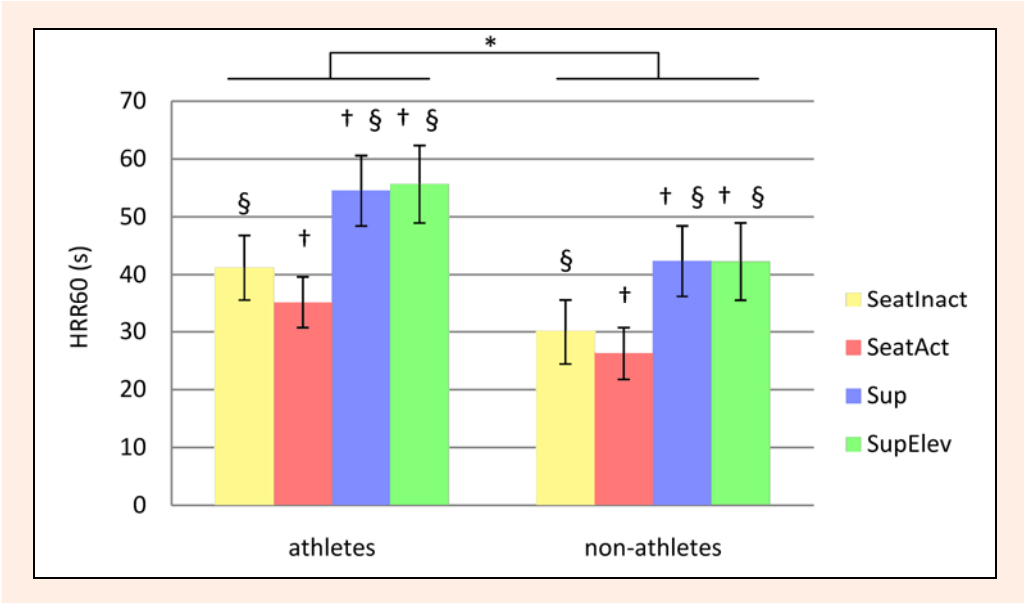

Figure 1. Heart rate recovery in four recovery conditions. Number of heart beats recovered in 60 s following exercise cessation (HRR_{60}). Values are presented for athletes and non-athletes for inactive recovery in the upright seated position (SeatInact), active recovery in the upright seated position (SeatAct), supine position (Sup), and supine position with elevated legs (SupElev) as means ± SD. * $p < 0.001$ athletes vs. non-athletes, † $p < 0.01$ vs. seated inactive, § $p < 0.01$ vs. seated active.

Table 2. Heart rate (bpm) and heart rate recovery indices for each recovery condition in athletes and non-athletes.

	Athletes				Non-athletes			
	SeatInact	SeatAct	Sup	SupElev	SeatInact	SeatAct	Sup	SupElev
HRrest	74 (8)	72 (11)	60 (10) †§	61 (11)†§	86 (8)*	88 (6)*	74 (10)†§*	77 (8)†§*
HRexercise	147 (1)	147 (2)	147 (2)	148 (2)	144 (3)*	144(3)*	143 (2)*	142 (2)*
T (s)	52.5 (14.6)§	74.1 (24.0)†	32.0 (9.1)†§	28.5 (5.8)†§	50.6 (14.9)§	90.6 (30.4)†*	36.4 (12.5)†§	32.9 (8.9)†§*
HR_0	89 (8)	88 (9)	79 (9)†§	79 (8)†§	100 (9)*	96 (8)*	90 (7)†*	90 (7)†*

HRrest, heart rate at rest; HRexercise, heart rate during exercise for the last 3 minutes; T, time constant of exponentially fitted 5 minute heart rate recovery; HR_0, stabilized heart rate following exercise. Values are presented for athletes and non-athletes for inactive recovery in the upright seated position (SeatInact), active recovery in the upright seated position (SeatAct), supine position (Sup), and supine position with elevated legs (SupElev) as means (± SD).* $p < 0.05$, athletes vs. non-athletes; † $p < 0.01$, vs. seated inactive; § $p < 0.01$, vs. seated active.

the seated upright position was associated with slower HRR compared with inactive recovery in the same position. HRR in athletes was accelerated in the supine position with elevated legs and during active recovery in the seated position as compared with non-athletes but showed the same within-subject differences as in non-athletes.

Complex control mechanisms of cardiovascular regulation during rest and recovery include central command, baroreflex control and muscle metaboreflex control (Carter et al., 2003). Different orthostatic conditions during recovery after metabolically demanding exercise may influence the interplay among these control mechanisms and result in different cardiac regulation leading to different HRR patterns.

Our results showed slower HRR in the upright seated position compared with the supine position regardless of physical activity level. Similar results were shown by Takahashi (2000) and Buchheit (2009). Cardiac sympathetic nerve activity decreases and vagus nerve activity increases when the body position changes from upright position to supine (Rowel, 1993). If a similar change in activity of the two components of the autonomic nervous system occurs during the post-exercise period, a faster decrease in HR would be induced in the supine position via predominance of vagus nerve activity, compared with the upright position (Takahashi et al., 2000).

The present study also investigated the difference between active and inactive recovery in the seated positions showing that active recovery is associated with slower heart rate recovery.

Inactive recovery from dynamic exercise is associated with cessation of the primary exercise stimulus from the brain and abrupt changes in the stimuli to baro- and metaboreceptors (Rowel, 1993). O`Leary speculated that the fall in HR during post-exercise was caused by a sudden rise of parasympathetic activity at the cessation of exercise due to a loss of central command (O`Leary, 1993). Slower HR decay might be due to maintained sympathetic outflow evoked by the arterial baroreceptors' response to more vigorous and rapid changes in pressure (Smith et al., 1999). When dynamic exercise is stopped and inactive recovery occurs in the upright position, arterial pressure decreases rapidly (Carter et al., 1999). Due to gravitational forces, blood from the central venous system is shifted to the lower extremities leading to decreased ventricular filling and decreased stroke volume and cardiac output (Rowel, 1993). The initial adjustments to orthostatic stress are mediated exclusively by neural pathways of the autonomic nervous system (Smith et al., 1999).To preserve blood pressure and prevent syncope, an increase in sympathetic-mediated vasomotor activity is elicited, at the same time preventing fast HR decay (Buchheit et al., 2009; Hainsworth, 2000). Suggestive evidence was provided by Levine and colleagues that highly fit endurance trained athletes may have diminished tolerance to orthostatic stress compared with moderately fit or sedentary controls (Levine et al., 1991). Throughout our investigation orthostatic hypotension was observed in three athletes by the end of the fourth minute of recovery and none of the non-athletes. They were immediately put into supine position and their heart rate returned to normal values. Lying supine during recovery after exercise may be an effective means of transiently restoring HR and vagal modulation and a safe position for prevention of syncope (Buchheit et al., 2009).

Maintained higher levels of HR_0 in both seated positions after the initial reflex adjustments might result from postural muscle tonus eliciting sympathetic reflex influence on the circulation.

During active recovery, the muscle pump facilitates the return of blood back to the heart preventing pooling in the lower extremities and the need for sympathetic-mediated vasoconstrictive activity, allowing a drop in post-exercise HR. On the other hand, central command from higher brain centres and inputs from mechano- and chemoreceptors in contracting muscles contribute to maintaining sympathetic activity (though to a lesser extent than during exercise) preventing faster HR decay (Carter et al., 1999). As during moderate exercise when HR is increased due to parasympathetic withdrawal (Rowel, 1993), with active recovery HR might be maintained above resting values for the same reason. This parasympathetic withdrawal is achieved by inhibitory central command influences (Takahashi and Miyamoto, 1998).

Even though HRR is slower with active recovery, lactate elimination after high intensity exercise might be more important for athletes than the decline of heart rate. Present knowledge supports the superiority of active recovery over passive recovery for lactate removal from the circulation (Coffey et al., 2004; Monerdo and Donne, 2000). Throughout active recovery an increased blood flow to the working muscle is believed to enhance the removal of lactic acid from the exercising muscle cells (Ahmaidi et al., 1996; Bangsbo et al., 1994). The reduction in blood lactate concentration is likely to be associated with enhanced lactate clearance by increasing metabolic rate and systemic blood flow, thereby accelerating lactate metabolism via oxidation and gluconeogenesis (Martin et al., 1998). Accelerating blood lactate clearance immediately post-exercise may be beneficial for successive bouts of high-intensity exercise, particularly during

athletic competitions that require multiple performances in a single day (Martin et al., 1998).

The initial difference in HRR between active and inactive recovery diminished when HR reached asymptotic values (i.e. no difference in HR_0) in athletes, but lower HR_0 was observed in non-athletes following active recovery. This might suggest that the effects of abrupt exercise cessation may have larger impact on autonomic nervous system perturbations.

In the supine position with elevated legs, blood is redistributed towards the heart increasing preload (Takahashi et al., 2000). The increased central blood volume via arterial baroreceptors imposes a greater vagal activation and reduces heart rate and cardiac output (Rowell, 1993). Whether the redistribution of blood in the supine position with elevated legs is sufficient enough to evoke greater perturbations in the cardiac autonomic regulation than in a simple supine position is not proved conclusively. Buchheit explains the lack of difference between the two supine positions with the presence of minimal sympathetically mediated vasomotor activity masking any potential baroreflex-mediated increase in parasympathetic activity resulting from increased venous return in the supine position with elevated legs (Buchheit et al., 2009).

The difference in HRR between the two supine positions diminished after HR stabilized. As the baroreceptors involved in this reflex regulation are effective only at buffering short term changes, major perturbations in autonomic nervous system activity would be evident only immediately after exercise cessation (Rowell, 1993).

Previous research has suggested that highly fit individuals present more effective autonomic activity than sedentary ones (Aubert et al., 2003; Buch et al., 2002; Carnethon et al., 2005; Carter et al., 2003; Darr et al., 1988; Imai et al., 1994). Studies suggest that not only athletes participating in endurance (Hagberg et al., 1980; Melanson and Freedson, 2001; Tulppo et al., 2003; Yamamoto et al., 2001), but also in strength exercise training (Otsuki et al., 2007) demonstrate accelerated HRR. Darr found that post-exercise HRR is primarily dependent on the cardiorespiratory fitness level of the individual and the intensity of exercise (Darr et al., 1988). A longitudinal study of physical activity and HRR failed to show any significant connection between post-exercise HR response and VO2max (Carnethon et al., 2005). Similarly, Buchheit also found no relationship between VO_2 and HRR indices but suggested that post-exercise HRR was rather associated with training loads (Buchheit and Gindre, 2006).

In our study we found larger HRR_{60} in the group of athletes for all four recovery conditions but revealed shorter recovery time constant only for active recovery in the seated position and the supine position with elevated legs.

Our study found no difference in the time constant of HR decay during inactive recovery in the upright supine position between athletes and non-athletes. On the basis of evidence that trained athletes possess relatively poor orthostatic tolerance and sub-optimal reflex ability to maintain arterial blood pressure we expected faster HRR in athletes (Levine et al., 1991; O`Sullivan and Bell, 2000). On the other hand active recovery led to faster HR decay in athletes. This might be due to training related diminished cardiopulmonary cardioacceleration (Mack et al., 1993). It seems that exercise training induced hypervolemia results in an attenuation of the cardiopulmonary baroreflex through increased tonic inhibition of cardiac afferent nerve activity (i.e. activation of cardiopulmonary mechanoreceptors) (Mack et al., 1993).

To explain faster HRR in athletes in the supine position with elevated legs the adaptation in baroreflex function should be considered (Yamamoto et al., 2001). Results of cross sectional studies comparing athletes with sedentary individuals show that athletes have higher baroreflex sensitivity and HRV than sedentary subjects (Aubert et al., 2003). Longitudinal studies in healthy subjects also demonstrated increase in HRV and/or baroreflex sensitivity with training (Melanson and Freedson, 2001; Yamamoto et al., 2001). Larger blood volume and stroke volume in athletes also maintain higher activation of arterial baroreceptors leading to increased parasympathetic activation (Boushel et al., 2000).

Values of HR did not reach baseline values after five minutes of recovery. Metaboreceptors with sympathetic afferents might be activated by accumulated waste products during exercise (O`Leary, 1993) possibly suggesting an alternative mechanism for sympathetic predominance and elevated HR levels in all four recovery protocols. Endurance training induces a lower level of muscle chemoreflex stimulation as a result of the increased oxidative capacity of the exercising muscle (Yamamoto et al., 2001). Reduced sympathetic influence on the heart activity in athletes is due in part to diminished afferent signals from muscle metaboreceptors (Boushel et al., 2000) resulting in lower HR_0 values in athletes compared with non-athletes.

Further investigation is needed to thoroughly study the mechanisms of HRR that lead to differences emerging from body positions and training level. Specific training modalities might also influence HRR and different sport specialities might show even greater differences in heart rate recovery after exercise. We investigated HRR after submaximal exercise, but it depends in great deal on the preceding exercise intensity as well. The results gained from this study might be of interest to clinicians wishing to prescribe exercise to clinical populations or fitness experts dealing with sedentary population in their everyday work. They might find supine position to be an optimal mode of recovery for patients with slower HRR or pre-syncope. Even though HRR is slower with active recovery, from a metabolic point of view (i.e. lactate elimination) it might be the optimal mode of recovery for athletes.

Conclusion

In conclusion, results from the present study suggest that the supine position with or without elevated legs accelerated HRR more than the two seated conditions. Active recovery in the seated upright position was associated with slower HRR compared with inactive recovery in the same position. HRR in athletes was accelerated in the supine position with elevated legs and during active recovery in the seated position as compared with non-

athletes but showed the same within-subject differences as non-athletes.

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Key points

- In order to return to a pre-exercise value following exercise, heart rate (HR) is mediated by changes in the autonomic nervous system but the underlying mechanisms governing these changes are not well understood.
- Even though HRR is slower with active recovery, lactate elimination after high intensity exercise might be more important for athletes than the decline of heart rate.
- Lying supine during recovery after exercise may be an effective means of transiently restoring HR and vagal modulation and a safe position for prevention of syncope.

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