The Effects of a Delay Following Warm-up on the Heart Rate Response to Sudden Strenuous Exercise

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ABSTRACT

Introduction: Sudden strenuous exercise (SSE) has previously been shown to result in electrocardiograph (ECG) abnormalities indicative of myocardial ischemia when not preceded by a warm-up. Athletes regularly undergo SSE and are often unable to warm-up immediately prior to competition. It is unknown whether a delay post warm-up will result in the same heart rate (HR) response to SSE as seen with no warm-up.
Aims: To compare the HR response and to observe for ECG abnormalities during SSE with a warm up, with a delay after warm up and without a warm up.
Methods: Seven male subjects randomly completed three SSE exercise conditions while being monitored by ECG; a 15 second supramaximal sprint following three conditions: no warm up (NW); immediately following a warm-up (WU); and following a 10-minute delay post warm up (D). There were no ECG abnormalities across any of the conditions indicative of myocardial ischemia.
Results: Significant differences ($p<0.05$) were found in the HR response for all time periods during exercise between WU and NW, between WU and D, but not between D and NW. A delay between warm-up and SSE resulted in a lowered HR response to the SSE compared with a warm-up immediately preceding, but a higher HR response to SSE with no warm-up.
Conclusion: The findings of this study suggest that a 10 minute delay following warm-up before SSE is too long to maintain the benefits of warm-up.

Keywords: warm-up, heart rate, ischemia, exercise, sports, vigorous intensity

1. INTRODUCTION

The cardiovascular responses to the initial onset of exercise has been well documented. These include increased cardiac output [1], stroke volume as a result of increased venous return [2], and increased heart rate (HR) due to sympathetic stimulation and circulating

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catecholamines [3]. It is commonly accepted that the cardiac responses are adequate to meet metabolic oxygen demand during submaximal aerobic exercise in healthy individuals when intensity is increased in a graded fashion. However, when very high-intensity exercise is initiated suddenly (sudden strenuous exercise, SSE), oxygen delivery is inadequate to meet metabolic demand leading to anaerobic energy metabolism and oxygen debt, a normal process familiar to any athletic individual accustomed to high-intensity exercise. This scenario may have significant relevance to the myocardium in which demand for oxygenated blood may surpass supply at the onset of SSE, potentially leading to transient sub-clinical myocardial ischemia. It has previously been shown that ST segment depression, a clinical sign of myocardial ischemia, can occur with SSE if it is not preceded by a warm up [4, 5]. Specifically, Barnard and colleagues [5] found an abnormal electrocardiogram (ECG) in 60% of 54 asymptomatic male subjects. This included minor ST or T wave abnormalities and ischemic ST segment depression post SSE and 68% presented with specific ST segment abnormalities in their ECG after SSE. A transient decrease in myocardial oxygen availability immediately (< 2s) upon exposure to SSE has previously been demonstrated in dogs as observed by a decrease in coronary blood flow [6]. However, the exact nature of the cardiac effects of SSE are unclear. Echocardiography during SSE (cycling exercise) showed no evidence of left ventricular dysfunction[7]. In humans, vigorous activity can acutely increase the risk of a cardiac event in susceptible athletes [8-10]. Athletes are frequently facing imbalances in myocardial oxygen supply and demand in vigorous intensity exercise which may increase the risk of myocardial ischemia in this population [11].

The organization of sporting events may create additional risk on the athlete heart as there is often a lapse in time between warm up and commencement of vigorous activities. It has been suggested that a hormonal response triggered by vigorous intensity exercise without prior warm up results in a large release of catecholamines; a response that may increase the oxygen requirement of the myocardium despite the same workload [12]. This increase in myocardial oxygen requirements beyond the requirements at rest can lead to ischemic hypoxia conditions when the body is not effectively prepared for maximal exercise. A two minute warm up preceding SSE reduced the presence of ECG abnormalities and allowed for a progressively higher HR, while a more aggressive warm up resulted in elimination of almost all previously viewed abnormalities [5]. Therefore it appears that a warm up allows organ systems to more adequately face the demands required with the onset of SSE, but even with a warm up, the demands of SSE will outmatch any supply by physiological systems. A warm up has, however, been shown to negate the sub-clinical myocardial ischemia that is observed with SSE with no preceding warm up activity [5], by potentially allowing the physiological systems to adjust to the workload ensuring that myocardial oxygen supply and demand are more properly matched.

The effects of a delay following warm-up on the HR response to SSE in young, relatively fit males, are unknown. It is also unknown whether this delay can result in the same ECG abnormalities indicative of ischemia as seen in previous studies. Therefore, the purpose of this study was to examine the HR responses to SSE under three conditions: warm-up (WU), no warm-up (NW), and a 10 minute delay following warm-up (D) and to determine if HR responses that are indicative of an ischemic response to SSE occur despite warm up when a 10 minute delay is implemented. We hypothesized that the HR response to SSE would be greater in WU than in NW and D; that the HR response to SSE in D would be less than in WU; and that any elevation in the HR response to SSE in D would disappear by the onset of SSE. We also hypothesized that ECG abnormalities indicative of ischemia would be present in D and NW but not in WU.

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2. METHODS

2.1 Subjects

Seven males (age = 22.4 ± 2.2 yr, height = 179 .9 ± 8.1 cm, mass = 75.3. ± 5.3 kg, resting blood pressure = 123 ± 0.7 / 80 ± 11.3 mm Hg, maximal oxygen uptake (VO_2max) 57.6 ± 2.1 mL.kg-1.min-1) volunteered for this study. Young (18 – 39 years), recreationally or competitively active males who were accustomed to treadmill running were included. Smokers, those with known cardiac, pulmonary, metabolic diseases, or with musculo-skeletal injuries were excluded. Informed written consent was obtained for all subjects under the standards set by the Declaration of Helsinki. The institutional Research Ethics Board approved this study.

2.2 Protocol

Subjects reported for testing on four separate occasions, with a minimum of 24 h between exercise sessions. The first involved a treadmill VO_2max test with a 12 lead ECG. The other three conditions were randomly ordered and had participants complete a supra-maximal sprint at 9.6mph and 15% grade for 15 seconds with differing warm up conditions. The three conditions were; no warm up (NW), warm up (WU) for 5 minutes at 5.5 mph (approximately 65% of their maximal HR) and lastly the warm up described previously followed by a 10 minute seated delay (D). During all conditions subjects were monitored by a 12 lead ECG with measurements taken prior, during and after the 15 second sprint. Before each bout of SSE subjects stood motionless in order to achieve a resting steady state HR and ECG trace. Once the treadmill reached full speed and grade subjects were instructed to carefully straddle the treadmill belt and lightly grip the handrails. Subjects tested the speed and grade of the treadmill with one foot while continuing to hold the handrail. When subjects felt comfortable they jumped onto the treadmill and sprinted for 15 seconds. Time began when both feet were on the treadmill and the subject had released both hands from the rail. Subjects straddled the treadmill belt when the 15 seconds were up.

2.3 Metabolic Testing

The metabolic cart (ParvoMedics, True One 2500, Utah) was calibrated prior to each test by comparing high and low oxygen (20.93 and 12.0%) and carbon dioxide (6.0 and 0.03%) levels. The pneumotach was calibrated with a known volume (3 L) of air prior to each test. Ambient humidity, temperature and barometric pressure were measured and recorded as part of the calibration.

Subjects avoided exercise, alcohol and caffeine for 6h before testing. The VO_2max tests were performed on a treadmill (Quinton, ST65, Washington) starting at 5.0 mph and increasing by 0.5 mph/min. Expired gases were collected by a one-way valve system (Hans Rudolph Inc., Kansas), and analyzed by the metabolic cart. At 8.0 mph subjects signaled whether they wanted to continue to increase by speed or by gradient of 0.5% every minute. If subjects chose speed, it was increased until they reached 9.5 mph at which point grade was increased (due to maximal speed restrictions of the treadmill). When subjects reached volitional exhaustion they safely removed themselves from the treadmill belt and the treadmill was stopped. Subjects were determined to be at VO_2max if RER was >1.1, they achieved a HR within 10% of age-predicted maxima, or a plateau in VO_2 was observed. All subjects reached VO_2max with the final value being determined as the average over the last minute of exercise.

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2.4 ECG testing

During exercise testing, electrocardiographic (ECG) signals were continuously monitored (Quinton, ST65, Washington). A 12 lead ECG was used to analyze HR rhythm with leads V1, V2, V3, V4, V5, V6, LA, RA, LL, and RL. Prior to placing leads on the upper body, subjects had chest hair removed using a medical disposable razor, a gauze pad was used to remove the most superficial layer of skin and ECG prepping gel was used to ensure proper electrical conductance. Electrodes were peeled and placed using anatomical landmarks determined by palpation. Leads were gathered and tied together and a waist strap was used to prevent excessive pulling of the electrodes or movement artefact of the leads during exercise. Heart rate was assessed by measuring the R-R interval over six consecutive heart beats.

2.5 Statistical analyses

A 3 X 5 repeated measures ANOVA was used to measure HR at pre, 0-5 seconds, 5-10 seconds, 10-15 seconds, and immediately post sprint, and between the conditions of NW, WU and D.

3. RESULTS

Participants were 20 to 26 years of age and were recreational or competitive athletes (see Table 1).

Table 1. Subject characteristics.

Variable	Value
Age (yrs)	22.4 ± 2.2
Height (cm)	179.9 ± 8.1
Weight (kg)	75.3 ± 5.3
BMI (kg/m^2)	23.3 ± 2.1
$VO_2max\,(L \cdot min^{-1})$	4.3 ± 0.4
$VO_2max\,(mL \cdot kg^{-1} \cdot min^{-1})$	57.6 ± 2.1

Heart rate was shown to increase from the start of the 15 second sprint until its completion in all three conditions. The NW condition showed the lowest HR values from 88 at the beginning of exercise to 137 at the cessation of exercise. The WU condition showed the smallest increase in HR across the 15 second bout of exercise, starting at 123 and increasing to 155. The D condition showed HR values from 100 at the start of exercise to 144 at completion. Statistically significant ($P < .05$) differences in HR were found between

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WU and NW throughout exercise, and between WU and D for all time points except immediately post-exercise. HR was not consistently different between D and NW through 15 seconds of SSE (Figure 1). One subject met the criteria for ischemia but due to the underlying condition of left ventricular hypertrophy, a true diagnosis could not be stated.

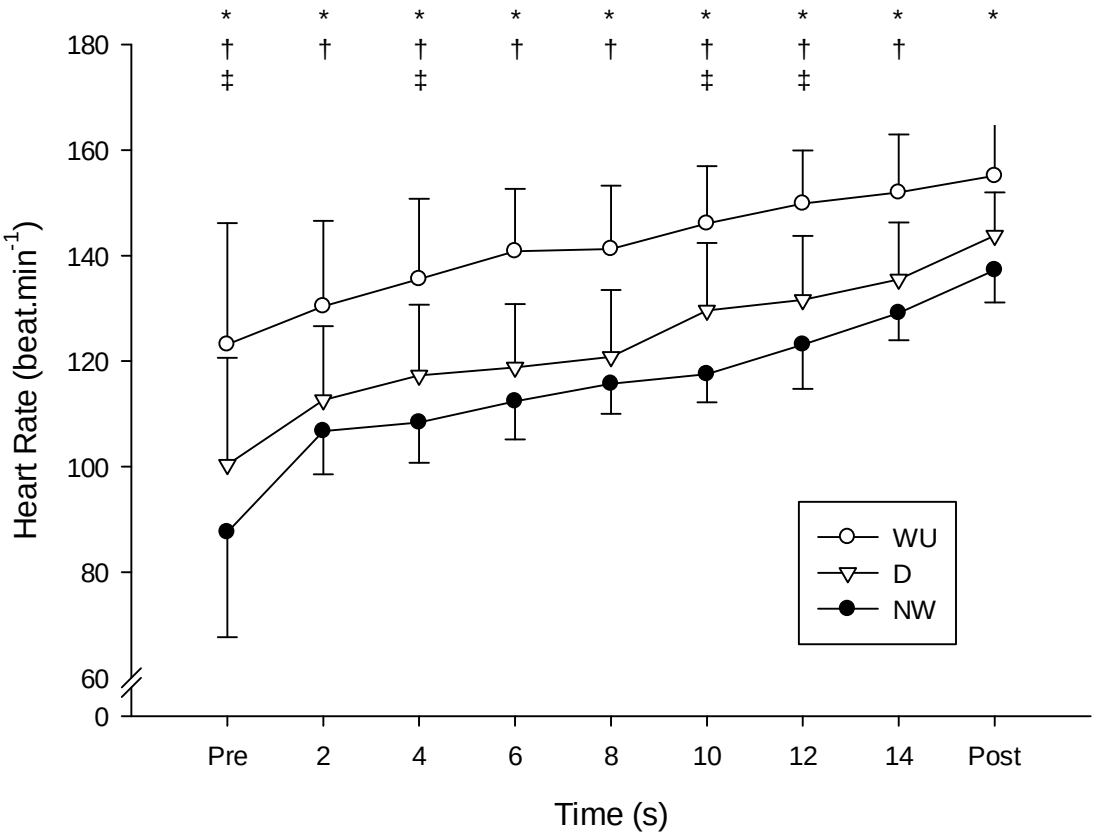

Fig. 1. HR response to SSE in three different conditions: WU, NW, and D.

Mean ± S.D.
* WU different from NW ($P < .05$)
† WU different from D ($P < .05$)
‡ D different from NW ($P < 0.5$)

4. DISCUSSION

The main finding of this study are the difference in the HR response with a warm-up prior to SSE, including when there is a delay following the warm-up prior to beginning the SSE. There was a relatively linear increase in HR throughout the sudden strenuous exercise

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across all conditions. The differences in the HR response to SSE between WU and NW confirm the well-established findings that warm-up aids in raising HR prior to exercise. The main novel finding of this study is that a 10 minute delay between warm-up and SSE does not consistently lead to a greater HR response prior to exercise than no warm up. While the HR response to SSE following the delay is lower than with no delay following warm-up, there appears to be some benefit to a warm-up followed by delay over no warm-up prior to exercise.

Differing HR responses between the three test conditions of WU, NW and D suggest that the warm up effect can significantly dissipate with a 10 minute delay. However, despite lower HR responses in the D condition compared to the WU condition, there was a higher HR during the SSE than observed in NW, though it was not statistically significant throughout the SSE. This could be explained by a heightened level of catecholamines still circulating in the blood after the 10 minute delay. An increase in symapthoexcitation was observed 30 and 45 minutes after moderate cycling, for instance, plasma norepinephrine levels were 15% and 12% higher than rest at 30 and 45 minutes into recovery [13].

Timing of warm up prior to vigorous exercise is imperative to prioritizing the safety of athletes, as susceptible individuals may have a cardiovascular event triggered by vigorous exercise combined with an inadequate myocardial perfusion response. This includes allowing for proper organization of sporting events to ensure an appropriate warm up period and reduce delays between warm up and competition. Warming up at a higher intensity or for a longer duration may be beneficial when it is known that a rest period will occur prior to competition as it may enhance the warm up effect. It has been suggested that 80% of maximal HR is an optimal warm-up intensity for performance benefits in athletes [14]. Subjects in this study warmed up at 65% of their maximal HR leading to the possibility that a more optimal intensity may further mitigate the effects of a delay.

Ischemic changes that were noted in the ECG's of previous studies involving SSE [4, 5] were not replicated in this study. Barnard et al. found ECG abnormalities with SSE diagnosable as ischemia in 19 of 44 subjects [5]. Subjects, however, ranged in age from 21 to 52 years and included a wide range of physical fitness levels from marathon runners to sedentary individuals. Results did not correlate between subject characteristics and signs of ischemia, and therefore assumptions cannot be inferred, but it is likely that the age and physical fitness level of participants impacted the findings. Trained subjects have been shown to have a reduction in myocardial oxygen demands [15]. In addition Thompson et al., noted that myocardial oxygen demands are reflective of oxygen requirements relative to maximal capacity and do not apply uniformly among individuals across absolute work rates [16]. Myocardial oxygen demand at rest in trained subjects have been shown to be 18% lower than in untrained subjects [15]. Trained subjects were shown to maintain a higher percentage of subendocardial blood flow at maximal levels of exercise reducing the likelihood of a decrement in coronary circulation during maximal exercise. As a result trained subjects show a more favorable oxygen supply/demand ratio than untrained subjects making them less likely to develop myocardial ischemia at maximal workloads [15].

Further, myocardial ischemia is not always detected by ECG and therefore it is difficult to determine if this study truly represents a negative finding or whether there was a failure to detect a sub-clinical ischemia. One of the limitations in using ECG recordings to detect myocardial ischemia is the variation in electrode placement. It has been shown that clinicians in the emergency department had wide variation in the anatomical placement of electrodes resulting in changes in ECG recordings with an impact on clinical assessment [17]. Ambulatory ECG monitoring has been shown to detect ischemic changes in only 40-60% of patients with positive diagnosis of coronary artery disease, suggesting a large source

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of error in the detection of disease [18]. Although the ECG's represented in this study were not ambulatory in nature, it likely corresponds to a similar ignorance of positive changes.

5. CONCLUSION

In conclusion, a 10 minute delay between warm up and SSE results in a lower HR response to SSE than a warm-up immediately prior to SSE. A 10 minute delay following warm-up leads to a HR response to SSE that is slightly elevated compared to no warm-up, but not consistently throughout 15 seconds of SSE. We did not observe ECG abnormalities indicative of ischemia as observed in prior studies. It is noted that the relatively small sample size in this study is a significant limitation in applying these results in general populations, and the findings may be viewed as preliminary with further research warranted. In addition, these results are limited in external generalizability outside of the subject pool of young, trained males. Athletes, coaches and sporting event organizers should be aware of the value of immediate warm up proceeding SSE, and of a longer duration and/or higher intensity warm-up when there is significant delay before the sudden strenuous event.

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COMPETING INTERESTS

No competing interests exist for any author.

AUTHORS' CONTRIBUTIONS

IA Lesser designed the study, wrote the protocol, performed the data collection and statistical analysis, wrote the protocol, and wrote the first draft of the manuscript. ANH Hodges supervised the study design, data collection and statistical analyses, and wrote the final draft of the manuscript. All authors read and approved the final manuscript.

CONSENT

All authors declare that written informed consent was obtained from the participants for publication of this study.

ETHICAL APPROVAL

All authors hereby declare that all experiments have been examined and approved by the appropriate ethics committee and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

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