ORIGINAL RESEARCH

Effects of Sitting Recovery Protocols on Postexercise Heart Rate in Young Adults

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ABSTRACT

Background: We aimed to compare the effects of 3 different upright seated recovery protocols on heart rate recovery (HRR) after a submaximal ergometer exercise.

Methods: The study included 30 young adult volunteers (15 men and 15 women). Participants performed a submaximal cycle exercise test at a constant workload of 60 Watts until a steady HR was achieved. This was followed by 5 min of: (a) inactive, (b) active loadless, and (c) passive recovery protocols. The HRR was assessed as the difference between the peak exercise HR and the HR recorded following 1 min of recovery and as the percentage HR decline after 1 min postexercise. Abnormal HRR was defined as a reduction of 1-min HRR ≤12 b · min^{-1} or ≤15 b · min^{-1} or ≤18 b · min^{-1}.

Results: In both sexes, HRR was faster during inactive recovery compared with active recovery. In males, HRR was faster during inactive recovery compared with passive recovery protocol. In females, HRR was faster during passive recovery compared with active recovery protocol. The prevalence of impaired HRR was greater in the active recovery compared with the passive and inactive recovery protocols in both sexes.

Conclusion: The present findings suggest that in both sexes, the postexercise HRR was mediated by a combined action of the central command and other inputs or stimuli arising from skeletal muscle activities. In addition, the active recovery protocol resulted to a slower HRR and elicited more abnormal postexercise HR responses compared with the other recovery protocols.

Keywords: heart rate recovery, active recovery, inactive recovery, passive recovery, central command

INTRODUCTION

One of the immediate responses of the cardiovascular system to exercise is increase in heart rate (HR) (1). The increase in HR is aimed at increasing cardiac output, thus satisfying the energy needs of the working muscles. At the end of the exercise, HR declines exponentially at the early minutes of recovery and further declines gradually, but progressively toward resting levels (2). Both the rise and fall of HR during and after exercise are respectively modulated by the autonomic nervous system. The model of autonomic control of HR in humans during dynamic exercise indicates that the rise in HR is largely because of rapid withdrawal of parasympathetic activity as well as increase in sympathetic tone (3). The initial exponential drop in HR is a result of rapid restoration of vagal tone after cessation of exercise (4), whereas the further decrease in HR is attributed to the progressive weakening of the sympathetic influence (5). The physiological implication of this response makes HR recovery kinetics a convenient tool for both clinicians (e.g., clinical exercise physiologists) and sports scientists. Heart rate recovery (HRR) has been identified as a powerful and independent predictor of cardiovascular and all-cause mortality in healthy adults (6–8) and in those with cardiovascular diseases (9). It is also commonly used as an indicator of cardiovascular fitness and as an independent predictor of endothelial dysfunction (10). A delayed HRR has also been reported to be a measure of autonomic dysfunction (11).

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Generally, it is established that postexercise HRR is affected by body postures (12). Some of the previous studies compared mostly the effects of sitting, standing, and supine positions on HRR and indicated that supine position caused a more accelerated HRR compared with either sitting or standing positions (12,13). However, little is known about the effects of different upright sitting recovery protocols on HRR. In particular, no previous study to the best of our knowledge has compared the effect of seated inactive recovery on postexercise HR with that of passive recovery mode. Previous studies on the effects of posture on postexercise HR responses have mostly made use of maximal exercise tests with less attention given to submaximal tests. Similarly, to the best of our knowledge, the prevalence of impaired HRR following submaximal exercise levels in young adults has not been investigated. Interestingly, submaximal exercise testing overcomes many of the limitations of maximal exercise testing and is considered the method of choice for the majority of individuals seen by physical therapists, such as those limited physically by pain and fatigue or have abnormal gait or impaired balance.

It is also noteworthy that, whereas a majority of previous studies (6,7,9,14–16) evaluating HRR as a prognostic tool for cardiovascular events employed the use of treadmill testing as the exercise modality, a limited number of studies assessed the prognostic importance of HR recovery using the cycle ergometer exercise testing (8). Furthermore, comparative studies on the clinical importance of seated recovery ergometer cycle protocols on postexercise HR outcomes are lacking. Bicycle ergometer exercise testing, one of the most common and most economical exercise methods used in eliciting cardiovascular responses, requires that the subject be seated in the upright position. During postexercise recovery, while sitting on the bicycle ergometer, 3 different recovery protocols can be employed: (a) inactive recovery, in which the subject stops exercise and sits completely still; (b) active loadless recovery, during which the subject pedals against zero resistance following exercise; and (c) passive recovery, during which the subjects' legs are passively pedaled for them on a tandem bicycle at the same rate as during active recovery (17).

The present study is designed to fill in the gaps of the lack of information on the effects of upright sitting protocols on postexercise HR responses after a submaximal cycle ergometer exercise test. We also sought to determine if different seated recovery protocols after a submaximal exercise may influence the interplay among the control mechanisms and result in different cardiac regulation leading to different HRR patterns. Furthermore, it is believed that the present study will enable clinical exercise physiologists to recommend the best mode of seated recovery protocol suitable for both asymptomatic individuals and those with cardiovascular problems. It will also assist in determining the best sitting recovery protocol that can elicit abnormal postexercise responses in an apparently healthy, noncardiopathic, young adult population compared with other recovery protocols.

Our aim was to compare the effects of the 3 different upright sitting recovery protocols (inactive, active loadless, and passive) on HRR after a submaximal ergometer exercise test. Furthermore, we examined the clinical importance of the 3 sitting recovery protocols with regard to revealing the presence of abnormal HRR responses in an apparently healthy, noncardiopathic, young adult population.

METHODOLOGY

The study included 30 apparently healthy young adult volunteers (15 men and 15 women; between 18 and 27 years of age), selected from the College of Health Sciences, Nnamdi Azikiwe University, Nnewi campus. All participants selected were nonathletic but physically active since they occasionally participated in recreational activities such as soccer, table tennis, lawn tennis, badminton, and basketball. A structured preexercise health and lifestyle screening questionnaire was administered to the participants prior to the exercise tests. Physical examinations and morphometric measurements were performed by experienced personnel. The participants' ability to complete a submaximal cycle ergometer exercise test at intensity of 60% to 85% of predicted maximum HR was considered as criteria for inclusion in the study. Exclusion criteria were self-reported presence of ill health; hypertension (systolic blood pressure [BP] $\geq$ 140 mm Hg and/or diastolic BP $\geq$ 90 mm Hg); history of unstable cardiovascular or respiratory disease; a malignancy, musculoskeletal lesions, a history of smoking or alcoholism, obesity (body mass index $\geq 30\ kg \cdot m^{-2}$), diabetes, and taking medication affecting cardiovascular function. Participants were informed (written and oral) of the experimental procedures, and their consents were obtained before participation. The experiments and Ethics Committee of the Faculty of Basic Medical Sciences of the University approved the study. The investigation conforms to the principles outlined in the Declaration of Helsinki.

Experimental Design

Testing was performed between 7:00 am and noon of each day in a well-ventilated room of 27°C to 29°C temperature. Participants were instructed not to consume beverages containing alcohol or caffeine, not to eat a heavy meal, and not to participate in any vigorous physical activity 24 h before the test.

Participants were asked to visit the exercise laboratory on 4 occasions. On the first day, they were properly instructed on how to perform the bicycle ergometer exercise test with demonstration. In addition, the participants' demographic and baseline characteristics were measured and their capacity to complete a submaximal exercise test at intensity of 60% to 85% of predicted maximum HR was also determined. During the subsequent 3 visits, the participants repeatedly performed an exercise protocol that consisted of a 1-min warm-up period at a work rate of 60 rpm at 0 resistance followed by 5 min of increased workload to elicit 60% to 85% of their age-predicted maximum HR (HRmax) at a constant work rate of 60 rpm at a resistance of 1 kp (≈60 Watts) until

a steady-state HR is achieved. HR was considered steady state when consecutive HRs between the last 3 minutes were within 6 b·min^{-1} of each other (18). A metronome was used to help participants maintain a constant pedal rate of 60 rpm.

A 5-min recovery period followed the termination of the exercise, with the participants maintaining an upright sitting position on the ergometer bicycle and performing 3 different recovery protocols: (a) inactive seated, (b) active loadless pedaling on the standard ergometer, and (c) passive cycling with the pedal rate maintained constant (60 rpm) by a tandem partner. The recovery protocols were performed in random order on separate days, and in each case recovery was studied for 5 min. During the passive recovery protocol, the participant's feet were secured to the pedals of the cycle ergometer and their legs fully relaxed so as to neither contribute to nor resist the pedal rotation.

To identify the mechanisms (mechanical and neural), which mediate the postexercise responses between recovery protocols in the present study, we employed a reductionist approach which has been used previously to assess the postexercise HRR (17). The unloaded active recovery test involves the mechanical mechanisms (skeletal muscle pumps), which force blood toward the heart, and neural mechanisms (central neural command and the mechanoreceptors), which regulate vagal tone and flow to meet the metabolic requirement of exercising muscles. The passive exercise is an involuntary physical activity that engages both the skeletal muscle pumps and the mechanoreceptors without the concomitant participation of central neural command or metaboreceptors (17). Because loadless active exercise involves central command, the passive pedaling mode was performed to serve as a control for this central command effect. The principal difference between active and passive recovery is the presence of central command during recovery (17). The inactive seated recovery protocol did not engage the skeletal muscle pump, thus the principal difference between inactive and passive recovery is the ongoing skeletal muscle pumping and mechanoreflex activity during passive recovery (17).

Anthropometric Measurements

Participants' heights were measured to the nearest 0.1 cm with a stadiometer (SECA, Hamburg, Germany). Weight was measured to the nearest 0.1 kg in light clothing without shoes using a standard scale. Body mass index was calculated as weight (kg) divided by height squared (m^2).

Blood Pressure Measurement

Resting BP was measured in a seated position and in a quiet room, using both cuff-stethoscope and electronic methods after 10 and 15 min of rest, respectively. There were no significant differences observed between the mean values of the 2 methods. The resting BP measurement was used to ascertain whether a participant was hypertensive or not. Those who were hypertensive (systolic/diastolic BP >140/90 mm Hg) were excluded from the study.

HR Measurement

The resting HR was measured twice on the first day of the participant's visit, using the Omron electronic monitor (Omron Health Care Inc., Vernon Hills, Illinois). The mean of the 2 values was used as the resting HR data. During subsequent visits prior to the exercise test, the HR measurement was repeated twice to obtain the preexercise HR. During exercise, HR was measured at 2-min intervals using the electronic monitor until a steady-state HR was achieved. The peak HR was recorded during the final minute of the exercise test after the steady-state has been achieved. At postexercise, HR was first measured at 1 min of recovery and subsequently at every 2-min interval until the 5th minute. The percentage maximum HR attained by subjects was expressed as ([peak HR/HRmax] × 100) (19). The age-predicted HRmax was determined as: HRmax = 208 − (0.7 × age), (20). Variables of HR recovery after exercise included absolute HR recovery and percentage HR decline at 1st min of HR recovery (19). Absolute or change in HRR from peak exercise (ΔHRR) was calculated as peak HR − 1st min postexercise HR; percentage HR decline after exercise was calculated as ([peak HR − 1st min postexercise HR]/[peak HR]) × 100 (21). An abnormal HRR was defined as a reduction of 1-minute ΔHRR ≤ 12 b · min^{-1} (14) or ≤15 b · min^{-1} (14,15) or ≤18 b · min^{-1} (16).

Determination of Submaximal Oxygen Uptake (Vo_2)

Peak submaximal exercise oxygen uptake (L · min^{-1}) was determined using the equation: 85.447 + 9.104 × sex (0 = women; 1 = men) − 0.2676 × age (years) − 0.415 × body mass (kg) + 0.1317 × power output (W) − 0.1615 × peak (steady-state) HR (22). The absolute Vo_2 values (L · min^{-1}) were converted to relative values (mL · kg^{-1} · min^{-1}) using the formula: (absolute Vo_2 (L · min^{-1}) × 1000)/body weight (kg).

Data Analysis

Descriptive data were expressed as mean and standard deviation for continuous variables and percentages for categorical data. Comparative analysis between 2 groups was done using independent sample *t*-test, while that of multiple groups was done using one-way analysis of variance involving the Bonferroni post hoc multiple comparison test. Statistical significance was set at $P < 0.05$. All statistics were performed using SPSS for windows (version 20.0, Chicago, Illinois).

RESULTS

The demographic and baseline characteristics of the study population are shown in Table 1. Data indicated a sex-matched young adult population. Independent sample *t* test shows that the males indicated higher mean age ($P < 0.001$) and height ($P < 0.001$) and lower resting diastolic BP ($P < 0.05$) compared with the females. No significant differences were observed in body weight, body mass index, resting systolic BP and resting HR between the 2 sexes.

Table 2 shows the exercise and postexercise test characteristics of the study population according to the 3 different

TABLE 1. Demographic and baseline characteristics of the study population.

Variables	Males (n = 15)	Females (n = 15)	*P* Value
Age, y	23.9 ± 2.0	20.5 ± 2.1	<0.001
Height, m	1.77 ± 0.05	1.64 ± 0.07	<0.001
Weight, kg	68.7 ± 10.8	60.3 ± 12.2	0.057
BMI, $kg \cdot m^{-1}$	21.8 ± 3.4	22.3 ± 3.6	0.738
Resting SBP, mm Hg	115 ± 10	111 ± 12	0.286
Resting DBP, mm Hg	67 ± 7	72 ± 7	0.049
Resting HR, $b \cdot min^{-1}$	77 ± 10	82 ± 8	0.165

BMI = body mass index; DBP = diastolic blood pressure; HR = heart rate; SBP = systolic blood pressure

upright sitting recovery protocols. Analysis of variance (ANOVA) indicated no significant differences in preexercise HR, exercise HR_{2min}, exercise HR_{4min}, peak exercise HR, attained %HRmax, peak exercise absolute and relative Vo_2. It is noteworthy that the peak exercise Vo_2 ($mL \cdot kg^{-1} \cdot min^{-1}$) and attained %HRmax data indicated that participants were moderately active and able to perform the exercise test at an intensity of 60% to 85% of their age-predicted maximum HR. A post-hoc analysis further revealed that mean 1st min and 3rd min postexercise HRs were significantly higher ($P < 0.05$ or $P < 0.001$) during active recovery compared with inactive recovery in both genders. These parameters also indicated significantly higher ($P < 0.01$) values during passive recovery compared with inactive recovery in males, but not in females. Active recovery protocol indicated significantly ($P < 0.01$) greater 1st min and 3rd min postexercise HRs compared with the passive recovery protocol in females but not in males.

In both males and females, $\%HRD_{1min}$ indicated significantly ($P < 0.001$) higher values in inactive compared with active recovery protocol (Figure 1). $\%HRD_{1min}$ values were also significantly ($P < 0.01$) higher in inactive vs. passive protocol in males, but no significant difference was found between the 2 protocols in females. In females, $\%HRD_{1min}$ values were higher in passive compared with active protocol, but no significant difference was observed between the 2 protocols in males.

In males, ΔHRR_{1min} values were significantly ($P < 0.01$) higher in inactive vs. passive protocol, but in females, no significant differences were found between the 2 recovery protocols (Figure 2). In both sexes, ΔHRR_{1min}, indicated significantly ($P < 0.001$) higher values in inactive compared with active recovery protocol. ΔHRR_{1min} value was higher in passive compared with active protocol in females ($P = 0.001$), but no significant difference was observed between the 2 protocols in males ($P = 0.419$). Throughout the 3rd min of recovery, HR decreased more during the inactive compared with passive ($P < 0.05$) and active loadless recovery ($P < 0.001$) in both sexes. ΔHRR_{3min} value was significantly greater in passive condition compared with active protocol in females ($P = 0.004$), but not in males ($P = 0.215$).

The prevalence of impaired HRR according to different HRR cut-offs in males are shown in Figure 3. Data indicated higher prevalence of impaired HRR in the active recovery (73.3%) compared with the passive (60%) and inactive (7%) recovery protocols in the $\leq 12\ b \cdot min^{-1}$ cut-off. The same trend was observed in the $\leq 15\ b \cdot min^{-1}$ cut-off (73% vs. 67% vs. 20%) and ≤18 cut-off (93% vs. 73% vs. 20%) respectively. Similarly, in females, results indicated higher prevalence of impaired HRR in the active recovery (80%) compared with the passive (13%) and inactive (7%) recovery protocols in the $\leq 12\ b \cdot min^{-1}$ cut-off. The same trend was observed in the

TABLE 2. Exercise and postexercise test characteristics of participants according to the 3 different upright sitting recovery protocols.

Variables	Males (n = 15)			Females (n = 15)		
	Inactive	Passive	Active	Inactive	Passive	Active
Preex HR, $b \cdot min^{-1}$	76 ± 8	76 ± 9	75 ± 7	82 ± 8	81 ± 7	81 ± 7
Ex HR_{2min}, $b \cdot min^{-1}$	96 ± 10	98 ± 13	101 ± 19	96 ± 12	95 ± 12	95 ± 12
Ex HR_{4min}, $b \cdot min^{-1}$	104 ± 14	107 ± 14	109 ± 15	102 ± 11	101 ± 12	101 ± 12
Peak Ex HR, $b \cdot min^{-1}$	130 ± 9	129 ± 10	126 ± 7	132 ± 15	126 ± 10	132 ± 14
Attained %HRmax	68 ± 5	67 ± 5	66 ± 4	68 ± 7	65 ± 5	68 ± 7
Peak Ex Abs Vo_2, $L \cdot min^{-1}$	3.15 ± 0.18	3.15 ± 0.17	3.19 ± 0.18	2.43 ± 0.20	2.49 ± 0.17	2.44 ± 0.21
Peak Ex Rel Vo_2, $mL \cdot kg^{-1} \cdot min^{-1}$	46.6 ± 5.1	46.7 ± 5.8	47.1 ± 5.1	41.5 ± 6.2	42.5 ± 6.2	41.6 ± 5.9
Postex HR_{1min}, $b \cdot min^{-1}$	104 ± 7	113 ± 8[a]	116 ± 7[a]	111 ± 12	108 ± 9[b]	123 ± 11 [a]
Postex HR_{3min}, $b \cdot min^{-1}$	93 ± 11	104 ± 10[a]	109 ± 6[a]	100 ± 9	100 ± 8[b]	113 ± 12 [a]

%HRmax = percentage of age-predicted maximum heart rate; Abs Vo_2 = absolute oxygen consumption; Ex = exercise; HR = heart rate; Postex = postexercise; Preex = preexercise; Rel Vo_2 = relative oxygen consumption
[a]significant difference between inactive vs. passive or active recovery protocols
[b]significant difference between active vs. passive recovery protocols

≤15 b·min^{-1} cut-off (93% vs. 33% vs. 13%) and ≤18 cut-off (93% vs. 40% vs. 40%) respectively (Figure 4).

DISCUSSION

This study aimed at explaining differences in the effects of 3 upright sitting recovery protocols on postexercise HRR after repeated submaximal cycle ergometer exercise tests of matched exercise intensity and peak Vo_2 by young adult participants.

We observed that the rate of HRR in the inactive recovery protocol was greater compared with the unloaded active condition. This agrees with a previous study that indicated that inactive recovery in the seated upright position was associated with faster HRR compared with the active recovery in the same position (13). The greater decline in HR in seated inactive recovery protocol may be because of a greater rise of parasympathetic activity compared with the seated active recovery, which is attributable to the loss of central command and disengagement of the skeletal muscle contraction during inactive recovery. Interestingly, our data shows that the change in HR during inactive recovery was significantly greater than the unloaded active recovery protocols, which is an indication of the presence of central command, mechanoreflex, and skeletal muscle activities during active recovery. Inactive recovery from dynamic exercise is associated with the cessation of the primary exercise stimulus from the brain (central command) as well as the disengagement of the skeletal muscle activity and abrupt withdrawal of the stimuli arising from muscle metaboreceptors and mechanoreceptors (17). The loss of central command and the resultant decrease in stimuli to the mechanoreceptors and metaboreceptors during inactive recovery will lead to a reduction in sympathetic nerve activity and an increase in parasympathetic tone, thus promoting a rapid decrease in HR in the early minutes of recovery (17,23). On the other hand, during the unloaded active recovery, there is a maintenance of some degree of activities of the central neural command, mechanoreceptors, as well as the skeletal muscle pump activities. The central command activities as well as inputs from the mechanoreceptors and chemoreceptors in the contracting muscles during the active-loadless recovery, would contribute to the maintenance of sympathetic activity, thereby increasing postexercise HR and slowing down HR recovery (17). Furthermore, the skeletal muscle pump activities during the active recovery would facilitate the return of blood back to the heart and prevent the pooling of blood in the lower extremities, thereby promoting the need for sympathetic-mediated vasoconstrictive activity, thus slowing down the postexercise HR recovery (13).

FIGURE 1. The rate of heart rate decline for the participants according to the 3 different upright sitting recovery protocols. * = significant difference between inactive vs. passive or active recovery protocols; † = significant difference between active vs. passive recovery protocols; HR = heart rate.

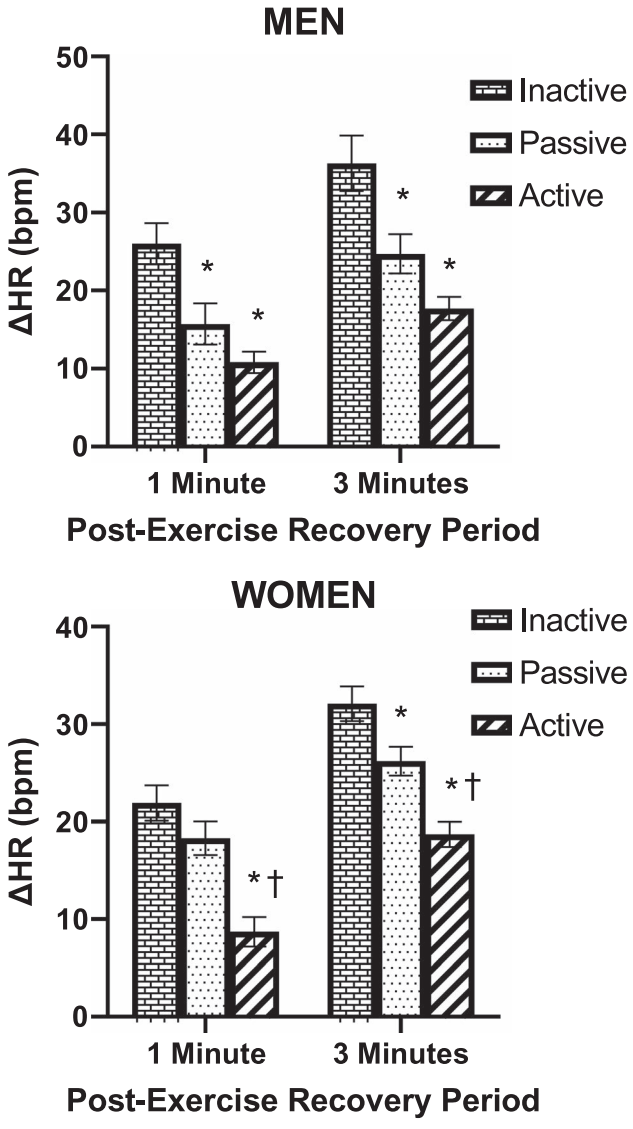

FIGURE 2. Changes in heart rate during recovery from peak exercise according to the 3 different upright sitting recovery protocols. * = significant difference between inactive vs. passive or active recovery protocols; † = significant difference between active vs. passive recovery protocols; bpm = beats per minute; HR = heart rate.

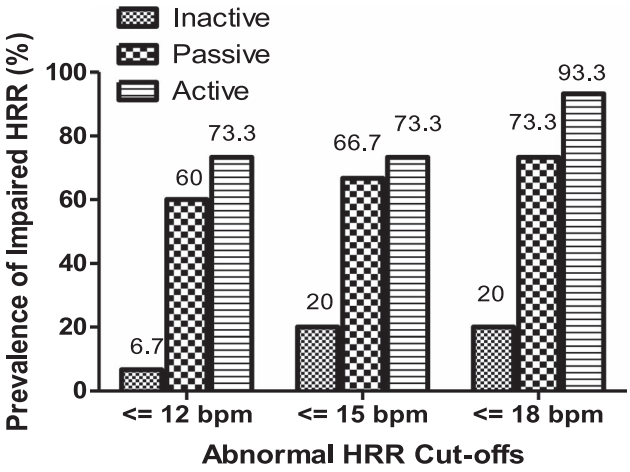

FIGURE 3. The prevalence of impaired HRR in males according to different HRR cut-offs. HR = heart rate; HRR = heart rate recovery.

Our finding in which HR decreased less (slower HRR) during the active loadless recovery than during the passive pedaling in females is in accordance with a previous study (17). It is noteworthy that both recovery protocols involve the engagement of the skeletal muscle pumping and mechanoreflexes during recovery. Therefore, we attributed any difference in response between the active and passive recovery to the influence of the central command. The slower decline in HR during active recovery from exercise indicates that the presence of central command contributed to the maintenance of HR increase in this recovery mode. The faster decline in postexercise HR during passive recovery protocol is attributed to a greater parasympathetic influence caused by the cessation of exercise and the loss of central command associated with passive recovery (17,24). The mechanism through which central command affects HRR has already been discussed above.

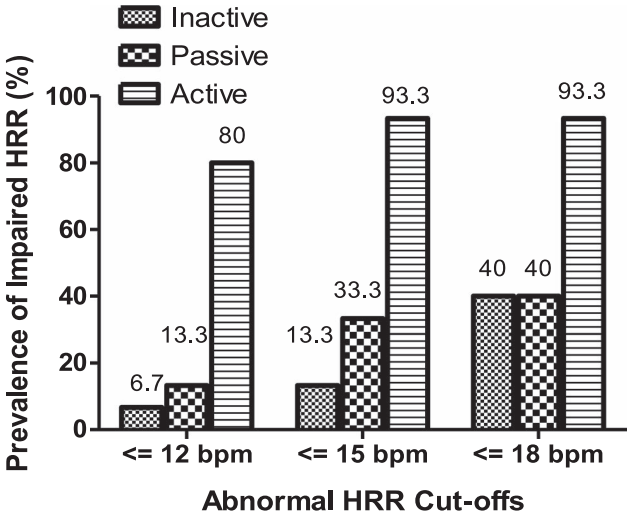

FIGURE 4. The prevalence of impaired HRR in females according to different HRR cut-offs. HR = heart rate; HRR = heart rate recovery.

To the best of our knowledge, no previous study has compared the effect of seated inactive recovery protocol on postexercise HR with that of passive recovery. Passive pedaling is usually employed as a recovery mode to examine the role of the skeletal muscle pump and mechanoreceptors during exercise recovery without the concomitant participation of central command (17). A common characteristic shared by both inactive and passive recovery protocols is the absence of the central command activities. However, skeletal muscle pumping and mechanoreflex activities are stopped during inactive recovery from exercise, while passive pedaling engages the skeletal muscle pumps and mechanoreceptors during recovery. The principal difference between inactive and passive recovery is therefore attributed to the ongoing skeletal muscle pumping and mechanoreflexes during passive recovery (17). The skeletal muscle pump is an important mechanism that facilitates venous blood flow to the heart and also is crucial in coordinating the local and systemic blood flow responses during dynamic exercise (25). In the present study, HRR was faster in inactive recovery compared with passive protocol in males. In females, the rate of deceleration of the HR also appeared greater in the inactive recovery protocol compared to the passive protocols (though the statistical difference was not significant).

The slower HRR observed in the passive recovery compared to inactive recovery in males is thought to be because of slower reactivation of the parasympathetic nerve activity and delayed withdrawal of the sympathetic nerve activity resulting from an ongoing mechanoreceptor activity stimulated by the skeletal muscle pumping activities during the passive leg movement. These factors in turn may have caused a reflex increase in HR that slowed down recovery at postexercise. Furthermore, contributions from metaboreceptors, sensitive to passive movement during the postexercise HRR should not be neglected. Although the muscles are not being deliberately exercised, in any muscle possessing tone, passive movements with stretching and postural changes of muscle during leg rotation are necessarily associated with work done by the myofilaments and therefore with changes in metabolism (26). The increase in metabolism in contracting muscles may have also engendered a large reflex increase in sympathetic activity via the metaboreceptor reflexes. On the other hand, inactive recovery is characterized by cessation of the primary exercise stimulus from the brain and loss of both central command and skeletal muscle pump influences, hence the faster fall in HR caused by a sudden rise of parasympathetic activity and a gradual withdrawal of the sympathetic nervous activity at the cessation of exercise and onset of recovery.

Although our findings suggest that both inactive and passive sitting positions during recovery appeared to be better protocols for a more rapid and transient restoration of HR and vagal modulation after ergometer exercise, this may not necessarily translate into better cardiometabolic performance among exercisers compared with the active recovery protocol. A previous finding has suggested that recovery of HR does not necessarily indicate recovery of metabolic systems for performance (27). Other studies supported this fact

by demonstrating the superiority of active recovery over passive recovery for lactate removal from the circulation (13,28). Lactate elimination after high intensity exercise might be more important especially for athletes than the decline of HR particularly during successive bouts of high-intensity exercise, and during athletic competitions that require multiple performances in a single day (13).

Comparative studies are lacking on the clinical outcomes of using different seated recovery protocols (inactive, active-loadless, passive) to evaluate postexercise HRR in apparently healthy young adults. It is not clear which of the 3 protocols elicits more of the presence of abnormal HRR response than the others in an apparently noncardiopathic, young adult population. In this study, we defined abnormal HRR using 3 different previously established cut-offs for HRR_{1min}: ≤ 12 b · min^{-1} (14), ≤ 15 b · min^{-1} (14,15) and ≤ 18 b · min^{-1} (16). The reason for our choice of different cut-offs was mainly to observe the trend of abnormal HRR prevalence across the different cut-offs, since there is the absence of a uniform recovery protocol as well as a universally accepted cut-off for abnormal HRR. Our data indicated that, irrespective of sex, active recovery elicited a greater prevalence of abnormal or blunted postexercise HRR compared with the passive and active recovery protocols. It is noteworthy that most of the individuals, who initially presented with normal HRR under the influence of inactive and passive recovery protocols, later indicated blunted HRR under the influence of the active recovery protocol in both genders. Our findings therefore suggest that compared with other seated recovery protocols, active recovery protocol appears to be a better option that may be used in clinical evaluation of HRR as a prognostic tool for diagnosing cardiovascular diseases and cardiovascular mortality.

Limitations of this study include lack of direct determination of the mechanisms that mediate the postexercise HR responses between the recovery protocols. For example, we could not confirm the direct leg muscle activation during passive exercise pedaling using electromyography. This confirmation would have given the confidence that no central command signals existed during passive pedaling. Also, we could not investigate the influence of other mechanisms such as metaboreflexes and respiratory pumps as this would have also ruled out their influences on the increased HR responses during unloaded active recovery. This is particularly important for the metaboreflex mechanism, since in all forms of movements, whether active or passive, metaboreceptor reflexes may be activated to some extent. We also did not measure oxygen consumption during exercise recovery. Previous studies have shown that whole body and muscle oxygen consumption are elevated during active recovery compared with inactive recovery (29).

CONCLUSION

This study indicates that the active recovery protocol had a slower rate of HRR compared with inactive-loadless and passive recovery protocols. Furthermore, active recovery elicited a greater prevalence of abnormal or blunted postexercise HRR compared with the passive and active recovery protocols. These findings suggest that the inactive and passive sitting positions during recovery appeared to be better protocols of choice for a more rapid and transient restoration of HR and vagal modulation after ergometer exercise, especially among nonathletes and individuals with blunted HR recovery. However, from a clinical point of view, active recovery appears to be the optimal mode of recovery that can be used to elicit the presence of abnormal postexercise HR responses in apparently healthy individuals, compared with other sitting recovery protocols.

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