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Physiological Responses at the Critical Heart Rate During Treadmill Running

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The purposes of this study were to: 1) determine if the critical heart rate (CHR) model for cycle ergometry can be applied to treadmill running; and 2) examine the times to exhaustion (Tlim) as well as the $\dot{V}_O_2$, ratings of perceived exertion (RPE), electromyographic amplitude (EMG AMP) and mean power frequency (MPF) responses during constant heart rate (HR) runs at CHR-5 b·min$^{-1}$ (CHR-5), CHR, and CHR+5 b·min$^{-1}$ (CHR+5). Thirteen runners performed an incremental treadmill test to exhaustion. On separate days, 4 constant velocity runs to exhaustion were performed. The total number of heart beats (HBlim) for each velocity was calculated as the product of the average 5 s HR and Tlim. The CHR was the slope coefficient of the HBlim versus Tlim relationship. The physiological responses were recorded during the constant HR runs. Polynomial regression analyses were used to examine the patterns of responses for all. The HBlim versus Tlim relationship ($r^2 = 0.995 – 1.000$) was described by the linear equation: $HB_{lim} = a + CHR(T_{lim})$. The CHR-5 (mean ± SD = 171 ± 8 b·min$^{-1}$, 88 ± 3% HRpeak), CHR (175 ± 8 b·min$^{-1}$, 91 ± 3% HRpeak), and CHR+5 (178 ± 8 b·min$^{-1}$, 94 ± 3% HRpeak) were maintained for 56.97 ± 1.23, 48.37 ± 11.04, and 20.11 ± 16.08 min, respectively. There was no change in HR, quadratic decreases in velocity and $\dot{V}_O_2$, and quadratic or linear increase in RPE during continuous runs at a constant HR. At CHR-5, EMG AMP decreased and EMG MPF increased. There was an increase in EMG AMP
and no change in EMG MPF at CHR, while there was no change in EMG AMP and EMG MPF decreased at CHR+5. These findings indicated the CHR model for cycle ergometry was applicable to treadmill running, and, on average, HR values that were less than or equal to the CHR, represented sustainable (30 to 60 min) intensities, while CHR+5 did not.
DEDICATION

To the One who makes all things possible. I am so grateful for this educational opportunity and I thank God for all the people in my life who have supported and encouraged me through the many challenges, failures, and successes that have enabled me to reach this point. To two of the most important people in my life, my loving parents, Jerry and Mary. I am eternally grateful to them for instilling in me the importance of pursuing excellence with integrity, persevering in the face of challenges, showing grace in defeat, and humility in success. To my brother, Bjorn who has always been my ally and my hero. To my mentor, Dr. Terry Housh for the countless hours he spent preparing me for the future and for giving me the opportunity to make one of my passions my lifework.

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CHAPTER I
INTRODUCTION

Moritani et al. (73) applied the critical power (CP) test of Monod and Scherrer (72) for synergistic muscle groups to cycle ergometry. The CP test relates the amount of work accomplished (or work limit; $W_{lim}$) and the time to exhaustion (or time limit; $T_{lim}$) from a series of three or four continuous, fatiguing cycle ergometer work bouts at varying power outputs (73). The $W_{lim}$ (power x $T_{lim}$) is plotted as a function of the $T_{lim}$ for each work bout. The $W_{lim}$ versus $T_{lim}$ relationship provides estimates of two parameters: CP is the slope and anaerobic work capacity (AWC) is the y-intercept. Theoretically, CP represents the highest sustainable power output (i.e., the asymptote of the power-duration curve), while AWC is a measure of the total work that can be performed utilizing only stored energy sources within the muscle (e.g., adenosine triphosphate (ATP), phosphocreatine, glycogen, and the oxygen bound to myoglobin) (72, 73).

Hughson et al. (48) showed that, like power output and $T_{lim}$ during cycle ergometry, running velocity and $T_{lim}$ could be described by a hyperbolic relationship (Figure 1a) during treadmill running. In addition, the authors (48) suggested that the y-intercept of the linear relationship between velocity and $1/T_{lim}$ represented the fatigue threshold that was analogous to CP during cycle ergometry, defined as the maximum running velocity that could be maintained for an extended period of time without fatigue. An algebraic manipulation of this mathematical model can be made in which the total distance (TD; velocity x $T_{lim}$) is plotted as a function $T_{lim}$ (77) (Figure 1b) for a series of exhaustive, constant velocity runs at different velocities. For this test, TD is analogous to WL from the CP test of Monod and Scherrer (72) and Moritani et al. (73) for synergic
muscle groups and cycle ergometry. The slope of the linear relationship between TD and $T_{lim}$ is the critical velocity (CV) and the y-intercept is the anaerobic running capacity (ARC).

Originally, Monod and Scherrer (72) suggested CP represented an exercise intensity that could be maintained for “a very long time without fatigue” (p. 329). In addition, Moritani et al. (73) defined CP as “the power output that a muscle group can maintain without exhaustion” (p. 339) and reported that CP occurred at the same exercise intensity as the “anaerobic threshold” determined from gas exchange parameters. The anaerobic threshold has been used to demarcate the moderate from heavy exercise intensity domains (31) and is defined as the highest power output that can be performed without a significant increase in blood lactate concentration. Other studies (52, 88), however, have reported that CP represented an intensity that was above the anaerobic threshold and overestimated the power output that can be maintained for an extended period of time without exhaustion (45, 50, 66). Furthermore, it has been shown (88) that during continuous exercise at CP, blood lactate concentration and $\dot{V}O_2$ increased before reaching steady state values after approximately 18 min of exercise. Thus, it has been suggested that CP represents the demarcation of the heavy and severe exercise intensity domains, and that for exercise above CP, $\dot{V}O_2$ will be driven to $\dot{V}O_2$ max by the slow component of oxygen consumption (31, 88). According to Gaesser and Poole (31), the $\dot{V}O_2$ slow component is defined as a gradual rise in $\dot{V}O_2$ (> 200 mL·min$^{-1}$) after the third minute of exercise. During continuous exercise within the heavy domain (i.e., between the anaerobic threshold and CP), $\dot{V}O_2$ and blood lactate reach steady state values after approximately 10 to 20 min. At power outputs greater than CP (i.e., within the severe
exercise intensity domain), however, blood lactate concentration fails to stabilize and the \( \dot{V}O_2 \) slow component drives oxygen consumption to \( \dot{V}O_2 \) max (31, 88).

It has been suggested (51, 56, 57) that the \( \dot{V}O_2 \) slow component is related to development of fatigue that results in a decreased efficiency of the recruited muscle fibers as well as the additional recruitment of less efficient fast-twitch muscle fibers. These fatigue induced neuromuscular responses are reflected in the time and frequency domains of the electromyographic (EMG) signal (21, 46, 82). For example, fatigue induced changes in the time domain are characterized by an increase in EMG amplitude (AMP) that reflects the recruitment of additional muscle fibers, increased firing rates, and/or synchronization (3, 38, 39). Within the frequency domain, the appearance of fatigue is associated with a decrease in action potential conduction velocities and a decline in EMG mean power frequency (MPF) (3, 38, 39). These specific responses have been shown to differ during continuous exercise in the heavy versus severe intensity domains (21, 38, 39, 46, 82). Specifically, greater positive slope coefficients for EMG amplitude and greater negative slope coefficients for EMG MPF over time have been reported for exercise intensities in the severe domain than the heavy domain (21, 38, 39, 46, 82). That is, higher power outputs are characterized by a greater increase in motor unit activation, as well as greater decreases in action potential conduction velocity and EMG MPF than lower power outputs (21, 38, 39, 46, 82).

Theoretically, CP represents the highest exercise intensity where metabolic steady state is reached and exercise at CP can be maintained for an extended period of time (~30 to 60 min) (21, 88). Although CP is expressed as a power output, it is characterized by specific physiological responses (i.e., \( \dot{V}O_2 \), HR, and blood lactate). Studies (14, 49) that
have examined the metabolic responses and sustainability of constant intensity exercise performed at CP have questioned the validity of the CP model to estimate the demarcation of the heavy from severe exercise domain. Specifically, it has been shown (14, 49) that continuous exercise at CP resulted in progressive increases in metabolic parameters and thus, overestimated a sustainable exercise intensity. For example, Brickley et al. (14) found that although the physiological responses did not reach maximal values during continuous exercise at CP, there were continuous increases in $\dot{V}O_2$ (3.7 L·min$^{-1}$ to 4.13 L·min$^{-1}$), HR (120 b·min$^{-1}$ to 186 b·min$^{-1}$), and blood lactate concentration (> 6.5 mmol·L$^{-1}$ after 20 min) throughout the rides. In addition, Jenkins and Quigley (49) found that only 25% of cyclists could complete 30 min of exercise at CP. To maintain exercise for 30 min, the power output had to be reduced by 6.7% for 75% of the cyclists. Thus, these findings (14, 49) suggested that CP did not represent the demarcation of the heavy and severe exercise intensity domains.

Based on the tendency for CP to overestimate the demarcation of the heavy and severe exercise intensity domains, Mielke et al. (70) proposed that the CP model, applied to a physiological parameter such as heart rate (HR), would provide a better estimation of the highest metabolic steady state exercise intensity. Therefore, Mielke et al. (70) developed a HR analog to the CP test called the critical heart rate (CHR) test. For this test, four exhaustive, constant power output rides were performed and the total number of heartbeats ($HB_{lim}$) was calculated for each power output. The $HB_{lim}$ was plotted against $T_{lim}$ and the slope of the line was CHR. Thus, CHR represented a physiological intensity that, theoretically, could be maintained for an extended period of time (70).
It has been shown (53, 69) that the responses during continuous exercise at a constant HR differ from those at a constant power output. For example, Mielke (69) showed that, during continuous rides at 5 b·min⁻¹ below CHR (CHR-5), CHR, and 5 b·min⁻¹ above CHR (CHR+5) power output was continuously reduced and the $\dot{V}O_2$ responses tracked the decrease in power output. There were, however, increases in the ratings of perceived exertion (RPE) during the constant HR rides at each intensity (CHR-5, CHR, and CHR+5). In addition, Mielke (69) found that there were decreases in EMG amplitude and increases in EMG MPF during continuous cycle ergometry at CHR-5, CHR, and CHR+5. Although no previous studies have examined neuromuscular responses during constant HR running, metabolic responses similar to those observed during constant HR cycle ergometry have been reported. Specifically, Kinderman et al. (53) showed that, during treadmill runs at a constant HR at 91% HR_peak, velocity was continuously reduced and $\dot{V}O_2$ as well as blood lactate concentrations decreased.

Together, the results of these studies (53, 69) indicated that during constant HR exercise, metabolic (i.e., $\dot{V}O_2$ and blood lactate concentration) and neuromuscular (i.e., EMG AMP and EMG MPF) responses differ from those typically associated with a fatiguing exercise bout.

Previous studies (17, 47, 77) that have examined the validity of CV have shown, that like CP, continuous, constant velocity exercise at CV overestimates the demarcation of the heavy and severe exercise intensity domains. For example, using power curve analyses ($y = ax^b$), Housh et al. (47) reported that there were significant differences between CV (14.0 ± 0.4 km·hr⁻¹) and the predicted running velocity that could be maintained for 30 min (12.1 ± 0.5 km·hr⁻¹). Furthermore, Pepper et al. (77) reported that
the mean time to exhaustion at CV was 16.43 ± 6.08 min and it has been shown (47, 77) that CV over-predicted a sustainable running intensity by 15 to 16%. No previous studies, however, have applied a physiological parameter to the CV model. It is possible that a running threshold based on a physiological measure (such as HR) would provide a better estimate of a sustainable exercise intensity (69, 70). In addition, although the physiological responses during constant power output and velocity exercise are well documented, less is known about these responses during constant HR exercise. Furthermore, no previous studies have examined the neuromuscular responses during treadmill running at a constant HR. Therefore, the purposes of this study were to: 1) determine if the CHR model of Mielke et al. (70) can be applied to treadmill running; and 2) examine the sustainability as well as metabolic ($\dot{V}O_2$), perceptual (RPE), and neuromuscular (EMG AMP and EMG MPF) responses during constant HR runs at CHR-5, CHR, and CHR+5. Based on the results of previous studies (13, 50, 53, 69, 70) we hypothesize that: 1) the CHR model for cycle ergometry can be applied to treadmill running; 2) the CHR-5 and CHR will be sustained for at least 30 min, but CHR+5 will not; 3) during each of the constant HR runs (CHR-5, CHR, and CHR+5), the velocity will be continuously reduced and $\dot{V}O_2$ will track the decreases in velocity, the RPE will increase, EMG amplitude will decrease, and EMG MPF will increase.
CHAPTER II
REVIEW OF LITERATURE

1) Development of the Critical Power and Critical Velocity Models

1.1 The Original Two-parameter Linear Critical Power Model

Monod and Scherrer (72)

The authors developed the critical torque (CT) and CP model for intermittent isometric and dynamic muscle actions. Critical power, defined as the maximum power output (P) that can be maintained without exhaustion, was described by the relationship between the time to exhaustion ($T_{lim}$) and the total amount of work performed ($W_{lim} = P \times T_{lim}$). The $T_{lim}$ and $W_{lim}$ were determined from three separate constant work rate tests performed to exhaustion. A mathematical model was used to linearly relate $W_{lim}$ and $T_{lim}$ described by the equation $W_{lim} = a + b(T_{lim})$. The authors concluded that three separate parameters can be identified from this mathematical model: 1) CP, defined as the slope ($b$); 2) anaerobic work capacity (AWC), where the y-intercept ($a$) represents the anaerobic work capacity (AWC), which is the total amount of work that can be performed above CP using only stored energy sources within the active muscles (i.e., independent of oxygen supply); and 3) $T_{lim}$ for any power output greater than CP ($T_{lim} = \frac{a}{P-b}$).

Moritani et al. (73)

The purpose of this study was to apply the CP concept to whole-body exercise. A secondary purpose was to determine the relationships among CP, the anaerobic threshold (AT), and maximal oxygen consumptions ($\dot{V}O_2$ max). Eight male (20-33 years) and eight
female (18-24 years) subjects performed an incremental test to exhaustion on an electrically braked cycle ergometer to determine the AT and $\dot{V}O_2$ max. Critical Power was determined from three work bouts performed at constant power outputs to exhaustion. For each test, the maximal work performed ($W_{\text{lim}} = P \times T_{\text{lim}}$) was plotted against the time to exhaustion ($T_{\text{lim}}$). Critical power was the slope and anaerobic work capacity (AWC) the y-intercept of the regression equation $W_{\text{lim}} = a + b(T_{\text{lim}})$. The relationship between $W_{\text{lim}}$ and $T_{\text{lim}}$ was highly linear ($r > 0.98$). There were significant correlations observed between $\dot{V}O_2$ max (mL·kg·min$^{-1}$) and CP ($r = 0.919$) and between the $\dot{V}O_2$ at CP and the $\dot{V}O_2$ at AT ($r = 0.927$). Based on these findings, the authors concluded that: 1) there was a linear relationship between $W_{\text{lim}}$ and $T_{\text{lim}}$; 2) the slope represents CP, which is dependent on oxygen supply; 3) CP and AWC could be used in a regression equation to predict $\dot{V}O_2$ max ($\dot{V}O_2$ max = 0.00795 x [(CP or b) + a] + 0.114); and 4) $T_{\text{lim}}$ can be estimated for any power output less than CP from the hyperbolic power time curve. The equation for the estimation of $T_{\text{lim}}$ was derived as follows:

$W_{\text{lim}} = P(T_{\text{lim}})$ and $W_{\text{lim}} = a + b(T_{\text{lim}})$,

Thus, $P(T_{\text{lim}}) = AWC + CP(T_{\text{lim}})$ and $T_{\text{lim}} = AWC/(P - CP)$

### 1.2 Application of the Critical Power Model to Treadmill Running

Hughson et al. (48)

The purpose of this study was to apply the CP model for cycle ergometry to treadmill running. The hyperbolic power-time relationship can be linearized by expressing power relative to the inverse of time ($P = AWC \cdot t^{-1} + CP$). This model was then applied to high velocity treadmill running by substituting velocity for power. Six
males (19 – 22 years) performed an incremental treadmill test to exhaustion for the determination of \( \dot{V}O_2 \) max. Six randomly ordered constant velocity runs were performed at a range of intensities (19.2 – 22.4 km·hr\(^{-1}\)) designed to exhaust the subjects within 2 to 12 min. Critical velocity was determined using linear regression for the velocity versus 1/time relationship for each subject. The velocity versus 1/time relationships were highly linear (0.979 – 0.997) and CV was correlated with \( \dot{V}O_2 \) max (0.84). In addition, the velocity-time relationship was used to predict the finishing time for a 10 km race. There was a significant correlation (\( r = 0.67 \)) between the predicted and actual times to exhaustion. The predicted time, however, over-predicted the actual finishing time by 2 to 3 min. The authors concluded that, based on the highly linear velocity versus 1/time relationships, the CP model could be applied to treadmill running to determine the velocity (CV) that reflects the aerobic intensity that can be sustained for a long duration. In addition, it was suggested that the differences between actual and predicted finishing time were the results of weather conditions and/or differences between track and treadmill running.

Summary

The articles outlined within this section provided the theoretical foundation for the CP concept. The authors (48, 72, 73) showed that the CP model can be applied to continuous and intermittent isometric muscle actions as well as dynamic muscle actions such as cycle ergometry and treadmill running. The studies by Monod and Scherrer (72) and Moritani et al. (73) suggested that a highly linear relationship (\( r = 0.98 \)) exists between the total work (\( W_{lim} \)) and time to exhaustion (\( T_{lim} \)) and can be described by the
equation \( W_{\text{lim}} = a + b(T_{\text{lim}}) \). The slope \( b \) and the y-intercept \( a \) represent CP and anaerobic work capacity (AWC), respectively. In addition, Hughson et al. (48) showed that the CP model could be applied to treadmill running and reported that there was a hyperbolic velocity-time relationship that could be linearized by expressing velocity relative to the inverse of time. It has been shown (48, 72, 73) that the CP model provides estimates of three separate parameters: (1) CP, defined as the maximum intensity that can be maintained without exhaustion; (2) AWC, described as the total work performed above CP that is derived from muscular energy reserves and independent of oxygen supply; and (3) the predicted time to exhaustion \( (T_{\text{lim}}) \) for any power output above CP from the hyperbolic power versus time relationship \( (T_{\text{lim}} = \frac{\text{AWC}}{\text{P-CP}}) \).

2) Parameter Estimates from the Critical Power and Critical Velocity Models

2.1 Critical Power and Critical Velocity as the Demarcation of Exercise Intensity Domains

deVries et al. (28)

The purpose of this study was to establish the relationship between CP and the neuromuscular fatigue threshold (FT) as estimated from electromyography (EMG) during cycle ergometry. Five males and six females between 19 and 32 years completed a maximal incremental test to determine the ventilatory threshold (VT) and four exhaustive, constant power output rides (1.73 – 4.96 min) to estimate CP and FT. The CP was the slope of the total work versus time to exhaustion. The slope coefficient for the EMG amplitude versus time relationship was determined for each subject for each power output. The power outputs were plotted as a function of the slope coefficients and the y-intercept was the FT. The mean \((\pm \text{SEM})\) CP \( (169.5 \pm 12.8 \text{ W}) \) was significantly lower
than the FT (190.5 ± 14.0 W), but the two thresholds were highly correlated (r = 0.869).
There was no difference between CP and the VT (187.1 ± 15.9 W) and they were
correlated at r = 0.877. The FT and VT were also highly correlated (r = 9.903). Based on
the significant correlations among these thresholds (CP, FT, and VT), the authors
suggested that they represented a similar underlying physiological mechanism. It was
suggested that the accumulations of lactate led to an increase in motor unit recruitment,
and firing rate, and decreases in force production, resulting in a greater EMG amplitude
to power output ratio.

Poole et al. (88)

The purpose of this study was to examine the respiratory and metabolic responses
during continuous cycle ergometry at and above critical power (CP). Eight untrained
males (Mean ± SD; age = 22 ± 1 years; weight = 75.6 ± 4.8 kg) performed an incremental
cycling test (25 W·min⁻¹) to exhaustion to determine the ventilatory threshold (VT) and
VO₂ max. Five exhaustive constant power tests (~4-8 min) were then performed and CP
(197 ± 12W) was estimated from the linear power versus the inverse of time relationship
(P = (AWC·t⁻¹) + CP). The subjects then performed two constant power rides at CP and
CP + 5% of the maximal power output (Pmax) achieved on the incremental test. Critical
power was 69% of Pmax and 164% of VT. In addition, CP was 46% of the difference
between the VT and Pmax. The constant load tests at CP (197 ± 12W) were maintained for
24 minutes, while tests at CP + 5% Pmax averaged only 17.7 ± 1.2 min. During the
continuous rides at CP, there was an initial rapid rise in VO₂ and blood [lactate] for the
first 3 min, followed by a gradual rise in VO₂ and blood [lactate] that reached steady state
values around 18 min of exercise. At a power output greater than CP, however, VO₂ and
blood [lactate] did not reach steady state and were driven to maximal values. The results indicated that work between the VT and CP can be maintained for an extended period of time (>24 min), while work done above CP elicits fatigue within 24 minutes. Therefore, the authors concluded that CP demarcates the heavy (prolonged exercise despite increased acidosis) from the severe ($\dot{V}O_2$ max and the maximum level of metabolic acidosis are reached) exercise intensity domain.

Hill and Ferguson (42)

The purpose of this study was to determine if critical velocity (CV) determined from the hyperbolic relationship between velocity and time to exhaustion (TTE) was equal to CV' determined from the velocity and time to achieve the $\dot{V}O_2$ max (TTmax) relationship. Five male (mean age ± SD; 33 ± 7 years) and seven female (24 ± 3 years) experienced runners performed an incremental treadmill test to exhaustion to determine $\dot{V}O_2$ max and the velocity at which $\dot{V}O_2$ max was elicited (Vmax). Four exhaustive, constant velocity tests (95 – 110% Vmax) were used to determine CV and CV'. The TTE was determined as the time from the onset of exercise until volitional exhaustion and the TTmax was determined as the time from the onset of exercise until the middle of the first 15 s period when $\dot{V}O_2$ reached or exceeded the highest $\dot{V}O_2$ value recorded during the test. The CV was estimated form the nonlinear velocity versus time model using the equation 

$$TTE = AWC\cdot(velocity - CV)^{-1}$$

and CV' was estimated from the equation 

$$TTmax = AWC'\cdot(velocity - CV')^{-1}.$$  

During the constant velocity runs, the TTE and TTmax were shorter for higher velocities and both the velocity versus TTE and TTmax relationships were described by a hyperbolic function. The mean CV was $238 \pm 24$ m·min$^{-1}$ (14.3 km·hr$^{-1}$; 88% of Vmax) and the mean CV' was $239 \pm 25$ m·min$^{-1}$ (14.5 km·hr$^{-1}$; 89%
Vmax). In addition, there was no significant difference between CV and CV' and they were highly correlated (r = 0.97). The authors theorized that CV' represented the velocity at or below which $\dot{V}O_2$ max cannot be elicited and metabolic steady state is reached.

Thus, based on the non-significant difference between CV and CV', the authors suggested that CV represented the highest sustainable velocity and demarcated the heavy from severe exercise intensity domain.

Dekerle et al. (26)

The purpose of this study was to determine if CP and the respiratory compensations point (RCP) corresponded to the maximal lactate steady state (MLSS). Eleven endurance-trained males (mean ± SD; age = 23 ± 2 years) performed an incremental cycle ergometer test to exhaustion for the determination of the maximal oxygen consumption rate ($\dot{V}O_2$ max), maximal aerobic power (MAP), VT, and respiratory compensation point (RCP). The MLSS was determined from a series of constant power output rides of various intensities. Four exhaustive constant power output rides at 90%, 95%, 100%, and 110% of $\dot{V}O_2$ max were used to estimate CP. Critical power was defined as the slope of the linear total work versus time to exhaustion relationship for each subject. The power output corresponding to the MLSS (239 ± 21) was significantly greater than the power output corresponding to VT (159 ± 23) but less than CP (278 ± 22) and the RCP (286 ± 28), which were similar. The MLSS was significantly correlated (r = 0.68-0.69) with the VT and RCP, however, CP was not correlated with any intensity. The authors concluded that CP and the RCP represented a similar intensity but did not correspond to the maximal work rate that can be maintained for a long period of time without increases in $\dot{V}O_2$ or lactate.
The purpose of this study was to determine if CP represented the highest work rate that can be sustained without continual increases in phosphocreatine concentration [PCr] and inorganic phosphate concentration [Pi], and a decrease in pH. Six males (mean ± SD; age = 30 ± 8 years) performed three to four single-leg, constant work rate leg-extensions to exhaustion to determine CP from the total work versus time to exhaustion relationship. Following the determination of CP, the subjects performed leg-extension exercise to exhaustion at work rates 10% below (<CP) and 10% above (>CP) CP. The metabolic response of the quadriceps was measured with magnetic resonance spectroscopy (P-MRS). At a work rate 10% below CP, the subjects completed 20 min of exercise without significant changes in [PCr], [Pi], or pH after the first 3 min of exercise. The work rate above CP, however, could only be maintained for 14.7 ± 7.1 min and [PCr] decreased to 26 ± 16% of the baseline value. In addition, the pH decreased to 6.87 ± 0.10, while [Pi] increased to 564 ± 167% of the baseline value. The authors concluded that the metabolic responses above CP were consistent with fatiguing high-intensity exercise, while those below CP suggested an exercise intensity that could be maintained without continuous depletion of high-energy phosphates or the accumulation metabolites. Therefore, it was suggested that CP represented the demarcation of the heavy from severe exercise intensity domain. Thus, exercise above CP is limited by high-energy phosphate depletion or the accumulation of metabolites, whereas, exercise below CP is limited by the availability of oxidative substrates (e.g. muscle glycogen), hyperthermia and/or central factors of fatigue.
2.2 Times to Exhaustion and Physiological Responses at Critical Power and Critical Velocity

Housh et al. (45)

This study examined the actual time to exhaustion (AT$_{lim}$) at various percentages of CP determined from the CP cycle ergometer test compared to the predicted time to exhaustion (PT$_{lim}$) derived from the power-curve analysis. Fourteen male subjects (Mean ± SD, age = 22.36 ± 2.13 years) volunteered to participate in this study. The CP was determined from four constant power output rides (range = 176-360 W) at a pedal rate of 70 rev·min$^{-1}$ on a Monarch cycle ergometer. The W$_{lim}$ (P·T$_{lim}$) was calculated for each of the CP rides. The CP test was used to calculate PT$_{lim}$ from the formula $T_{lim} = AWC/(P - CP)$. The subjects then completed rides at CP – 20%, CP, CP + 20%, CP + 40% and CP + 60%. The results of this study indicated that AT$_{Lim}$ and PT$_{Lim}$ were highly correlated ($r = 0.84 – 0.893; p<0.05$) for the power outputs above CP. The power output that could be maintained for 60 min was estimated from the power curve (power output = ax$^b$) derived from the hyperbolic relationship between the four power loadings and AT$_{Lim}$. Theoretically, CP represents a power output capable of being maintained indefinitely without exhaustion. Therefore, the estimated power loading for 60 min was compared to CP. The mean CP of 197 ± 39 W was significantly greater than (17%; p<0.05) the predicted value that could be maintained for 60 min (164 ± 32 W), while the mean AT$_{lim}$ was 33.31 ± 15.37 min. The results of this study suggested the equation $T_{lim} = AWC/(P – CP)$ could be used to predict time to exhaustion for power outputs above CP. Furthermore, the power curve analysis indicated the CP derived from the CP test was 17% greater than the power output that could be maintained for 60 min.
Housh et al. (47)

The purpose of this study was to determine the \( \dot{V}O_2 \), HR, and plasma lactate responses as well as the time to exhaustion at the fatigue threshold (CV). Ten physically active males (mean ± SD; age = 21.1 ± 1.3 years; \( \dot{V}O_2 \) max range = 41.9 – 60.3 mL·kg\(^{-1} \)·min\(^{-1} \)) performed a continuous incremental treadmill test for the determination of \( \dot{V}O_2 \) max. Critical velocity was determined from four randomly ordered treadmill runs to exhaustion at a range of velocities (14.5 to 19.3 km·hr\(^{-1} \)) chosen to elicit exhaustion within 2 to 12 min. The CV was defined as the y-intercept of the relationship between the running velocities and the inverse of the times to exhaustion. Separate linear regressions for the \( \dot{V}O_2 \) and HR versus running velocity (6.4 – 14.5 km·hr\(^{-1} \)) relationships from the incremental test were used to estimate the \( \dot{V}O_2 \) and HR values that corresponded to CV. The plasma lactate value corresponding to CV was estimated from the plasma lactate versus \( \dot{V}O_2 \) relationship from the incremental test. There were no significant differences between CV (mean ± SEM; 14.0 ± 0.4 km·hr\(^{-1} \)) and the running velocity corresponding to \( \dot{V}O_2 \) max (14.4 ± 0.4 km·hr\(^{-1} \)). The maximal \( \dot{V}O_2 \), HR, and plasma lactate values from the incremental tests, however, were significantly greater than the \( \dot{V}O_2 \), HR, and plasma lactate values corresponding to CV, respectively. In addition, power curve analyses (\( y = ax^b \)) indicated that there were significant differences between the CV (14.0 ± 0.4 km·hr\(^{-1} \)) and the predicted running velocity that could be maintained for 30 min (12.1 ± 0.5 km·hr\(^{-1} \)). The authors concluded that CV over predicted a sustainable running intensity by approximately 16% and, thus, suggest that CV does not represent the maximal running velocity that can be maintained for an extended period of time.
Pepper et al. (77)

The purpose of this study was to determine if the $T_{\text{lim}}$ could be accurately predicted from the CV test for treadmill running. Ten males (mean ± SD; age = 23 ± 2 years; $\dot{V}O_2\max = 54.4 ± 6.6 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) performed a continuous incremental treadmill test to exhaustion for the determination of $\dot{V}O_2\max$. Four randomly ordered exhaustive, constant velocity tests (12.88 to 21.74 km·hr$^{-1}$) were used to determine CV. The total distance (TD = velocity x time) and $T_{\text{lim}}$ were recorded for each test. Critical velocity was defined as the slope of the linear TD versus $T_{\text{lim}}$ relationship. The predicted $T_{\text{lim}}$ was derived from the equation $T_{\text{lim}} = \text{ARC}/(V - \text{CV})$, where ARC is the anaerobic running capacity (y-intercept of the TD vs. $T_{\text{lim}}$ relationship) and V is any running velocity. The actual times to exhaustion were then recorded for five randomly ordered constant velocity runs at 70%, 80%, 100%, 115%, and 130% of CV and compared with the predicted times to exhaustion. The TD versus $T_{\text{lim}}$ relationship was highly linear ($r = 0.987 – 0.999$) and the mean (± SD) CV was 13.43 ± 2.04 km·hr$^{-1}$. There were no differences between the predicted and actual velocity at 85% and 115% of CV. The predicted $T_{\text{lim}}$ values at 100% and 130% of CV, however, were significantly greater than the actual $T_{\text{lim}}$ values. The mean (± SD) time to exhaustion at CV was 16.43 ± 6.08 min with a range of 9.96 to 31.90 min. In addition, CV was not different from and significantly correlated ($r = 0.81$) with the velocity corresponding to $\dot{V}O_2\max$. Thus, the authors concluded that the CV test over predicted the running velocity that could be maintained for 60 min by 15% and did not represent a sustainable exercise intensity.
This study compared CP estimates from five mathematical models and determined the time to exhaustion and physiological responses at the lowest estimate of CP from the five models. Nine male subjects (mean ± SD; age = 25 ± 3 years) completed eight or nine exhaustive cycling tests. An incremental test was performed to determine the peak power ($P_{\text{peak}}$) and peak heart rate ($HR_{\text{peak}}$). The subjects then completed five or six randomly ordered trials at power outputs ranging from $P_{\text{peak}}$ minus 50W to $P_{\text{peak}}$ plus 50 W, with at least one trial lasting longer than 10 min. Critical power was estimated from two linear, two non-linear, and one exponential mathematical model. For all subjects, the 3-parameter, non-linear model (Nonlinear-3) resulted in the lowest estimate of CP. Therefore, all subjects performed two exhaustive trials at CP estimated from the Nonlinear-3 model. There were significant differences among the estimates of CP from the five mathematical models. Specifically, the exponential model resulted in the highest estimates of CP, while the Nonlinear-3 produced the lowest estimates. Seven of the nine subjects completed 60 min at CP during one of the two CP trails and five subjects completed 60 min during both trials. The mean heart rates at exhaustion for those subjects who completed 60 min at CP in the first and second trial were $166 \pm 10 \text{ b} \cdot \text{min}^{-1}$ ($93 \pm 5\% \text{ of } HR_{\text{peak}}$) and $165 \pm 12 \text{ b} \cdot \text{min}^{-1}$ ($91 \pm 5\% \text{ of } HR_{\text{peak}}$), respectively. The mean heart rates for those subjects who did not complete 60 min at CP were $175 \pm 5 \text{ b} \cdot \text{min}^{-1}$ ($96 \pm 1\% \text{ HR}_{\text{peak}}$) and $175 \pm 7 \text{ b} \cdot \text{min}^{-1}$ ($97 \pm 6\% \text{ HR}_{\text{peak}}$) for trial one and two, respectively. In addition, the mean rating of perceived exertion (RPE) values at 60 min for the first and second trials at CP were $19 \pm 1$ and $17 \pm 3$, respectively. The mean RPE for those subjects who did not complete 60 min in either trial was $19 \pm 1$. Based on the findings
that 22-33% of the subjects could not complete 60 min at CP performed at the lowest estimate of CP, the authors concluded that CP does not represent a “fatigueless task”

Housh et al. (44)

The purpose of this study was to compare estimates of CV from five different mathematical models, as well as examine the $\dot{V}O_2$, HR, and plasma lactate values that corresponded to each estimate of CV. Ten physical active males (mean ± SD; age = 22 ± 2 years) performed a continuous incremental treadmill test (3 min stages) to exhaustion for the determination of $\dot{V}O_2$ max. During the incremental test, $\dot{V}O_2$ and HR were recorded continuously and the plasma lactate concentration was analyzed from blood samples taken in the last 30 s of each stage. Two linear, two non-linear, and one exponential model were used to estimate CV from four randomly ordered exhaustive treadmill runs (14.5 to 19.3 km·h$^{-1}$). The $\dot{V}O_2$ and HR values from the incremental test were plotted against running velocity and linear regression was used to determine the $\dot{V}O_2$ and HR values that corresponded to each of the five estimates of CV. Power curve analyses ($y = ax^b$) from the relationship between plasma lactate concentration and running velocity were used to determine the plasma lactate concentration that corresponded to the CV estimates from the five mathematical models. The 3-parameter, nonlinear (Nonlinear-3) model produced the lowest estimate of CV, $\dot{V}O_2$ (89% $\dot{V}O_2$ max), HR (93% HR max), and plasma lactate (63% maximal plasma lactate). The authors concluded that the estimates of CV, and thus, the physiological responses corresponding to CV depended on the mathematical model used. The authors identified a need for further research to identify which mathematical model would provide the most valid estimate of the demarcation of the heavy from severe exercise intensity domain.
Brickley et al. (15)

This study examined the sustainability and physiological responses during continuous cycle ergometer rides at critical power. Seven males (mean \(\pm\) SD; age = 23.4 ± 3.1 years; \(\dot{V}O_2\) max = 61 ± 9 mL·kg\(^{-1}\)·min\(^{-1}\)) completed an incremental cycle ergometer test to exhaustion for the determination of \(\dot{V}O_2\) max. Critical power was estimated from three constant power rides to exhaustion at power outputs that were 95%, 100%, and 120% of maximal power output from the incremental test. On a separate day, the subjects performed a continuous ride to exhaustion at CP. Oxygen consumption and blood lactate concentration were measured every 5 min, while HR, power output, and cadence were recorded continuously during the CP rides. The times to exhaustion at CP ranged from 20.02 to 40.62 min. Although the physiological responses did not reach maximal values, there were significant increases in \(\dot{V}O_2\) (3.7 L·min\(^{-1}\) to 4.13 L·min\(^{-1}\)), HR (120 b·min\(^{-1}\) to 186 b·min\(^{-1}\)), and blood lactate concentration (> 6.5 mmol·L\(^{-1}\) after 20 min). Based on the times to exhaustion (< 60 min) and significant increases in \(\dot{V}O_2\), HR, and blood lactate concentration, the authors concluded that CP represented a power output that could be maintained between 20 and 40 min with non-steady state physiological responses.

Bull et al. (17)

The purpose of this study was to examine the \(\dot{V}O_2\) and HR responses during continuous runs to exhaustion at critical velocity estimated from five different mathematical models. Six males and four females (mean \(\pm\) SD; age = 22 ± 2 years; \(\dot{V}O_2\) peak = 51 ± 6 mL·kg\(^{-1}\)·min\(^{-1}\); HRmax = 195 ± 7 b·min\(^{-1}\)) completed an incremental treadmill test to exhaustion for the determination of \(\dot{V}O_2\) peak and HRmax. The subjects
then completed four or five randomly ordered, exhaustive runs at velocities that resulted in fatigue within 3 to 20 min. Critical velocity was estimated from two linear, two non-linear, and one exponential mathematical model. Runs to exhaustion were performed at each of the five estimates of CV. The CV estimate from the three-parameter, nonlinear model (Nonlinear-3) was significantly lower than all other models and resulted in the lowest estimate of CV for each subject. The mean time to end of exercise was greater (57 ± 12.9 min) for the Nonlinear-3 model when compared to the other four models (22.0 ± 13.7 min – 34.5 ± 14.5 min). In addition, the mean HR at exhaustion for the Nonlinear-3 model (179 ± 18 bmin⁻¹; 92% HRmax) was significantly less than HRmax (195 ± 7 bmin⁻¹), while the HR at exhaustion was not different from HRmax for any of the other models.

There were significant increases in $\dot{V}O_2$ after the third minute of the CV runs for all five models and the increase in $\dot{V}O_2$ suggested a $\dot{V}O_2$ slow component (>200 ml kg min⁻¹) for all but the exponential model. The $\dot{V}O_2$ values at exhaustion, however, were significantly lower than $\dot{V}O_2$peak for all models. Based on these findings, the authors concluded that CV does not represent a fatigueless exercise intensity. Furthermore, it was suggested that estimates of CV that differed by as much as 18% did not support the hypothesis that CV represents the demarcation of the heavy from severe exercise intensity domain.

2.3 Neuromuscular Responses at Critical Power

Bull et al. (16)

The purpose of this study was to examine the electromyographic (EMG) amplitude responses during continuous cycle ergometry at CP. Seven moderately active males (mean age ± SD; 25 ± 3 years) completed an incremental cycle ergometer test to
exhaustion to determine peak power ($P_{\text{peak}}$) and peak heart rate ($HR_{\text{peak}}$). Critical power was determined from five or six randomly ordered rides to exhaustion at power outputs ranging from 130 W below and 50 W above $P_{\text{peak}}$. The EMG amplitude responses were recorded from the vastus lateralis during a 60 min work bout at the CP estimated from the three-parameter, non-linear model. The mean CP was 175 ± 25 W (range = 149 to 218 W), which occurred at 56 ± 5% $P_{\text{peak}}$ and 78 ± 5% $HR_{\text{peak}}$. The slope coefficient for the mean EMG amplitude versus time relationship was not significantly different from zero. The authors concluded that the lack of change in EMG amplitude indicated that no additional motor unit recruitment was required. Therefore, it was suggested that CP represented heavy-intensity exercise at which $\dot{V}O_2$ and blood lactate reach steady state.

Summary

One of the primary applications of CP is the demarcation of the exercise intensity domains. Originally, Moritani et al (73) found that CP was not different from the anaerobic threshold and deVries et al. (28) reported that CP was equal to the venilatory threshold. Theoretically, the AT and VT describe an exercise intensity that can be maintained without an increase in blood lactate concentration (73). Thus, the findings of Moritani et al. (73) and deVries et al. (28) suggested that CP represented the demarcation of the moderate from heavy exercise intensity domains. In contrast, Poole et al. (88) reported that, during continuous exercise at CP, blood lactate concentration and $\dot{V}O_2$ increased before reaching steady state values after approximately 18 min of exercise. Above CP, however, blood lactate concentration did not stabilize and $\dot{V}O_2$ was driven to its maximal value. The authors (88) suggested that CP represented the highest sustainable
power output where blood lactate and $\dot{V}O_2$ reach steady state and demarcates the heavy from severe exercise intensity domain. Similarly, Jones et al. (52) reported that during continuous exercise 10% below CP, phosphocreatine concentration [PCr], inorganic phosphate concentration [Pi], and pH reached steady state values. During continuous exercise 10% above CP, however, [PCr] and pH decreased, while [Pi] increased. Therefore, it was suggested (24) that CP represented the demarcation of the heavy from severe exercise intensity domain. Other studies (14, 26), however, have suggested that CP overestimates the highest sustainable exercise intensity where metabolic steady state is reached. Specifically, Dekerle et al. (26) reported that CP was not different from the respiratory compensation point but did not correspond to the maximal work rate that can be maintained for a long period of time without increases in $\dot{V}O_2$ or lactate.

Several studies have examined the time to exhaustion and physiological responses during continuous exercise at CP or CV. Most studies (45, 47, 77) have reported that CP overestimated the power output that could be maintained for 60 min. For example, Housh et al. (45) found that CP could be maintained for $33.31 \pm 15.37$ min and was $17\%$ greater than the power loading that could be maintained for 60 min. In addition, Pepper et al. (77) reported that CV could be maintained for $16.43 \pm 6.08$ min and overestimated the velocity that could be maintained for 60 min by $16\%$. Other studies (14) have reported that, during continuous exercise at CP, $\dot{V}O_2$, and lactate did not reach steady state.

The physiological responses at CP and CV have been shown to differ depending on the mathematical model used. Specifically, a number of studies (15, 17, 44) have shown that CP or CV estimated from the three-parameter, nonlinear model (Nonlinear-3) was significantly lower than all other models and resulted in the lowest estimate of CP or
CV for each subject. In addition, Bull et al. (16) found that EMG amplitude did not change. These findings (16) indicated that no additional motor unit recruitment was required. Therefore, it was suggested (16) that CP represented heavy-intensity exercise at which \( \dot{V}O_2 \) and blood lactate reach steady state. Together, these studies (15, 45, 47, 52, 88) suggested that CP and CV are similar to or slightly greater than the power output associated with metabolic steady state. Furthermore, the sustainability and physiological responses during continuous exercise at CP or CV are dependent on the mathematical model used to estimate these parameters (15, 44).

3) Neuromuscular Responses During Continuous, Submaximal Cycle Ergometry and Treadmill Running

Petrofsky (82)

The purpose of this study was to examine the EMG amplitude and center frequency responses during continuous cycle ergometry at power outputs between 20 and 100% of \( \dot{V}O_2 \) max. Three male subjects performed an incremental test to exhaustion to determine \( \dot{V}O_2 \) max. The subjects then cycled for 80 min or to exhaustion at 20, 40, 60, 80, and 100% of \( \dot{V}O_2 \) max. During the constant power output work bouts the EMG signals were recorded from the vastus lateralis and normalized to a maximal isometric contraction performed prior to each ride. The work bouts at 20 – 60% \( \dot{V}O_2 \) max were maintained for 80 min, while the work bouts at 80 and 100% of \( \dot{V}O_2 \) max could only be maintained for a mean of 39.2 and 7.3 min, respectively. The EMG amplitude did not change during the rides at 20 and 40% of \( \dot{V}O_2 \) max. There were, however, increases in EMG amplitude during the rides at 60, 80, and 100% of \( \dot{V}O_2 \) max. In addition, for the
work bouts at 80 and 100% $\dot{V}O_2$ max the EMG amplitude was equal to the maximal isometric contraction at exhaustion. The EMG center frequency increased during the first 20 min and then plateaued for the remainder of the work bouts at 20 and 40% of $\dot{V}O_2$ max. At 60% and 80% of $\dot{V}O_2$ max, the EMG center frequency increased and then decreased. There was a constant decrease in EMG center frequency during rides at 100% $\dot{V}O_2$ max. The author hypothesized that the lack of change in EMG amplitude and plateau in center frequency at 20 and 40% were the result of a balance between the effects of temperature and fatigue. Specifically, it was suggested that fatigue resulted in increases in EMG amplitude and decreases in center frequency, while increased muscle temperature caused decreases in EMG amplitude and increases in center frequency. The author suggested that, at higher power outputs (i.e., 60, 80, and 100% $\dot{V}O_2$ max), the increases in EMG amplitude and decreases in center frequency indicated that fatigue (increased motor unit recruitment and decreased firing rate) had a greater affect on the EMG signal than increased muscle temperature.

Borrani et al. (12)

The purpose of this investigation was to examine the relationship between the $\dot{V}O_2$ slow component and the additional recruitment of fast twitch muscle fibers. Thirteen competitive runners completed two separate running tests on a treadmill. An incremental test was performed to determine $\dot{V}O_2$ max. The second treadmill test required the subjects to run to exhaustion at 95% of $\dot{V}O_2$ max. During the constant velocity run, $\dot{V}O_2$ kinetics were assessed and EMG mean power frequency (MPF) was determined for the vastus lateralis, gastrocnemius lateralis, and soleus muscles of both lower limbs. There were
decreases in EMG MPF during the initial portion of the run, followed by significant increases in MPF that corresponded to the beginning of the \( \dot{VO}_2 \) slow component. The authors proposed that the initial decrease in MPF was the result of muscle wisdom, changes in muscle fiber conduction velocity, and/or synchronization of the slow motor units. It was suggested that the increase in MPF during the \( \dot{VO}_2 \) slow component was the result of additional recruitment of less efficient fast twitch fibers with higher firing frequencies.

Housh et al. (46)

The purpose of this study was to examine the EMG responses during continuous cycle ergometry at constant power outputs. Eight males and females (mean age ± SD; 21.5 ± 1.6 years) performed an incremental test to exhaustion on a cycle ergometer for the determination of peak power (P\(_{\text{peak}}\)). Four constant power output work bouts (50, 65, 80, and 95% P\(_{\text{peak}}\)) were performed for 15 min or to exhaustion. The EMG amplitude was recorded from the vastus lateralis (VL) and vastus medialis (VM) and normalized to the first recorded value. All of the subjects completed the 15 min work bouts at 50 and 65% of P\(_{\text{peak}}\) and three subjects completed 15 min at 80% P\(_{\text{peak}}\). The times to exhaustion at 80% (n = 5) and 95% (n = 8) P\(_{\text{peak}}\) were 9.2 ± 2.2 min and 4.3 ± 1.4 min, respectively. The slope coefficients for the mean EMG amplitude versus time relationship for the VL were significantly greater than zero at 50% (0.03), 65% (0.08), 80% (0.20), and 95% (0.47) P\(_{\text{peak}}\). The slope coefficient for the mean EMG amplitude versus time relationship for the VM at 50% (0.00) was not different from zero, however, the slope coefficients at 65% (0.05), 80% (0.23), and 95% (0.56) P\(_{\text{peak}}\) were greater than zero. The authors hypothesized that the increases in EMG amplitude were the result of peripheral, low-
frequency fatigue where increased motor unit recruitment was required to maintain the power output.

Perry et al. (79)

This study examined the mechanomyographic (MMG) and EMG responses during continuous, constant power output cycle ergometer exercise. Eight males (mean ± SD: 23.6 ± 3.5 years) performed an incremental test to exhaustion to determine peak power (P_{peak}). The MMG and EMG responses were then observed during three continuous, one-hour rides at 28% (78 ± 34 W), 35% (91 ± 42 W), and 42% (109 ± 50 W) of peak power output (P_{peak}). The MMG amplitude and EMG amplitude values were normalized to the initial amplitude value during the continuous rides. There were significant decreases in MMG amplitude over time (slope coefficient) at each power output. In addition, there were significant differences among the slope coefficients (MMG amplitude versus time) for 28% P_{peak} (-0.42 %·min^{-1}), 35% P_{peak} (-0.34 %·min^{-1}), and 42% P_{peak} (-0.48 %·min^{-1}). The EMG amplitude versus time relationship (slope coefficient) was significantly greater than zero at each power output. There were, however, no differences among the slope coefficients for the three power outputs (28% P_{peak} = 0.14; 35% P_{peak} = 0.17; 42% P_{peak} = 0.14 %·min^{-1}). The authors suggested that the decreases in MMG amplitude were due to the effects of “muscular wisdom” and/or decreases in muscular compliance, whereas, the increases in EMG amplitude were likely the result of additional motor unit recruitment.
Summary

Electromyography has been used to assess neuromuscular function during continuous constant intensity exercise. It has been shown (46, 79, 82) that EMG amplitude increases during fatiguing exercise. Specifically, Petrofsky (82) reported increases in EMG amplitude during exercise performed at an intensity greater than or equal to 60% of $\dot{V}O_2\max$. In addition, Housh et al. (46) found that there were greater increases in EMG amplitude at higher power outputs. It was suggested that the increases in EMG amplitude reflected fatigue induced recruitment of additional muscle fibers.

Fatigue has also been characterized by decreases in the frequency domain of the EMG signal. For example, Petrofsky (82) reported that there were fatigue induced decreases in EMG center frequency during continuous fatiguing exercise between 60% and 100% of $\dot{V}O_2\max$. Interestingly, the frequency domain of the EMG signal has also been shown to increase during a fatiguing task (12). It has been suggested that EMG mean power frequency (MPF) reflects changes in motor unit conduction velocity. Typically, fatigue is associated with increased metabolic byproducts that can decrease conduction velocity and EMG MPF. Borrani et al. (12) suggested, however, that decreases in EMG MPF may be offset by the fatigue-induced recruitment of additional fast-twitch motor units with higher conduction velocities. Specifically, Borrani et al. (12) examined the relationship between the $\dot{V}O_2$ slow component and the additional recruitment of fast twitch muscle fibers during a constant velocity run at 95% of $\dot{V}O_2\max$. There were decreases in EMG MPF during the initial portion of the run, followed by significant increases in MPF that corresponded to the beginning of the $\dot{V}O_2$ slow component. The authors (12) hypothesized that the initial decrease in MPF was the result of muscle wisdom, changes
in muscle fiber conduction velocity, and/or synchronization of the slow motor units, while the increase in MPF during the VO₂ slow component was the result of additional recruitment of less efficient fast twitch fibers with higher firing frequencies. These studies (12, 46, 79, 82) suggested that fatigue during continuous, constant intensity exercise is reflected by specific neuromuscular changes in the time and frequency domains of the EMG signal.

4) Heart Rate Fatigue Thresholds

4.1 Physical Working Capacity at the Heart Rate Threshold

Wagner and Housh (103)

The purposes of this study were to: 1) develop a new heart rate technique for cycle ergometry, called the physical working capacity at the heart rate threshold (PWC_HRT) test, to estimate the maximal power output that can be sustained for an extended period of time with an increase in HR less than 0.1 b·min⁻¹·min⁻¹ and 2) validate the PWC_HRT test during continuous 1-hour cycle ergometer work bouts at power outputs above and below the PWC_HRT. Eight sedentary males (mean age ± SD; 22 ± 2 years) performed four, eight min continuous, constant power output rides between 105 and 200 W. Heart rates were recorded every 15 s throughout the test. The HR values recorded during the last 5 min of each test were used to calculate the rate of rise in HR as a function of time (slope coefficient) for each subject. Following the determination of PWC_HRT, the subjects performed five randomly ordered constant power output rides at 80%, 100%, 120%, 140%, and 160% of PWC_HRT. The rate of rise in HR (after deleting the HR values for the first 3 min) was determined for each subject for each 1-hour ride. The mean slope coefficients for HR over time for the 1-hour rides at 80%, 100%, 120%,
140%, and 160% of $\text{PWC}_{\text{HRT}}$ were all greater than zero (0.011, 0.066, 0.189, 0.229, and 0.198 b·min$^{-1}$, respectively). The slope coefficients at power outputs less than or equal to $\text{PWC}_{\text{HRT}}$ were significantly less than 0.1 b·min$^{-1}$·min$^{-1}$, while those greater than $\text{PWC}_{\text{HRT}}$ were significantly greater than 0.1 b·min$^{-1}$·min$^{-1}$. Based on these findings, the authors suggested that the $\text{PWC}_{\text{HRT}}$ test provided an accurate estimate of the power output that can be sustained for an extended period of time with an increase in HR less than 0.1 b·min$^{-1}$·min$^{-1}$.

Perry et al. (80)

The purpose of this study was to examine the HR and ratings of perceived exertion (RPE) responses during continuous exercise above, below, and at the physical working capacity at the heart rate threshold ($\text{PWC}_{\text{HRT}}$). Ten males (mean ± SD; age = 23.3 ± 2.9 years) performed eight exercise tests on a cycle ergometer. The first involved determination of the maximal values for power output, HR ($\text{HR}_{\text{max}}$), and RPE ($\text{RPE}_{\text{max}}$). The $\text{PWC}_{\text{HRT}}$ protocol involved four randomly ordered, eight-minute work bouts. The power outputs for the four work bouts were low enough for the subject to complete eight minutes, but high enough to result in a positive slope coefficient for the HR versus time relationship. The HR was recorded throughout the work bout and HR values for the last five minutes of each work bout were plotted against time. The $\text{PWC}_{\text{HRT}}$ was the $y$-intercept of the relationship between power output plotted as a function of the slope coefficient of each HR versus time relationship. After the determination of the $\text{PWC}_{\text{HRT}}$, the subjects completed three continuous, one-hour constant power output rides at 80%, 100%, and 120% of $\text{PWC}_{\text{HRT}}$ and the slope coefficient for the HR versus time relationship (from 3 to 60 min) was calculated for each subject. The RPE was also recorded every 5
min during the one hour work bouts. The mean slope coefficients for HR and RPE versus

time were significantly greater than zero at 80%, 100%, and 120% of PWC_{RPE}. In
addition, the slope coefficient for HR versus time was significantly greater than 0.1
b·min^{-1}·min^{-1} and represented 56%, 63%, and 66% of the mean HR_{max} at 80%, 100%, and
120% of PWC_{HRT}, respectively. The authors concluded that the PWC_{RPE} test over
estimated the power output associated with a steady state heart rate and, based on the rate
of rise (0.2 b·min^{-1}·min^{-1}) in HR over time, suggested that the PWC_{HRT} could be
maintained for four hours.

Malek et al. (64)

This study applied the mathematical model used to estimate the physical working
capacity at the oxygen consumption (PWC_{\dot{V}O_2}) and heart rate fatigue thresholds (PWC_{HRT})
for cycle ergometry compared to treadmill running to determine the running velocity at
the oxygen consumption (RV_{\dot{V}O_2}) and heart rate thresholds (RV_{HRT}). Seven aerobically
trained males (mean ± SD; age = 24.0 ± 3.9 years; \dot{V}O_2 max = 56.7 ± 7.1 ml·kg^{-1}·min^{-1})
performed an incremental treadmill test to exhaustion for the determination of \dot{V}O_2 peak,
HR max, and the VT. The RV_{\dot{V}O_2} and RV_{HR} were determined from four randomly ordered
eight min treadmill runs at velocities that ranged from 8.05 to 14.49 km·hr^{-1}. The selected
velocities were low enough to allow the subject to complete a full eight min but high
enough to result in positive slope coefficients for the \dot{V}O_2 and HR versus time
relationships. The \dot{V}O_2 and HR responses for the first three minutes of each test were
eliminated to account for the initial metabolic and cardiac adjustment to the exercise
intensity. The slope coefficients for the \dot{V}O_2 and HR versus time relationships were
determined from the last five minutes of each test. The velocities were then plotted as a function of the slope coefficients ($\dot{V}O_2$ vs. time and HR vs. time) and the $RV_{\dot{V}O_2}$ and $RV_{HRT}$ were defined as the y-intercept of the velocity versus slope coefficient plot. The $\dot{V}O_2$ ($r = 0.66 - 0.96$) and HR ($r = 0.96 - 0.99$) versus time relationships as well as the velocity versus slope coefficients for $\dot{V}O_2$ ($r = 0.87$) and HR ($r = 0.60$) were linear. There were no significant mean differences among the velocities for the $RV_{\dot{V}O_2}$ (10.7 ± 0.4 km·h$^{-1}$), $RV_{HRT}$ (9.9 ± 0.6 km·h$^{-1}$), or VT (10.4 ± 0.5 km·h$^{-1}$). All three fatigue thresholds ($RV_{\dot{V}O_2}$, $RV_{HRT}$, and VT) were significantly inter-correlated (0.77 to 0.91). The authors concluded that the mathematical model that had been used to estimate the $RV_{\dot{V}O_2}$ and $PWC_{HRT}$ could be applied to treadmill running to determine the $RV_{\dot{V}O_2}$ and $RV_{HRT}$. In addition, the authors suggested that the $RV_{\dot{V}O_2}$ and $RV_{HRT}$ may provide submaximal protocol for estimating the VT.

4.2 Maximal Constant Heart Rate Method

Vobejda et al. (102)

The purpose of this investigation was to determine if the maximal constant heart rate ($HR_{MC}$) method could be used to predict the anaerobic threshold. Six females (31.8 ± 8.9 years) and 25 males (26.5 ± 4.0 years) performed two incremental treadmill tests to determine the anaerobic threshold (4 mmol·L$^{-1}$ lactate), two to five constant heart rate (HR) runs to identify the $HR_{MC}$, and two to five constant velocity runs to determine the maximal lactate steady state (MLSS). The first constant HR run began at 175 bpm and velocity was continuously adjusted to maintain the desired HR. If the subject was unable
to maintain a HR of 175 b·min⁻¹ or exhaustion did not occur prior to 30 min the target HR was decreased or increased by 10 b·min⁻¹ for the subsequent test. This procedure was continued until the subject could maintain the target HR for 30 min. A similar procedure was followed to determine MLSS, but velocity was kept constant during each run. The velocity for the first test to determine MLSS was set at the velocity that corresponded to the anaerobic threshold (determined from the incremental test). If the subject did not exhaust at 30 min, the velocity was increased by 0.1 m·s⁻¹ for the following test. This procedure was continued until the subject could maintain the velocity with an increase of less than a 1 mmol·L⁻¹ in blood lactate concentration for the last 20 min of the test. The mean HR_{MC} was 177.9 ± 9.0 b·min⁻¹ and the velocity at the HR_{MC} was 3.09 ± 0.45 m·s⁻¹. During the constant HR runs, velocity was continuously reduced from the 15th minute onward and blood lactate concentration tracked velocity. The velocity at MLSS was 3.39 ± 0.40 m·s⁻¹ and blood lactate concentration was 3.77 ± 1.12 mmol·L⁻¹. Heart rate increased ~10 b·min⁻¹. There were no significant differences between the velocities at the HR_{MC} and MLSS or between the velocities at the MLSS and anaerobic threshold. The velocity at the HR_{MC}, however, was greater than the velocity at the anaerobic threshold. There were, however, no significant differences among the HR values for the HR_{MC}, MLSS, and anaerobic threshold. The authors concluded that the HR_{MC} could be used to estimate the HR and velocity at the MLSS during treadmill running.
4.3 The Critical Heart Rate Model

Mielke et al. (70)

The purpose of this study was to determine if the mathematical model used to estimate CP could be applied to HR to estimate the critical heart rate (CHR). In addition, CHR was compared to the HR values at CP (CP_{HR}), ventilatory threshold (VT_{HR}), and respiratory compensation point (RCP_{HR}). Fifteen females (Mean age ± SD; 21.7 ± 2.1 years) performed an incremental test to exhaustion to determine VT_{HR}, RCP_{HR}, and \( \dot{V}O_2 \text{peak} \). The CP and CHR were determined from four randomly ordered rides to exhaustion at four different power outputs (power outputs were selected to elicit exhaustion within 8 – 20 min). Heart rate (b·min\(^{-1}\)) values (5 s means) were continuously recorded and the total number of heartbeats (HB_{lim}) was calculated as the product of the mean HR (b·min\(^{-1}\)) and time to exhaustion (T_{lim}). In addition, the product of the power output and T_{lim} were used to calculate the total work (W_{lim}) for each of the exhaustive rides. The HB_{lim} or W_{lim} was plotted against T_{lim} for each of the four power outputs and the slope coefficients of the linear HB_{lim} and W_{lim} versus T_{lim} relationships were defined as the CHR and CP, respectively. Heart rate values from the incremental test were plotted against power output values, and the regression equation derived was used to determine the VT_{HR}, RCP_{HR}, and CP_{HR}. The coefficients of determination (r\(^2\)) for HB_{lim} and W_{lim} versus T_{lim} relationships ranged from 0.985 to 1.0 and 0.866 to 0.999, respectively. The mean CHR (172 ± 11 b·min\(^{-1}\), 92.9 ± 2.7% HR_{max}) was not different from the RCP_{HR} (172 ± 9 b·min\(^{-1}\), 92.9 ± 2.2% HR_{max}), but was higher than CP_{HR} (154 ± 10 b·min\(^{-1}\), 83.2 ± 3.7% HR_{max}) and VT_{HR} (152 ± 12 b·min\(^{-1}\), 82.1 ± 4.3% HR_{max}). The authors concluded that these findings indicated that the CP model could be applied to HR to determine the
CHR from the relationship between HR and $T_{\text{lim}}$. Furthermore, the non-significant difference between CHR and RCP$_{HR}$ suggested that the CHR may provide a new method for estimating the RCP and, thus, the demarcation of the heavy and severe exercise intensity domains, without the need to measure expired gas samples.

Summary

Although fatigue threshold models are typically described by a power output or velocity, the studies in this section show that these models can be applied to physiological measures (e.g., heart rate and $\dot{V}O_2$). For example, Wagner and Housh (103) developed a new heart rate technique for cycle ergometry, called the physical working capacity at the heart rate threshold (PWC$_{HRT}$) test, to estimate the maximal power output that can be sustained for an extended period of time with an increase in HR less than 0.1 b·min$^{-1}$·min$^{-1}$. Perry et al. (80) further examined the PWC$_{HRT}$ and found that HR responses during constant power output rides at, above, and below the PWC$_{HRT}$ were significantly greater than 0.1 b·min$^{-1}$·min$^{-1}$. It was suggested (80) that the PWC$_{RPE}$ test over estimated the power output associated with a steady state heart rate and, based on the rate of rise (0.2 b·min$^{-1}$·min$^{-1}$) in HR over time, that the PWC$_{HRT}$ could be maintained for four hours. Malek et al. (64) applied the mathematical model used to estimate the PWC$_{HRT}$ for cycle ergometry to treadmill running to determine the running velocity at the heart rate thresholds (RV$_{HRT}$). There were no significant mean differences between the RV$_{HRT}$ (9.9 ± 0.6 km·h$^{-1}$) or VT (10.4 ± 0.5 km·h$^{-1}$). Thus, the authors (64) concluded that the mathematical model that had been used to estimate the PWC$_{HRT}$ could be applied to treadmill running to determine the RV$_{HRT}$ and the RV$_{HRT}$ may provide submaximal
protocol for estimating the VT. Vobejda et al. (102) suggested that heart rate measures could be used estimate to estimate the maximal lactate steady state. Specifically, the authors (102) found that the maximal constant heart rate (HRMC) was not different from the heart rate at the maximal lactate steady sate. A recent study by Mielke et al. (70) showed that the mathematical model used to estimate CP could be applied to HR to estimate the CHR. Furthermore, the non-significant difference between CHR and RCPHR suggested that the CHR may provide a new method for estimating the RCP and, thus, the demarcation of the heavy and severe exercise intensity domains, without the need to measure expired gas samples (70).

5) Physiological Responses During Continuous Exercise at a Constant Heart Rate

Kinderman et al. (53)

This study examined differences in the physiological responses during continuous treadmill runs at the heart rate or velocity associated with the anaerobic threshold. Seven cross-country skiers (mean age ± SD, 20.9 ± 1.7 years) performed an incremental test to exhaustion on a treadmill. Oxygen consumptions and heart rate were recorded continuously and blood lactate samples were collected every three min during the test and during the three min of recovery. The anaerobic threshold was defined as the point where blood lactate levels reached 4 mmol·l⁻¹. Linear regression was used to determine the speed, oxygen intake, and heart rate associated with lactate levels of 4 mmol·l⁻¹. The subjects then performed two 30 min treadmill runs. One run was performed at the heart rate associated with the anaerobic threshold, while the other was performed at the speed associated with this threshold. The anaerobic threshold occurred at 84% of the peak
velocity and 91% of heart rate peak during the incremental test. During the constant heart rate runs, the velocity was continuously reduced and oxygen consumption and blood lactate concentrations decreased. At a constant velocity, however, there was a gradual increase in heart rate throughout the test, but lactate remained stable at 4 mmol·l⁻¹. The authors concluded that velocity must be reduced to maintain a constant HR associated with the anaerobic threshold. In addition, the authors suggested that HR serve as the regulatory parameter for endurance training at intensities corresponding to a blood lactate concentration of 4 mmol·l⁻¹.

Mielke (69)

The purpose of this study was to examine the \( \dot{V}O_2 \), EMG, and MMG responses during continuous cycle ergometry at, above, and below the CHR. Ten moderately trained women (mean ± SD; age = 21.4 ± 2.5 years; \( \dot{V}O_2 \text{peak} = 40.5 ± 4.4 \text{ mL·kg}^{-1}·\text{min}^{-1} \)) performed an incremental test to exhaustion on a cycle ergometer. The CHR, CP, and the HR associated with CP (CP\(_{HR} \)) were determined from four randomly ordered rides to exhaustion. The power outputs were selected to elicit fatigue within 8 to 20 min. Heart rate was recorded every 5 s. The total number of heartbeats (HB\(_{lim} \)) was calculated as the product of the mean HR (b·min⁻¹) and time to exhaustion (T\(_{lim} \)). In addition, the product of the power output and T\(_{lim} \) were used to calculate the total work (W\(_{lim} \)) for each of the exhaustive rides. The HB\(_{lim} \) or W\(_{lim} \) was plotted against T\(_{lim} \) for each of the four power outputs and the slope coefficients of the linear HB\(_{lim} \) and W\(_{lim} \) versus T\(_{lim} \) relationships were defined as the CHR and CP, respectively. Heart rate values from the incremental test were plotted against power output values, and the regression equation derived was used to determine the CPHR. The subjects then performed three, randomly ordered,
continuous rides to exhaustion at CHR, CHR-5 b·min⁻¹, and CHR+5 b·min⁻¹. Oxygen consumption, EMG amplitude, EMG mean power frequency (MPF), MMG amplitude, and MMG MPF were recorded during each ride. The CHR (175 ± 11 b·min⁻¹; 92.6 ± 2.8% HRmax) was higher than CPHR (156 ± 11 b·min⁻¹ 83.0 ± 4.0% HRmax). The power output was continuously decreased to maintain the HR during the continuous rides at CHR, CHR-5, and CHR+5. In addition, VO₂ responses tracked power output. The EMG amplitude decreased and EMG MPF increased over time at all three intensities. The MMG amplitude increased at CHR and CHR-5, but did not change at CHR+5. The MMG MPF did not change at CHR and CHR-5, but increased at CHR+5. The time to exhaustion was 37.2 ± 20.3, 51.6 ± 16.8, 23.9 ± 16.6 min for the continuous rides at CHR, CHR-5, and CHR+5, respectively. There were significant increases in the rating of perceived exertion during the exhaustive, constant HR rides. The author found that the EMG amplitude and EMG MPF responses did not reflect fatigue, while the patterns of response for RPE, MMG amplitude and MMG MPF were consistent with those observed during fatiguing exercise. The authors concluded that the CHR test overestimated the intensity that could be maintained for an extended period of time, but suggested that CHR-5 may represent the demarcation of heavy and severe exercise.

Summary

Physiological responses are typically reported during continuous exercise at a constant power output or velocity. A few studies (53, 69), however, have examined responses during exercise at a constant heart rate. These studies (53, 69) have shown that there are dissociations between the power output/velocity and the physiological measures. For
example, Kinderman et al. (53) examined differences in the physiological responses during continuous treadmill runs at the heart rate or velocity associated with the anaerobic threshold. During the constant heart rate runs, the velocity was continuously reduced and oxygen consumption and blood lactate concentrations decreased. At a constant velocity, however, there was a gradual increase in heart rate throughout the test, but lactate remained stable. The authors (53) showed that velocity must be reduced to maintain a constant HR and that there is a dissociation between HR and other physiological responses (i.e., \( \dot{V}O_2 \) and blood lactate concentration). In addition, Mielke (69) examined the \( \dot{V}O_2 \), EMG, and rating of perceived exertion responses during continuous cycle ergometry at, above, and below the CHR. During the constant heart rate rides, power output was continuously decreased to maintain the HR and \( \dot{V}O_2 \) responses tracked power output. There was, however, an increase in RPE. In addition, the EMG amplitude decreased, while EMG MPF increased over time at all three intensities. The author (69) showed that during constant HR exercise, power output, \( \dot{V}O_2 \), EMG amplitude and EMG mean power frequency (MPF) did not reflect fatigue. It was suggested that a HR 5 bpm below the CHR may represent the demarcation of heavy and severe exercise. These investigations (53, 69) demonstrated that there are differences in the physiological responses when exercise is performed at a constant HR compared to exercise performed at a constant power output or a constant velocity. It has been shown that the power output and velocity must be reduced to maintain a constant HR and there is a dissociation between HR and metabolic responses. Furthermore, the study by Mielke (69) suggested that during constant HR exercise, neuromuscular responses may not be used to describe peripheral fatigue.
6) Critical power, Critical Velocity, Heart Rate and Performance

6.1 The Effects of Endurance Training on CP

Jenkins and Quigley (50)

The purposes of this study were to: 1) examine CP as a measure of endurance ability; 2) monitor the effects of eight weeks of endurance training at CP on the slope (CP) and y-intercept (AWC); and 3) examine whether an increase in CP resulted in an increased ability to exercise at a higher intensity. Eighteen physically active untrained male cyclists were divided into two groups, an experimental group (N = 12, mean ± SD, age = 19.1 ± 0.8 yr, height = 1.75 ± 0.09 m, weight = 72.9 ± 6.4 kg) and a control group (N = 6, age = 18.8 ± 0.7 yr, height = 1.76 ± 0.19 m, weight = 70.7 ± 4.6 kg). An incremental cycle ergometer test to exhaustion was used to determine \( \dot{V}O_2 \) max. CP was determined from three constant work rate rides to exhaustion (270, 330, and 390 W). Each subject then completed a 40-min continuous cycle ergometry test at CP. If the subject could not maintain the power output at CP, power was reduced (6-W increments) as necessary to allow for the subjects to continue cycling. The experimental group undertook an eight-week endurance-training program (30 – 40 min at CP) while the control group did no training. Following the eight weeks of training or rest, each group performed an incremental test to exhaustion, CP post-test, and a 40 min ride at CP. As a result of endurance training, there was a 31% increase in CP (196 ± 40.9 W to 255 ± 28.4 W) and an 8.5% increase in \( \dot{V}O_2 \) max (49.2 ± 7.8 ml·l⁻¹·min⁻¹ to 53.4 ± 6.4 ml·l⁻¹·min⁻¹). Mean power output for the experimental group increased 28% from pre to post training for the 40 min endurance test (190 ± 34.5 W to 242 ± 34.9, P < 0.001). The 40 min CP test indicated CP was overestimated (6%) for both the experimental and control group.
There was a significant increase in the slope but not the y-intercept of the CP function (P < 0.01). The results of this study indicate the CP function is sensitive to endurance training and that there is a strong relationship between endurance performance and CP.

6.2 Relationship Between Critical Velocity and Running Performance

Florence and Weir (30)

The purpose of this study was to examine the relationship between CV and marathon running performance. Six male and six female trained runners (mean age ± SD; 29 ± 4 years) performed an incremental treadmill test to exhaustion was completed to determine the gas exchange threshold and \( \dot{V}O_2 \text{peak} \). The CV was determined from four randomly ordered treadmill runs at velocities ranging from 3.6 to 6.0 m·s\(^{-1}\). The CV was defined as slope of the total distance versus time to exhaustion relationship. All of the subjects then completed the New York City Marathon and marathon time (MT) was recorded. Critical velocity was more highly correlated with MT (\( r^2 = 0.76 \)) than \( \dot{V}O_2 \text{peak} \) (\( r^2 = 0.51 \)) or GET (\( r^2 = 0.28 \)). The stepwise multiple linear regression analysis included CV and GET in the prediction equation, but excluded \( \dot{V}O_2 \text{peak} \) in the equation to predict MT. In addition, CV (4.43 ± 0.48 m·s\(^{-1}\)) was significantly higher than the speed associated with the GET (4.04 ± 0.48 m·s\(^{-1}\)) and both CV and the speed associated with the GET were greater than marathon speed (3.07 ± 0.35 m·s\(^{-1}\)). The authors suggested that the CV test provides a useful measure for assessing endurance capabilities and predicting marathon performance.
Kolbe et al. (55)

The purpose of this study was to describe the relationship between CV and track-running performance in events ranging from 40 m to 21.1 km. Seventeen long-distance runners (mean ± SD; age = 31.7 ± 7.3 years; $\dot{V}O_2$ max = 59.2 ± 4.6 km·hr\(^{-1}\) mL·kg\(^{-1}\)·min\(^{-1}\) ) performed an incremental treadmill test to exhaustion to determine $\dot{V}O_2$ max. Critical velocity was determined from six exhaustive, constant velocity, treadmill runs at speeds ranging from 17 to 25 km·hr\(^{-1}\). The subjects then completed timed runs on the track for 40 m, 1 km, 10 km, and 21.1 km. The mean CV was 18.5 ± 1.6 km·hr\(^{-1}\). The 40-m times ranged from 5.57 to 6.95 s, the 1-km times ranged from 2.76 to 3.92 min, the 10-km time ranged from 30.72 to 42.03 min, and the 21.1-km times ranged from 67.00 to 95.75 min.

Critical velocity and $\dot{V}O_2$ max were highly correlated (r = 0.77). There were significant correlations between CV and running time for 1 km (r = -0.75), 10 km (r = 0.85), and 21.1 km (-0.79). In addition, CV accounted for 72% of the variance in 10 km running time. The authors concluded that, although the CV test is repeatable and significantly correlated with $\dot{V}O_2$ max and running times, the fact that greater than 28% of the variability in running time could not be predicted by CV suggested that it may not provide an accurate estimate of running performance.

6.3 Heart Rate and Performance

Boulay et al. (13)

The purposes of this study were to: (1) determine if the HR associated with the ventilatory threshold (VT) could be sustained during continuous cycle ergometry and (2) observe the pulmonary ventilation, power output, and blood lactate responses during the
continuous exercise test at a constant HR. Fifteen males (mean age ± SD; 23 ± 3 years) completed a maximal incremental cycle ergometer test for the determination of the VT, the HR associated with the VT, and the onset of blood lactate (OBLA). The subjects then performed a 90-min cycling test at a HR 5 b·min\(^{-1}\) lower than that observed at VT. The power output was continuously adjusted to maintain the HR. The first and last 10 min of the test were not included in the analyses. The HR values were stable (179 b·min\(^{-1}\)) from 20 to 80 min. The \(\dot{V}O_2\) decreased from 83% of \(\dot{V}O_2\) max at 20 min to 73% of \(\dot{V}O_2\) max at 80 min, but remained unchanged from 30 to 80 min. During the test, power output decreased continuously and was never equal to the power output associated with VT. In addition, there were gradual decreases in blood lactate concentration and pulmonary ventilation after the first 20 min of the continuous cycling test. The authors suggested that a HR 5 b·min\(^{-1}\) lower than that associated with the VT represented an intensity that could be sustained for a prolonged period of time. In addition, based on the continual decreases in power output, blood lactate concentration, and ventilation, the authors concluded that these variables could not be used to determine a sustainable exercise intensity.

Lucia et al. (60)

The purpose of this study was to determine the stability of target heart rate (HR) values corresponding to the lactate threshold (LT), VT, and RCP in cyclist throughout a training season. Thirteen professional road cyclists (mean age ± SD; age = 24 ± 2 years) were tested during the “active” rest (fall: November), precompetition (winter: January), and competition (spring: May) periods of the sports season. Each subject completed an incremental cycle ergometer test to exhaustion at the beginning of each training period to determine the LT, VT, and RCP as well as the heart rate associated with each threshold.
There were significant improvements in the power output associated with the LT, VT, and RCP. The target HR associated with each threshold (HR at LT: 154 ± 3, 152 ± 3, and 154 ± 2 $\text{b·min}^{-1}$; HR at VT: 155 ± 3, 156 ± 3, and 159 ± 3 $\text{b·min}^{-1}$; HR at RCP: 178 ± 2, 173 ± 3, and 176 ± 2 $\text{b·min}^{-1}$ during rest, precompetition, and competition periods, respectively), however, was relatively unchanged. Specifically, the HR at RCP during the rest period was significantly higher than the precompetition or competition periods. No other differences in HR between training periods were observed. The results indicated that the HR associated with the LT, VT, and RCP remained relatively stable, despite significant increases in the power output associated with each threshold. The authors concluded that a single incremental test performed at the beginning of the training season could be used to prescribe training intensities based on HR in elite cyclists.

Summary

Critical power has been used to measure endurance ability (50), monitor training adaptations (30), and design training protocols (Lucia et al. 2000). For example, Jenkins and Quigley (50) found that CP was significantly increased after an eight-week endurance training protocol. In addition, the authors (50) found a significant increase in the power output that could be maintained for 40 min. Thus, Jenkins and Quigley (50) showed that the CP function is sensitive to endurance training and that there is a strong relationship between endurance performance and CP.

The CP and CV concept has also been used to predict endurance performance. For example, Florence and Weir (30) reported a significant relationship between CV and marathon running performance. Kolbe et al. (55), however, reported that although CV
and \( \dot{V}O_2 \) max were highly correlated and there were significant correlations between CV and running time for endurance races, the fact that greater than 28% of the variability in running time could not be predicted by CV suggested that it may not provide an accurate estimate of running performance.

Other studies (13, 60) have used HR to examine endurance exercise and prescribe training intensities. Specifically, Boulay et al. (13) examined the sustainability of a HR threshold and the physiological responses during constant HR exercise. It was suggested (13) that a HR 5 b·min\(^{-1}\) lower than that associated with the VT represented an intensity that could be sustained for a prolonged period of time. In addition, based on the continual decreases in power output, blood lactate concentration, and ventilation during the constant HR ride, the authors concluded that these variables could not be used to determine a sustainable exercise intensity. In addition, Lucia et al. (60) showed that the HR associated with the lactate threshold, ventilatory threshold, and respiratory compensation point remained relatively stable during a training season, despite significant increases in the power output associated with each threshold. The results of these studies (13, 60) suggested that HR could be used throughout the course of a training season to prescribe a training intensity.
Chapter III

METHODS

Experimental Design

This study involved a total of eight visits, separated by 24 – 48 hours. During the first visit, the gas exchange threshold (GET), peak oxygen consumption rate ($\dot{V}O_2^{\text{peak}}$), and velocity associated with $\dot{V}O_2$ peak ($v\dot{V}O_2^{\text{peak}}$), as well as the RPE ($\text{RPE}^{\text{peak}}$), HR ($\text{HR}^{\text{peak}}$), EMG AMP ($\text{AMP}^{\text{peak}}$), and EMG MPF ($\text{MPF}^{\text{peak}}$) at $\dot{V}O_2^{\text{peak}}$ were determined from an incremental treadmill test to exhaustion. Heart rate was measured during four, randomly ordered, exhaustive, constant velocity runs during visits two through five. The mathematical model that has been used to estimate CP and CV was applied to the heart rate data to derive the CHR. During visits six through eight, the subjects performed three, randomly ordered, constant HR runs at CHR-5, CHR, and CHR+5. During each of the constant HR runs, $\dot{V}O_2$, velocity, HR, RPE, EMG AMP, and EMG MPF were measured.

Sample Population, Inclusion, and Exclusion Criteria

Thirteen moderately trained runners (7 men and 6 women; mean ± SD age = 23 ± 3 years, height = 175 ± 8 cm, weight = 71 ± 12 kg) completed this study. Moderately trained was defined as running a total of approximately 16 to 48 km·wk$^{-1}$ most weeks during the previous 6-months. All subjects were instructed to avoid exercising the day prior to each test. The subjects had no known cardiovascular, pulmonary, metabolic, muscular and/or coronary heart disease. This study was approved by the University Institutional Review Board for Human Subjects and all subjects complete a health history questionnaire and signed a written informed consent document before testing.
Thirteen subjects completed each of the three constant HR runs. Due to equipment malfunctions, however, complete data sets for all variables were not available for 4 of the 13 subjects (1 at CHR-5, 2 at CHR, and 1 at CHR+5) and these subjects were excluded from the regression analyses. At CHR+5, an additional 3 subjects were excluded because the $T_{\text{lim}}$ values (0.82 – 2.71 min) did not provide enough data points for the regression analyses. Thus, 12 subjects at CHR-5, 11 subjects at CHR, and 9 subjects at CHR+5 were included in the regression analyses.

**Electromyographic Measurements**

The EMG signals were measured for the vastus lateralis (VL) on the dominant leg during the incremental test and constant heart rate runs. Prior to electrode placement, the skin at each site was shaved, carefully abraded, and cleaned with alcohol. A bipolar surface electrode (circular 24 mm, Kendall disposable EMG electrodes, Covidien LTD; Gosport Hampshire, UK) arrangement (2.0 cm center-to-center) was placed based on the recommendations from the SENIAM Project for EMG electrodes placement (41).

Specifically, a reference line was drawn over the vastus lateralis (VL), one-third of the distance between the lateral superior border of the patella and the anterior superior iliac spine. In addition, the electrode-placement site was located 5 cm lateral to the reference line so that the electrodes were over the VL muscle (62). A goniometer (Smith & Nephew Rolyan, Inc., Menomonee Falls, WI) was used to orient the EMG electrodes at a 20° angle to the reference line to approximate the pennation angle of the muscle fibers for the VL (1). The EMG signal was amplified (gain: $\times 1,000$) using differential amplifiers (Free EMG 300, BTS, Milan, Italy, bandwidth = 10-500 Hz).
Signal Processing

The raw EMG signals were digitized at 1,000 Hz, stored in a personal computer (MacBook Pro OSX, version 10.6.8, Apple Inc., Cuperino, CA) for subsequent analysis and processed with a custom program written with LabVIEW programming software (version 7.1, National Instruments, Austin, TX). The EMG signals were bandpass-filtered (fourth-order Butterworth) at 10-500 Hz. Continuous 10 s epochs for the EMG AMP (microvolts root mean square, µVrms) and EMG MPF (MPF in Hz) were calculated. For the MPF analyses, each data segment was processed with a Hamming window and a discrete Fourier transform (DFT) algorithm in accordance with the recommendations of Hermens et al. (41). The MPF was selected to represent the power spectrum on the basis of the recommendations of Hermans et al. (41) and was calculated as described by Kwatny et al. (58) using the following equation:

\[ \text{MPF} = \frac{\sum_{f=f_0}^{f_c} f P(f)}{\sum_{f=f_0}^{f_c} P(f)} \]

where \( f \) is the frequency, \( f_0 \) is 0 Hz, \( f_c \) is the cutoff frequency (i.e., the last frequency in the last summation), and \( P(f) \) is the power density (V\(^2\)/Hz) of the EMG signal (58).

Determination of the GET and Peak Values

Each subject performed an incremental treadmill test to exhaustion (Precor Inc., Bothell, WA USA) to determine the GET, \( \dot{V}O_2 \text{peak} \), \( \dot{V}O_2 \text{peak} \), HR\(_{\text{peak}}\), AMP\(_{\text{peak}}\), MPF\(_{\text{peak}}\), and RPE\(_{\text{peak}}\). Prior to the test, the EMG electrodes were placed as previously described and each subject completed a 3 min warm-up on the treadmill at a velocity of 4.8 km·h\(^{-1}\) and 0% grade, followed by a 3 min passive recovery. Following the warm-up, each subject was fitted with a nose clip and breathed through a 2-way valve (Hans Rudolph
2700 breathing valve, Kansas City, MO, USA). Expired gas samples were collected and analyzed using a calibrated TrueMax 2400 metabolic cart (Parvo Medics, Sandy, UT, USA). The gas analyzers were calibrated with room air and gases of known concentration prior to all testing sessions. The O₂, CO₂, and ventilatory parameters were recorded breath-by-breath and expressed as 20 s averages (89). In addition, HR was recorded with a Polar Heart Rate Monitor (Polar Electro Inc., Lake Success, NY) that was synchronized with the metabolic cart. Heart rate was recorded continuously throughout the test and expressed as both 5 and 20 s averages. The EMG signals were recorded throughout the test and expressed as 10 s averages. Each subject was asked to give a rating of perceived exertion during the last 10 s of each minute using the Borg 6-20 RPE scale (10). The incremental test began at a treadmill velocity of 6.4 km·h⁻¹ and 0% grade. Thereafter, the velocity was increased by 1.6 km·h⁻¹ every 2 min to 14.4 km·h⁻¹ and 0% grade. Following the 14.4 km·h⁻¹ stage, the velocity was no longer increased, however, the treadmill grade was increased by 2% every 2 min until the subject could no longer maintain the running velocity and grasped the handrails to signal exhaustion. The VO₂peak was defined as the highest 20 s average VO₂ value recorded during the test. The velocities performed at 0% grade (6.4 to 14.4 km·h⁻¹), were plotted against VO₂ and the regression equation derived was used to determine the vVO₂peak. The HRpeak was defined as the 20 s average HR value associated with VO₂peak and the RPEpeak was defined as the RPE taken at the end of the last full min completed during the test. The AMPpeak and MPFpeak were the 10 s averages associated with VO₂peak.
The GET was determined using the V-slope method described by Beaver et al. (4). The GET was defined as the $\dot{V}O_2$ value corresponding to the intersection of two linear regression lines derived separately from the data points below and above the breakpoint in the carbon dioxide produced ($\dot{V}CO_2$) versus $\dot{V}O_2$ relationship. The velocities performed at 0% grade (6.4 to 14.4 km·h$^{-1}$), were plotted against $\dot{V}O_2$ and the regression equation derived was used to determine the velocity associated with the GET.

**Determination of CHR and vCHR**

Four, constant velocity, randomly ordered treadmill runs at velocities ranging from 79 – 102% of the $v\dot{V}O_2$ peak were performed on separate days. This range of velocities was selected so that each subject could complete 3 to 20 min of exercise before exhaustion (17). Prior to the start of the run, each subject practiced getting on and off the treadmill at the velocity associated with that run to become familiarized with that velocity. In addition, each subject completed a self-paced walking or jogging 5 min warm-up, followed by 3 min of passive rest. The treadmill was then set to the selected velocity at 0% grade. Timing for each treadmill run began when the subject released the handrails (usually 2 – 3 s after getting on the treadmill) and was terminated when the subject grasped the handrails to signal exhaustion. Heart rate values were continuously monitored and recorded as 5 s averages. For each velocity, the total number of heartbeats ($HB_{lim}$) was calculated as the product of the average 5 s HR ($b\cdot min^{-1}$) and time to exhaustion ($T_{lim}$). For each velocity, the $HB_{lim}$ was plotted as a function of the $T_{lim}$. The CHR was defined as the slope coefficient of the regression line between $HB_{lim}$ and $T_{lim}$ (Figure 2).
The velocity from the incremental test was plotted against HR and the regression equation derived was used to determine the velocity associated with CHR-5 (vCHR-5), CHR (vCHR), and CHR+5 (vCHR+5).

**Constant Heart Rate Runs**

Three, randomly ordered, constant HR treadmill runs were performed at CHR-5, CHR, and CHR+5 to determine the $T_{lim}$ as well as the metabolic ($\dot{V}O_2$), perceptual (RPE), and neuromuscular (EMG AMP and MPF) responses at each intensity. Prior to the run, the EMG electrodes were placed as previously described. Each subject then completed a self-paced walking or jogging 5 min warm-up, followed by 3 min of passive rest. For the CHR-5, CHR, and CHR+5 runs, the initial intensities were set at a mean velocity ± SD of $88 \pm 6\% (76 – 98\%)$, $91 \pm 5\% (81 – 100\%)$, and $97 \pm 3\% (92 – 102\%)$ of $\dot{V}O_2_{peak}$ respectively. This range of velocities was selected so that the subject reached CHR-5, CHR, and CHR+5 within approximately 2 to 5 min. Two subjects at CHR-5, 3 subjects at CHR, and 2 subjects at CHR+5 did not reach the selected HR within 5 min and the velocity was increased by 0.32 to 0.80 km·h$^{-1}$. For these subjects, the selected HR was reached within 5.22 to 7.67 min. All of the runs were performed at a 0% grade. During the runs, the velocity was adjusted to ensure that the selected HR at which each subject exercised remained constant. Pilot data indicated that a decrease of 0.32 km·h$^{-1}$ resulted in a 1 b·min$^{-1}$ reduction in HR. During each run, the $\dot{V}O_2$ and HR values were collected as previously described and recorded as 20 s averages. Ratings of perceived exertion were taken in the last 10 s of each minute, and the EMG signals were collected as previously described and recorded as 10 s averages. In this study, an extended period of time was defined as 60 min. Therefore, the test was terminated when the subject completed 60 min
or reached exhaustion (grasped the handrails) at the selected HR intensity. The $T_{\text{lim}}$ was recorded as the total time the subject ran at the selected HR. Thus, the time to reach CHR-5, CHR, or CHR+5 was subtracted from the total time.

**Determination of Metabolic Efficiency**

The $\dot{V}O_2$ values (L·min$^{-1}$) from the incremental test were plotted against velocity (km·h$^{-1}$) and the regression equation derived was used to determine the $\dot{V}O_2$ ($\dot{V}O_2_{\text{EST}}$) associated with the velocity at the termination of exercise (END velocity) at CHR-5, CHR, and CHR+5. The estimated metabolic efficiency ($E_{\text{EST}}$) was determined as the $\dot{V}O_2_{\text{EST}}$ / END velocity ratio for each intensity. The actual metabolic efficiency ($E_{\text{END}}$) at the end of each constant HR run was calculated as the the $\dot{V}O_2$ measured at the termination of exercise ($\dot{V}O_2_{\text{END}}$) and END velocity ratio ($\dot{V}O_2_{\text{END}}$/END velocity).

**Statistical Analyses**

Differences between the $E_{\text{EST}}$ and $E_{\text{END}}$ for CHR-5, CHR, and CHR+5 were examined using separate paired samples $t$-tests. An alpha level of $p \leq 0.05$ was considered statistically significant for all analyses.

The individual and mean responses for each variable ($\dot{V}O_2$, velocity, HR, RPE, EMG AMP, EMG MPF) were examined for each of the constant HR runs at CHR-5, CHR, and CHR+5. Each variable recorded during the constant HR runs was normalized as a percentage of the value at $\dot{V}O_2$ peak (i.e., each value was divided by its value at $\dot{V}O_2$ peak and multiplied by 100 to get a percentage). Because each subject had a different time to exhaustion, time was normalized as a percentage of $T_{\text{lim}}$ and 11 data points (0, 10, 20, 30, 40, 50, 60, 70, 80, 90, and 100% of $T_{\text{lim}}$) were used for all analyses.
The relationships for the normalized \( \dot{V}O_2 \), velocity, HR, RPE, EMG AMP, and EMG MPF versus normalized time were examined using polynomial regression models (linear and quadratic). The statistical significance \((p \leq 0.05)\) for the increment in the proportion of the variance that would be accounted for by a higher-degree polynomial was determined using the following \(F\)-test (76):

\[
F = \frac{(R_2^2 - R_1^2)/K_2 - K_1}{(1 - R_2^2)/n - K_2 - 1}
\]

where \(n\) is the number of data points, \(K_2\) is the number of predictors from the larger \(R^2\), and \(K_1\) is the number of predictors from the smaller \(R^2\). All statistical analyses were performed with Statistical Package for the Social Sciences software (v.19.0. IMB SPSS Inc., Chicago, Illinois, USA).
CHAPTER IV

ANALYSIS OF RESULTS

RESULTS

Incremental Test and Constant Velocity Runs

The mean ± SD and range of peak values for each variable (GET, $\dot{V}O_2$peak, $v\dot{V}O_2$peak, HRpeak, RPEpeak, AMPpeak, and MPFpeak) from the incremental test are included in Tables 1 and 2. The Tlim and velocities for the constant velocity runs used to estimate the CHR values ranged from 5.99 to 22.25 min and 79-102% $v\dot{V}O_2$peak, respectively. The $r^2$ values for the HBlim versus Tlim relationship ranged from 0.995 – 1.000. The mean CHR-5, CHR, and CHR+5 occurred at 171 ± 8 b·min⁻¹, 175 ± 8 b·min⁻¹, and 178 ± 6 b·min⁻¹, respectively, and represented a mean of 88 ± 3%, 91 ± 3%, and 94 ± 3% of HRpeak, respectively. The vCHR-5, vCHR, and vCHR+5 were 78 ± 7%, 82 ± 7%, and 88 ± 7% of $v\dot{V}O_2$peak, respectively (Table 3).

Constant HR Runs at CHR-5, CHR, and CHR+5

CHR-5 (n=12)

During the CHR-5 run, the subjects reached the selected HR within 1.83 – 5.60 min (3.03 ± 1.22 min) and the Tlim was 56.97 ± 1.23 min (Table 4). The mean percent changes (%Δ) from the time CHR-5 was reached to the end of the run for $\dot{V}O_2$, velocity, RPE, EMG AMP, and EMG MPF were -14 ± 7%, -23 ± 4%, 11 ± 21%, -12 ± 12%, and 5 ± 14%, respectively (Table 5). The results of the polynomial regression analyses for the mean responses during the continuous runs at CHR-5 indicated that there was no change in HR, but quadratic decreases in velocity and $\dot{V}O_2$ (Figure 3a). There was, however, a
quadratic increase in RPE. In addition, there was a linear decrease in EMG AMP and a linear increase in EMG MPF (Figure 3a).

The polynomial regression analyses for the individual responses at CHR-5 indicated no changes in HR, quadratic decreases in velocity, and linear (n= 6) or quadratic (n= 6) decreases in \( \dot{V}O_2 \) for all 12 of the subjects (Figure 3b). For RPE, there were quadratic increases for 2, linear increases for 4, a quadratic decrease for 1, and no changes for 5 of the 12 subject. In addition, for EMG AMP, there were quadratic decreases for 3, linear decreases for 2, a linear increase for 1, and no change for 6 of the 12 subjects. For EMG MPF, there were quadratic decreases for 3, linear increases for 4, and no changes for 5 of the 12 subjects (Figure 3b).

The \( \dot{V}O_2_{END} \) (mean ± SD = 2.392 ± 0.296 L·min\(^{-1}\)) was significantly greater (p = 0.021) than the \( \dot{V}O_2_{EST} \) (2.234 ± 0.255 L·min\(^{-1}\)) at the end of the run at CHR-5. The \( E_{END} \) (mean ± SD = 0.224 ± 0.041 L·min\(^{-1}\)/km·h\(^{-1}\)) ratio was significantly greater (p = 0.014) than the \( E_{EST} \) (0.209 ± 0.029 L·min\(^{-1}\)/km·h\(^{-1}\)) ratio for CHR-5 (Table 6).

CHR (n= 11)

During the CHR run the subjects reached the selected HR within 3.25 – 6.57 min (4.34 ± 1.06 min) and the \( T_{lim} \) was 48.37 ± 11.04 min (Table 4). The \( %\Delta \) from the time CHR was reached to the end of the run for \( \dot{V}O_2 \), velocity, RPE, EMG AMP, and EMG MPF were -14 ± 6%, -23 ± 6%, 25 ± 21%, 14 ± 31%, and -2 ± 19%, respectively (Table 7). The polynomial regression analyses for the mean responses during the continuous runs at CHR indicated there was no change in HR, but quadratic decreases in velocity and
\( \dot{V}O_2 \) (Figure 4a). In addition, there were linear increases in RPE and EMG AMP, but no change in EMG MPF (Figure 4a).

The polynomial regression analyses for the individual responses at CHR indicated there were no changes in HR, quadratic decreases in velocity, and linear (n= 5) or quadratic (n= 6) decreases in \( \dot{V}O_2 \) for all 11 subjects. For RPE, there were quadratic increases for 6, linear increases for 3, a linear decrease for 1, and no change for 1 of the 11 subjects. In addition, for EMG AMP, there was a quadratic increase for 1, linear increases for 4, quadratic decreases for 1, linear decreases for 2, and no changes for 3 of the 11 subjects. For EMG MPF, there was a quadratic increase for 1, linear increases for 2, linear decreases for 3, and no changes for 5 of the 11 subjects (Figure 4b).

The \( \dot{V}O_2 \) END (mean ± SD = 2.627 ± 0.643 L·min\(^{-1}\)) was significantly greater (p < 0.001) than the \( \dot{V}O_2 \) EST (2.394 ± 0.521 L·min\(^{-1}\)) at the end of the run at CHR. The E END (mean ± SD = 0.240 ± 0.047L·min\(^{-1}\)/km·h\(^{-1}\)) ratio was significantly greater (p <0.001) than the E EST (0. 0.219 ± 0.041 L·min\(^{-1}\)/km·h\(^{-1}\)) ratio for CHR (Table 8).

CHR+5 (n=9)

During the CHR-5 run, the subjects reached the selected HR within 2.47 – 7.67 min (4.45 ± 1.52 min) and the T lim was 20.11 ± 16.08 min (Table 4). The mean %Δ from the time CHR+5 was reached to the end of the run for \( \dot{V}O_2 \), velocity, RPE, EMG AMP, and EMG MPF were -9 ± 10%, -16 ± 10%, 19 ± 17%, -2 ± 13%, and -7 ± 15%, respectively (Table 9). The polynomial regression analyses for the mean responses during the continuous runs at CHR+5 indicated there was no change in HR, but quadratic
decreases in velocity and EMG AMP, and a linear decrease in $\dot{V}O_2$ and EMG MPF (Figure 5a). There were, however, linear increases in RPE (Figure 5a).

The polynomial regression analyses for the individual responses at CHR+5 indicated there were no changes in HR over time for any of the 11 subjects. For velocity, there were quadratic decreases for 6, linear decreases for 2, and no change for 1 of the 9 subjects. The $\dot{V}O_2$ responses indicated quadratic decreases for 4, linear decreases for 3, and no changes for 2 of the 9 subjects. For RPE, there were quadratic increases for 3, linear increases for 3, and no changes for 3 of the 9 subjects. In addition, for EMG AMP, there were quadratic increases for 2, linear increases for 3, linear decreases for 3, and no change for 1 of the 9 subjects. The EMG MPF indicated a quadratic decrease for 1, linear decrease for 1, and no changes for 7 of the 9 subjects (Figure 5b).

The $\dot{V}O_2_{END}$ (mean ± SD = 3.073 ± 0.492 L·min$^{-1}$) was significantly greater (p = 0.037) than the $\dot{V}O_2_{EST}$ (2.889 ± 0.608 L·min$^{-1}$) at the end of the run at CHR+5. The $E_{END}$ (mean ± SD = 0.244 ± 0.038 L·min$^{-1}$/km·h$^{-1}$) ratio was significantly greater (p= 0.028) than the $E_{EST}$ (0.228 ± 0.038 L·min$^{-1}$/km·h$^{-1}$) ratio for CHR+5 (Table 10).
DISCUSSION

Applicability of the CHR Model to Treadmill Running

The mathematical model used to estimate CP (72, 73) has been applied (70) to HR measurements during cycle ergometry to derive a fatigue threshold called the critical heart rate (CHR). One purpose of the present study was to determine if the CHR model for cycle ergometry (70) could be applied to treadmill running. During treadmill running, the total number of heartbeats (HB_{lim}) were plotted as a function of the T_{lim} values for 4 constant velocity runs to exhaustion. The HB_{lim} versus T_{lim} relationship ($r^2 = 0.995 – 1.000$) was described by the linear equation: $HB_{lim} = a + CHR(T_{lim})$. These $r^2$ values were similar to those previously reported ($r^2 = 0.966 – 1.000$) during cycle ergometry (70). The highly linear relationship between HB_{lim} and T_{lim} in the present study indicated that the mathematical model used to derive CHR during cycle ergometry (Mielke 2009) was also applicable to treadmill running. Thus, theoretically, the treadmill based CHR estimates in the present study represented the maximal HR that could be maintained for an extended period of time without fatigue (70).

The Sustainability of the CHR

A sustainable bout of continuous exercise has been operationally defined by T_{lim} values of 30 to 60 min (16, 20, 100). In the present study, the T_{lim} values at CHR-5, CHR, and CHR+5 reflected the total time the subjects were able to maintain the selected HR, minus the time to reach that HR (Table 3). The current findings showed that all 12 subjects were able to complete the 60 min work bout (mean $T_{lim} \pm SD = 56.97 \pm 1.23$ min) at CHR-5. At CHR ($T_{lim} = 48.37 \pm 11.04$), all but 1 of the 11 subjects completed at
least 30 min, and 7 subjects completed the 60 min work bout. At CHR+5, 1 of the 9 subjects was able to complete the 60 min work bout, however, the other 8 subjects exhausted prior to 30 min ($T_{\text{lim}} = 20.11 \pm 16.08$). These $T_{\text{lim}}$ values were similar to those previously reported for CHR-5 (51.6 ± 16.8 min), CHR (37.2 ± 20.3 min), and CHR+5 (23.9 ± 16.6 min) for cycle ergometry (69). Thus, the results of the present study and those of Mielke (69) indicated that on average, HR values that were less than or equal to CHR for running or cycling, represented sustainable (minimum of 30 to 60 min) intensities, while CHR+5 did not. These findings suggest that the CHR represents the highest, sustainable heart rate during both running and cycling.

The CHR model was adapted from the CP test for cycle ergometry (73) and CV test for treadmill running (48, 77). Previous studies (14, 17, 45, 47, 50, 66, 77), however, have reported $T_{\text{lim}}$ values at CP and CV that ranged from 14 to 33 min, and have suggested (45, 47, 77) that CP and CV overestimated a sustainable intensity by 15 to 17%. It is possible that the greater $T_{\text{lim}}$ values at CHR during cycling (69) and running (37.2 ± 20.3 and 48.37 ± 11.04 min, respectively) than those previously reported for CP and CV (14, 17, 45, 47, 50, 66, 77) were due to the use of HR in the determination of the fatigue threshold, rather than the power or velocity versus $T_{\text{lim}}$ relationships. Thus, the present findings, in conjunction with those of Mielke (69), suggested that a threshold associated with the highest sustainable exercise intensity should be based on the responses of a physiological parameter, such as HR, rather than a specific power output or velocity.

Generally, non-sustainable ($T_{\text{lim}} < \sim 30$ min), fatiguing exercise intensities are characterized by specific $\dot{V}O_2$ and HR responses that include the presence of a $\dot{V}O_2$ slow
component that drives $\dot{V}O_2$ to $\dot{V}O_2_{\text{peak}}$ (31, 43) and HR increases to HR$_{\text{peak}}$ (7, 8, 9). There is evidence (8, 9, 22, 23, 94), however, that exhaustion can occur at power outputs or velocities slightly greater (~5 to 8%) than CP (93) and CV (8, 22) without the attainment of $\dot{V}O_2_{\text{peak}}$ (end $\dot{V}O_2 = 73$ to 93% $\dot{V}O_2_{\text{peak}}$), although HR still reaches HR$_{\text{peak}}$. In the present study, when exercise was performed at a constant HR that was 3% above CHR (CHR+5 = 94 ± 3% HR$_{\text{peak}}$), exhaustion occurred within 30 min for most (89%) subjects (T$_{\text{lim}} = 20.11 ± 16.08$ min) and the $\dot{V}O_2$ at exhaustion (85 ± 10% $\dot{V}O_2_{\text{peak}}$) was less than $\dot{V}O_2_{\text{peak}}$ for all but 1 subject. Thus, the $\dot{V}O_2$ responses at exhaustion for exercise performed 5 b·min$^{-1}$ above CHR were consistent with exercise intensities performed at a constant power output or velocity that was slightly greater than CP or CV, respectively, and indicated that exhaustion does not necessitate the attainment of $\dot{V}O_2_{\text{peak}}$. The results of the present study also showed that exhaustion can occur at a HR less than HR$_{\text{peak}}$. These findings indicated that, when running at a constant HR, neither $\dot{V}O_2$ nor HR could be used as an indicator of exhaustion.

Although exhaustion could not be identified by the attainment of $\dot{V}O_2_{\text{peak}}$ or HR$_{\text{peak}}$ in the present study, the perception of effort was closely related to the early termination of exercise during constant HR running. That is, in general, the RPE reached peak or near peak values for those subjects who exhausted prior to 60 min, but was less than RPE$_{\text{peak}}$ for those who did not. Specifically, after the 60 min work bout at CHR-5, the perception of effort (RPE= 73 ± 16% RPE$_{\text{peak}}$) was less than RPE$_{\text{peak}}$ for 83% of the subjects. Exercise at CHR was perceived as more demanding (RPE= 88 ± 16% RPE$_{\text{peak}}$) and RPE was at or near peak values for all 4 of the 11 subjects who exhausted prior to 60 min and
2 of the subjects who completed the work bout. At CHR+5, however, 8 of the 9 subjects
exhausted within 30 min and the perception of effort was equal to RPE\textsubscript{peak} (98 ± 5%
RPE\textsubscript{peak}). Thus, the results of the present study indicated that, on average, exhaustive
constant HR exercise resulted in a perception of effort at or near RPE\textsubscript{peak}, while exercise
that could be maintained for 60 min did not. These findings suggested that the perception
of effort, but not $\dot{V}O_2$ or HR, can be used to identify exhaustion during continuous
exercise at or above CHR.

**Metabolic, Perceptual, and Neuromuscular Patterns of Responses During Treadmill
Running at a Constant HR**

During treadmill running at CHR-5, CHR, and CHR+5 in the present study, the
velocity was reduced (mean %Δ ± SD = -23 ± 4, -23 ± 6, and -16 ± 10%, respectively) to
maintain the selected HR, and the $\dot{V}O_2$ responses (-14 ± 7, -14 ± 6, and -9 ± 10%,
respectively) tracked the changes in velocity. There were, however, increases in the
perception of effort throughout the runs at CHR-5, CHR, and CHR+5 (11 ± 21, 25 ± 21,
and 19 ± 17%, respectively). Previous studies (13, 53, 69, 99) have reported similar
magnitudes of change for velocity or power output (-11 to -20%), $\dot{V}O_2$ (-12 to -16%), and
RPE (19 to 29%) during continuous running and cycling at a constant HR. Thus, the
present findings supported those of previous studies (13, 53, 69, 99) and indicated that,
during fatiguing constant HR running and cycling, $\dot{V}O_2$ responses tracked velocity and
power output, but were dissociated from HR, which remained stable, and RPE, which
increased.

The dissociations between $\dot{V}O_2$ and HR during fatiguing running and cycling at a
constant HR differed from the typical responses during constant velocity or power output
exercise. Specifically, during constant velocity or power output work bouts, fatigue is associated with an \( \dot{V}O_2 \) drift (a rise in \( \dot{V}O_2 \) over 60 min or more of < 200 ml of \( O_2 \)) during moderate exercise intensities, and a \( \dot{V}O_2 \) slow component (increase in \( \dot{V}O_2 \) of ≥ 200 ml of \( O_2 \)) during heavy and severe exercise intensities, where \( \dot{V}O_2 \) may reach a delayed steady state, increase to exhaustion, or drive \( \dot{V}O_2 \) to \( \dot{V}O_2 \) peak (31, 87, 94). Constant velocity or power output exercise is also associated with a cardiovascular drift that has been attributed to increases in core temperature and/or sympathetic activation and is defined by a gradual rise in HR and the arteriovenous \( O_2 \) difference, a decline in stroke volume (SV), but no change in the cardiac output (HR x SV) (24). The cardiac output during continuous exercise is dependent upon the intensity (metabolic demand) of the exercise, where a decrease in exercise intensity is associated with a decrease in cardiac output (104). The continuous decrease in the velocity and metabolic demand and, thus, cardiac output, during constant HR running in the present study likely reflected decreases in SV and the arteriovenous \( O_2 \) difference. Unlike constant velocity or power output exercise, however, the reduction in SV was not related to increases in HR, because HR was held constant. The increase in the perception of effort during constant HR running in the present study, however, was consistent with RPE responses during constant velocity or power output exercise (7, 34). Thus, the current findings, in conjunction with those of others (13, 53, 69, 99), indicated that RPE responses, but not \( \dot{V}O_2 \) or HR, could be used as an indicator of fatigue during continuous exercise at a constant HR, velocity, or power output. In addition, the decrease in \( \dot{V}O_2 \) throughout the runs at a constant HR suggested that the RPE and fatigue were not related to \( O_2 \) availability or sympathetic activation. It is
possible, however, that the increase in the perception of effort was related to increases in core temperature (24).

Neuromuscular responses including motor unit recruitment, firing rate, and action potential conduction velocity are reflected in the time and frequency domains of the EMG signal and have also been used to characterize fatigue during constant velocity or power output exercise (3, 27, 46, 65). De Luca (27) described changes in the EMG signal as myoelectric manifestations of fatigue that result in a ‘slowing’ of the surface EMG signal, causing an increase in the time, and a decrease in the frequency domains. During fatiguing cycle ergometry exercise, an increase in EMG AMP reflects the fatigue-induced recruitment of additional muscle fibers, increases in firing rate, and/or synchronization (3; 38, 39), while a decrease in EMG MPF reflects a reduction in the muscle fiber action potential conduction velocity and changes in the shape of the waveform (27).

It has been hypothesized (29, 36, 61, 90, 105) that the specific EMG AMP and MPF responses (i.e., increased motor unit recruitment, firing rate, or decreased action potential conduction velocity) to fatiguing constant velocity or power output exercise are the result of the accumulation of metabolites and ions such as lactate, inorganic phosphate, hydrogen (H\(^+\)), ammonium (NH\(_4^+\)), and potassium (K\(^+\)) ions. Previous studies have suggested that the buildup of these metabolites and ions causes decreases in membrane excitability (29), excitation-contraction coupling involving Ca\(^{++}\) release and uptake from the sarcoplasmic reticulum, myofibrillar Ca\(^{++}\) sensitivity for binding with troponin, actin-myosin binding, and ATP production and breakdown (36, 61, 90, 105).

Currently, it is unclear which specific metabolite or ion is responsible for the neuromuscular fatigue responses that are reflected in the EMG signal. Various
hypotheses (5, 6, 21), however, have been developed regarding the contribution of these metabolites and ions to neuromuscular fatigue by examining the exercise intensity at which fatigue-induced changes in EMG AMP and MPF occurs relative to the gas exchange threshold (GET) and respiratory compensation point (RCP). During an incremental exercise test, the non-linear increase in $\dot{V}CO_2$ relative to $\dot{V}O_2$ at the GET reflects the excess CO$_2$ that results from bicarbonate buffering of H$^+$, produced during nonmitochondrial adenosine triphosphate (ATP) turnover (78, 90). Exercise intensities performed at or above the GET have also been shown (18, 63, 106) to result in an increase in the plasma [NH$_4^+$]. The RCP occurs at a higher intensity than the GET, and is defined as the point of dissociation between the ventilatory rate ($\dot{V}_E$) and $\dot{V}CO_2$. Although there is evidence (67) to indicate the ventilatory response at the RCP is related to the break down of the bicarbonate buffering system and the accumulation of H$^+$, the excess H$^+$ does not fully account for the ventilatory response. In fact, McArdle’s patients have shown (75) a breakpoint in the $\dot{V}_E$ versus $\dot{V}CO_2$ relationship during incremental cycling exercise, despite their inability to develop metabolic acidosis. Additional evidence (25, 75) has indicated that the arterial chemoreceptor sensitivity to increased arterial [K$^+$] provides an important stimulus for the increases in $\dot{V}_E$ at the RCP. Although it is unknown which ion (H$^+$, NH$_4^+$, and K$^+$) specifically affects EMG AMP and MPF, Camic et al. (21) hypothesized that the increase in EMG AMP that occurred at a power output $\sim$11% greater than the GET was the result of decreased pH that altered muscle contractility, while the decrease in EMG MPF at a power output $\sim$1% greater than the RCP was due to elevated interstitial and/or arterial [K$^+$] which reduced membrane excitability. In contrast, Bergstrom et al. (6) found that the increases in EMG AMP were
more closely related to the RCP than the GET and hypothesized that muscle activation was more sensitive to the accumulation of interstitial $\text{K}^+$ than $\text{H}^+$. Previous studies (27, 40) have also suggested that the fatigue induced decreases in the frequency domain are associated with the accumulation of $\text{NH}_4^+$ that accompanies the recruitment of fast twitch glycolytic fibers. Taken together, these studies (6, 21, 25, 27, 40, 67, 75) indicated that neuromuscular fatigue is likely a multifactorial process that is not related to any one specific metabolite or ion, but rather an interaction among these factors, where the relative contribution of each metabolite or ion to fatigue is dependent upon the intensity of exercise.

Previous evidence (69) indicated that, during fatiguing constant HR cycle ergometry exercise, EMG AMP and MPF responses were not consistent with those typical of fatigue during a constant power output work bout. Specifically, Mielke (69) reported decreases in EMG AMP and increases in EMG MPF during cycle ergometry at CHR-5, CHR, and CHR+5. Consistent with the findings of Mielke (69), continuous treadmill running at CHR-5 in the present study resulted in decreases in EMG AMP and muscle activation that tracked the decreases in velocity, while EMG MPF and action potential conduction velocity increased throughout the run (Figure 3a). For each subject (n = 12) in the present study, CHR-5 represented a sustainable intensity, and most of the subjects (58%) completed the work bout at a metabolic intensity (mean ± SD $\dot{\text{V}}\text{O}_2 = 72 ± 6\%$ of $\dot{\text{V}}\text{O}_2\text{peak}$) at or below the GET (70 ± 6% of $\dot{\text{V}}\text{O}_2\text{peak}$). Thus, it is possible that the decrease in EMG AMP and muscle activation during continuous running at CHR-5 resulted from a reduction in the metabolic intensity that was great enough to attenuate the accumulation of metabolites and ions, ameliorate the cellular environment, and delay the
onset of neuromuscular fatigue. It is also possible that the increase in EMG MPF during treadmill running at CHR-5 in the present study was related to increases in muscle temperature that occur during sustained dynamic exercise (82, 92). Increases in muscle temperature have been shown to correspond to increases in EMG MPF and action potential conduction velocity (37, 82, 83, 93, 98). For example, the temperature of the quadriceps muscles has been shown to increase from ~33 °C to 39 °C after 10 to 20 min of submaximal exercise (82) and temperature changes of this magnitude have been shown to correspond to increases in EMG MPF (83). It has been suggested that elevated muscle temperature accelerates the opening and closing of voltage-gated Na+ channels, which decreases the rate of depolarization and increases muscle fiber action potential conduction velocity along the sarcolemma (92). Thus, the EMG AMP and MPF responses at CHR-5 during treadmill running in the present study were consistent with the findings of Mielke et al. (69) and indicated that unlike constant power output or velocity exercise, the EMG AMP and MPF responses during continuous exercise at CHR-5 did not reflect those typical of fatigue. In addition, these findings suggested that the level of muscle activation and action potential conduction velocity may be sensitive to changes in metabolic intensity and muscle temperature. Future studies should simultaneously examine EMG MPF, action potential conduction velocity, and muscle temperature during continuous treadmill running or cycle ergometry at a constant HR to further elucidate the relationships among these variables.

In the present study, during continuous running at CHR, there was an increase in EMG AMP, but no change in EMG MPF. These findings were not consistent with those previously reported (69) for continuous exercise performed at CHR during cycle
ergometry, but did reflect responses typical of neuromuscular fatigue. Despite the continuous decreases in velocity necessary to maintain CHR, 91% of the subjects completed the run at a \( \dot{V}O_2 \) (mean ± SD \( \dot{V}O_2 = 76 ± 5\% \) of \( \dot{V}O_2 \) peak) that was greater than the GET (70 ± 7\% \( \dot{V}O_2 \) peak). Thus, it is possible that the increase EMG AMP and muscle activation resulted from the accumulation of metabolites and/or ions and reflected the fatigue-induced recruitment of additional less efficient, fast-twitch motor units. The non-significant change in EMG MPF in the present study may be related to the competing influences of increased muscle temperature, which tends to increase EMG MPF, and the accumulation of metabolites and/or ions, which tends to decrease EMG MPF. Petrofsky (82) demonstrated that increased muscle temperature caused an increase in the frequency domain during constant power output exercise performed at lower intensities (20 and 40\% of \( \dot{V}O_2 \) max), while EMG MPF decreased during exercise performed at higher intensities (60 to 100\% of \( \dot{V}O_2 \) max). It was hypothesized that at higher intensities, the fatigue induced accumulation of metabolites and/or ions had a greater influence on the signal than the effects of temperature. Thus, it is possible the lack of change in EMG MPF at CHR in the present study reflected the effects of increased muscle temperature that was balanced by fatigue-induced reduction in action potential conduction velocity that occurs from the accumulation of various metabolites and/or ions. Future studies should simultaneously examine neuromuscular responses, muscle temperature, and interstitial ion concentrations to determine the relative contributions of increases in muscle temperature and the accumulation of \( H^+ \), \( NH_4^+ \), and/or \( K^+ \) to neuromuscular fatigue.

During continuous treadmill running at CHR+5, there was quadratic decrease in
EMG AMP. From the start of exercise to exhaustion, however, EMG AMP decreased by only 2% percent. This magnitude of change did not reflect a significant decrease in muscle activation across time. There was, however, a significant decrease in EMG MPF and action potential conduction velocity. These findings were not consistent with the linear decrease in EMG AMP and increase in EMG MPF previously reported (69) at CHR+5 during cycling ergometry. Although the velocity was reduced by 16 ± 10% during the run, muscle activation remained relatively stable, which indicated a decreased efficiency of muscle activation. In addition, despite the decrease in velocity, the metabolic demand of the exercise (Table 2) was greater than the GET (71 ± 6% of $\dot{V}O_2$ peak) for all 9 of the subjects throughout the run. Continuous exercise performed at this intensity likely resulted in the accumulation of metabolites and ions (lactate, inorganic phosphate, H+, NH₄⁺, and/or K⁺) that are typically associated with the fatigue-induced increases in muscle activation and EMG AMP as well as decreases in action potential conduction velocity and EMG MPF (36, 61, 90, 105). Thus, it is possible the non-significant change in EMG AMP reflected a balance between de-recruitment of motor units as the velocity was reduced, which tends to decrease EMG AMP, and the accumulation of metabolites and/or ions, which tends to increase the signal (29, 36, 61, 90, 105). In addition, the decrease in EMG MPF and action potential conduction velocity during treadmill running at CHR+5 may have been related to the accumulation of metabolites and/or ions, which may affect local muscular fatigue by altering cell membrane potentials (40). Thus, the EMG AMP and MPF responses during treadmill running at CHR+5 in the present study indicated that exhaustion may, in part, be related to neuromuscular fatigue and the accumulation of metabolites and/or ions.
Dissociation Between $\dot{V}O_2$ and Muscle Activation

Fatiguing exercise intensities performed above the GET are associated with a decrease in metabolic efficiency (i.e., greater $\dot{V}O_2$/work rate ratio) that is typically defined by a gradual increase in $\dot{V}O_2$ over time at a constant work rate, or by a decrease in the work rate when $\dot{V}O_2$ is held constant (51, 101). In the present study, however, the $\dot{V}O_2$ responses tracked the decrease in velocity that was required to maintain a constant HR at CHR-5, CHR, and CHR+5. Despite the continuous decrease in $\dot{V}O_2$ at each intensity, the $\dot{V}O_2$/work rate ratio increased significantly (Tables 6, 8, and 10) for each of the constant HR runs at CHR-5, CHR, and CHR+5. These findings indicated a fatigue induced decrease in metabolic efficiency during continuous treadmill running at a constant HR that was not defined by the pattern of response typical for $\dot{V}O_2$ during fatiguing constant work rate or $\dot{V}O_2$ exercise.

The decline in metabolic efficiency during fatiguing exercise has been attributed to multiple factors including increases in respiratory muscle work, HR, arterial blood lactate concentration, catecholamine concentration, muscle temperature, the additional recruitment of fast twitch muscle fibers, and a reduced efficiency of the already recruited fibers (51, 84, 85, 86). Although all of these may play a role, Poole et al. (87) indicated that approximately 86% of the decrease in metabolic efficiency was accounted for by factors within the working muscle. Subsequent studies have shown that increased blood lactate concentration (86), muscle temperature (54), and catecholamine concentrations (32) do not account for a significant proportion of the decreased metabolic efficiency. There is evidence, however, that the progressive loss of muscle efficiency is related to the
additional recruitment of fast-twitch glycolytic fibers, which have a higher O₂ cost per unit of work rate (19, 57, 96), and/or an increased metabolic demand (decreased metabolic efficiency) within the already recruited fibers (2, 95, 101, 107, 108). For example, studies (19, 96) have shown that the decreased metabolic efficiency was related to increased EMG AMP and muscle activation, while other findings (2, 94, 101, 107, 108) indicated that the decreased efficiency was related to fatigue-induced decreases in efficiency (greater ATP/work rate ratio and/or lower ATP/"O₂ [P/O] ratio) within the active muscle fibers.

The present findings indicated dissociations between "O₂ and EMG AMP during continuous treadmill running at a CHR and CHR+5, but not CHR-5. Specifically, the decrease in "O₂ at each intensity corresponded to a decrease, increase, and no change in EMG AMP at CHR-5, CHR, and CHR+5, respectively. The decrease in muscle activation at CHR-5 and no change at CHR+5 corresponded to decreased metabolic efficiency (Tables 6 and 10) and, thus, likely reflected fatigue-induced decreases in efficiency within the recruited fibers. At CHR, the decrease in metabolic efficiency was associated with an increase in muscle activation (Table 8). Thus, consistent with previous findings (19, 57, 94, 95, 100, 107), the current results indicated that the decreased metabolic efficiency was likely the result of a combination of both recruitment (at CHR) and fatigue-induced decreases in efficiency within the recruited fibers (at CHR-5 and CHR+5).

Factors affecting the perception of effort and development of fatigue

The results of the present study as well as those of others (34, 69) showed that RPE is a sensitive indicator of fatigue during continuous exercise at a constant work rate
or HR. The perception of effort and the development of fatigue during exercise may be mediated, in part, by central (HR and \( \dot{V}O_2 \)), peripheral (metabolic acidosis and changes in metabolite and/or ion concentration) and/or non-specific (e.g., changes in core or muscle temperature) factors (71, 91). The Borg RPE (6-20) scale, commonly used to examine perceptual responses during exercise, relies on the assumption that HR provides “a good indicator of metabolic strain” (11, p. 142) and is linearly related to the perception of effort and exercise intensity. Although HR, \( \dot{V}O_2 \), and the perception of effort increase linearly during incremental exercise (104), continuous, fatiguing exercise performed at CHR-5, CHR, and CHR+5 in the present study resulted in no change in HR, a decrease in \( \dot{V}O_2 \), and an increase in RPE. These findings were consistent with Mielke (69) for cycle ergometry at a constant HR, and suggested the neither fatigue nor the perception of effort, were mediated by changes in HR or O2 availability.

Although central factors (HR and \( \dot{V}O_2 \)) were not associated with increases in RPE or the development of fatigue in the present study, it is possible that peripheral (metabolic acidosis and changes in metabolite and/or ion concentration) and/or non-specific (e.g., changes in core or muscle temperature) factors mediated the fatigue process and influenced the perception of effort. It has been suggested that changes in the cellular environment (i.e., decreased pH and the accumulation of metabolites and/or ions) that occur during exercise performed above the GET result in the development of fatigue that is reflected by changes in neuromuscular responses and decreased metabolic efficiency (29, 36, 51, 61, 90, 105). These changes (decreased pH and the accumulation of metabolites and/or ions) may also provide sensory cues for the perception of effort (71). In the present study, the development of fatigue, reflected by the decrease in metabolic
efficiency that resulted from additional recruitment (CHR) or fatigue-induced decreases in efficiency within the recruited fibers (CHR-5 and CHR+5) was possibly related to the accumulation of metabolites and/or ions which may have provided sensory cues that resulted in an increased perception of effort.

It is also possible that fatigue and the perception of effort were related to changes in core and/or muscle temperature. Prolonged (>10 min), fatiguing exercise performed at submaximal and maximal (20 to 100% \( \dot{V}O_2 \text{ max} \)) intensities has been shown to result in elevated muscle temperature (82) that reflected similar increases in core temperature (93) and resulted in a greater perception of effort (74). Based on these findings (74, 82, 93), it has been suggested (71) that “…the processes which regulate core temperature during exercise may provide sensory input for perception of effort” (p. 160). In the present study, it is likely that the relative intensities (72 – 94% \( \dot{V}O_2 \text{ peak} \)) and durations of the runs at CHR-5, CHR, and CHR+5 (mean \( T_{lim} \pm SD = 56.97 \pm 1.23 \text{ min} \), 48.37 \( \pm 11.04 \), and 20.11 \( \pm 16.08 \), respectively) were great enough to induce significant elevations in core and/or muscle temperatures, which may have provided sensory cues signaling the increases in the perceptions of effort.

**Implications**

According to the overload principle of training, exercise performed below a minimum intensity, or threshold, will not provide a great enough stimulus to elicit significant physiological adaptations, such as increases in the GET or \( \dot{V}O_2 \text{ peak} \) (33). It appears (68) the threshold for improving cardiorespiratory fitness is dependent upon an individual’s training status, where intensities of 70 – 80% \( \dot{V}O_2 \text{ peak} \) in moderately trained individuals and 95 – 100% \( \dot{V}O_2 \text{ peak} \) in highly trained individuals may be required to
increase \( \dot{V}O_2 \text{peak} \). Currently, the American College of Sport Medicine (ACSM) (82) recommends vigorous (77 – 95% \( HR_{\text{peak}} \) or 64 – 90% \( \dot{V}O_2 \text{peak} \)) aerobic exercise for 30 to 60 min to provide the greatest stimulus to improve cardiorespiratory fitness.

Exercise prescriptions for aerobic training programs are often based on HR (35, 59), which is linearly related to \( \dot{V}O_2 \) during incremental exercise (104). During the constant HR runs in the present study, however, \( \dot{V}O_2 \) was dissociated from HR and decreased by 9 to 14% throughout the work bouts. These findings indicated that exercise maintained at a constant HR results in a decrease in \( \dot{V}O_2 \). Thus, exercise prescribed as a percentage of \( HR_{\text{peak}} \) may not result in \( \dot{V}O_2 \) responses within the vigorous intensity range for the duration of the work bout. Therefore, the initial HR should be prescribed at a relative intensity that is high enough to maintain the \( \dot{V}O_2 \) within the desired range for at least 30 min.

In the present study, the \( \dot{V}O_2 \) values (72 – 88% \( \dot{V}O_2 \text{peak} \)) for exercise at CHR-5 (88 ± 3% \( HR_{\text{peak}} \)) and CHR (91 ± 3% \( HR_{\text{peak}} \)) were within the range associated with vigorous intensity exercise (64 – 90% \( \dot{V}O_2 \text{peak} \)) and could be maintained for 30 to 60 min, while the \( \dot{V}O_2 \) at the beginning of the work bout at CHR+5 (94 ± 4% \( \dot{V}O_2 \text{peak} \)) was greater than this range and resulted in exhaustion within 30 min. These findings indicated that the CHR model may provide a useful threshold for prescribing vigorous intensity exercise to improve cardiorespiratory fitness among moderately trained individuals (81). The advantage of prescribing exercise intensity based on the CHR rather than a population-based HR range is that the CHR is determined from individual HR responses to continuous exercise. Thus, the CHR provides a HR that is high enough to maintain a
vigorous intensity $\dot{V}O_2$, but low enough to be sustained for at least 30 min for each individual.

**Limitations and Future Directions**

This study was the first to examine the application of the CHR model to treadmill running as well as the physiological responses during continuous runs at CHR-5, CHR, and CHR+5. There were several limitations, however, to this study. The primary limitation was that, due to equipment malfunctions or a lack of data points, subjects had to be removed from the analyses, which resulted in a loss of power and limited the comparisons among the CHR-5, CHR, and CHR+5 groups. Although 13 subjects completed each of the constant HR runs at CHR-5, CHR, and CHR+5, 4 subjects had to be removed from the analyses due to equipment malfunctions and 3 subjects were removed due to a lack of data points ($T_{lim}$ values 0.82 – 2.71 min). Thus, complete data sets were available for 12 subjects at CHR-5, 11 subjects and CHR, and 9 subjects at CHR+5. Due to the different number of subjects in each group, direct comparisons of the responses for $\dot{V}O_2$, EMG AMP, EMG MPF, or RPE at CHR-5, CHR, CHR+5 were not possible. In addition, there were complete data sets for only 8 subjects for all of the dependent variables ($\dot{V}O_2$, EMG AMP, EMG MPF, RPE, and $T_{lim}$) at all three intensities (CHR-5, CHR, and CHR+5). With only 8 subjects available for separate 3 x 11 repeated measures analyses of variance (ANOVAs) (group [CHR-5, CHR, CHR+5] x time [0, 10, 20, 30, 40, 50, 60, 70, 80, 90, 100% of $T_{lim}$]) there was not enough statistical power to detect a significant difference in the patterns of responses among the groups.

Although direct comparisons of the responses among the 3 different intensities (CHR-5, CHR, and CHR+5) could not be made in this study, the current results provided
preliminary data that indicated potential differences in neuromuscular responses at CHR-5, CHR, and CHR+5. Due to the limitations of this study, future studies should replicate the present study using a larger sample size (n = 18-20) to ensure there are enough subjects to complete regression analyses as well as 2-way repeated measures ANOVAs. This will allow for direct comparisons among the groups to further examine the potential differences in physiological responses.

Another potential limitation of the present study was the use of only one muscle, the vastus lateralis, in the analyses of the neuromuscular responses. Studies (39, 97) have indicated significant differences in the level of activation of the lower limb muscles with the adductors, semitendinosus, gracilis, biceps femoris, and semimembranosus most highly activated during horizontal treadmill running. It has also been shown (39) that EMG AMP increased for the rectus femoris and biceps femoris muscles prior to the vastus lateralis. Thus, the pattern of responses observed for the vastus lateralis in the present study may not reflect the patterns of other muscles. Therefore, future studies should measure the neuromuscular responses in multiple lower limb muscles during continuous runs at CHR-5, CHR, and CHR+5 to further examine the recruitment patterns associated with the onset of neuromuscular fatigue.

The CHR model may provide an estimate of a sustainable HR within the vigorous exercise intensity domain for each individual and, thus, has potential applications in exercise prescription to induce increases in cardiorespiratory fitness. Additional research is needed to compare the physiological adaptations to training at the CHR versus training within the population-based HR range (77 – 95% HR_{peak}) currently recommended by the ACSM (81). Future studies should also compare the training adaptations (e.g., changes in
the GET or $\dot{V}O_2$ peak) to exercise prescribed at the CHR in highly trained, moderately trained, and untrained runners to determine if the CHR model is applicable to a range of training statuses.

**Summary**

The results of the present study indicated that the mathematical model used to derive the CHR during cycle ergometry (69) was also applicable to treadmill running. Theoretically, the treadmill based CHR estimates in the present study represented the maximal HR that could be maintained for an extended period of time without fatigue (70). The current findings indicated that all of the subjects (n = 12) at CHR-5 and 64% of the subjects at CHR completed the 60 min treadmill running work bout, while 89% of the subjects exhausted prior to 30 min at CHR+5. Thus, the results of the present study indicated that on average, HR values that were less than or equal to the CHR, represented sustainable (minimum of 30 to 60 min) intensities, while CHR+5 did not. The $T_{lim}$ values at CHR during continuous treadmill running were greater than those previously reported (14, 17, 45, 47, 50, 66, 77) for CV or CP, and indicated that a threshold associated with the highest sustainable exercise intensity should be based on the responses of a physiological parameter, such as HR, rather than a specific velocity or power output. During the continuous runs at CHR-5, CHR, and CHR+5, velocity was reduced to maintain the selected HR, and the $\dot{V}O_2$ responses tracked the changes in velocity. There were, however, increases in the perception of effort throughout the each of the runs. Although there were submaximal $\dot{V}O_2$ and HR responses at exhaustion for each intensity (CHR-5, CHR, and CHR+5), RPE increased to RPE$_{peak}$ at CHR+5. These findings suggested that the perception of effort, but not $\dot{V}O_2$ or HR, can be used to identify exhaustion during
continuous exercise at or above CHR. In addition, the decrease in $\dot{V}\text{O}_2$ throughout the runs at a constant HR suggested that the RPE and fatigue were not related to $O_2$ availability or sympathetic activation, but increases in the perception of effort may have been related to increases in core temperature (24). Continuous treadmill running at CHR-5 in the present study resulted in decreases in EMG AMP and muscle activation that tracked the decreases in velocity, while EMG MPF and action potential conduction velocity increased throughout the run, possibly due to increases in muscle temperature. During continuous running at CHR, there was an increase in EMG AMP, but no change in EMG MPF. At CHR+5, there was no change in EMG AMP and a decrease in EMG MPF. These fatigue-induced changes in the time and frequency domains may be related to several factors, including the accumulation of metabolites and/or ions as well as changes in muscle temperature. The current results also indicated a decreased metabolic efficiency during the continuous runs at CHR-5, CHR, and CHR+5 that was likely the result of a combination of both recruitment (at CHR) and fatigue-induced decreases in efficiency within the recruited fibers (at CHR-5 and CHR+5). It is possible that the development of fatigue, reflected by the decrease in metabolic efficiency during each of the runs (CHR-5, CHR, and CHR+5), was related to accumulation of metabolites and/or ions and elevations in core and/or muscle temperatures, which may have provided sensory cues signaling the increases in the perceptions of effort. The relative intensity of CHR-5 and CHR (72 – 88% $\dot{V}$O$_2$ peak), but not CHR+5 (94 ± 4% $\dot{V}$O$_2$ peak) were within the range associated with vigorous intensity exercise (64 – 90% $\dot{V}$O$_2$ peak) recommended by the ACSM (81) for inducing the greatest improvements in cardiorespiratory fitness. These findings indicated that the CHR, which is determined from individual HR
responses to continuous exercise, provides a HR that is high enough to maintain a vigorous intensity $\dot{V}O_2$, but low enough to be sustained for at least 30 min for each individual.
Table 1. Mean ± (SD) and range of peak values for each variable recorded during the incremental test for the critical heart rate minus 5 b·min\(^{-1}\) (CHR-5), CHR, and CHR+5 b·min\(^{-1}\) (CHR+5) groups.

<table>
<thead>
<tr>
<th></th>
<th>CHR-5 (n=12)</th>
<th></th>
<th>CHR (n=11)</th>
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<th>CHR+5 (n=9)</th>
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<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Range</td>
<td>Mean ± SD</td>
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<tr>
<td>(\dot{V}O_2)\textsubscript{peak} (mL·kg(^{-1})·min(^{-1}))</td>
<td>47.58 ± 7.07</td>
<td>36.72 – 61.18</td>
<td>46.63 ± 6.38</td>
<td>36.72 – 57.41</td>
<td>47.37 ± 6.49</td>
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<td>v(\dot{V}O_2)\textsubscript{peak} (km·h(^{-1}))</td>
<td>15.70 ± 1.97</td>
<td>13.48 – 20.58</td>
<td>15.34 ± 1.39</td>
<td>13.48 – 17.97</td>
<td>15.59 ± 1.37</td>
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<td>HR\textsubscript{peak} (b·min(^{-1}))</td>
<td>193 ± 10</td>
<td>176 – 212</td>
<td>194 ± 10</td>
<td>176 – 212</td>
<td>190 ± 7</td>
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<tr>
<td>AMP\textsubscript{peak} (µVrms)</td>
<td>120.88 ± 42.30</td>
<td>78.84 – 205.03</td>
<td>123.83 ± 43.05</td>
<td>78.84 – 205.03</td>
<td>128.83 ± 45.12</td>
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<td>MPF\textsubscript{peak} (Hz)</td>
<td>82.48 ± 24.39</td>
<td>43.89 – 144.30</td>
<td>78.76 ± 16.68</td>
<td>43.89 – 103.73</td>
<td>79.11 ± 18.62</td>
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<td>RPE\textsubscript{peak}</td>
<td>20 ± 0.5</td>
<td>19 – 20</td>
<td>20 ± 0.5</td>
<td>19 – 20</td>
<td>20 ± 0.4</td>
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</table>

Peak oxygen consumption rate (\(\dot{V}O_2\)\textsubscript{peak}), the velocity associated with \(\dot{V}O_2\)\textsubscript{peak} (v\(\dot{V}O_2\)\textsubscript{peak}), the HR at \(\dot{V}O_2\)\textsubscript{peak} (HR\textsubscript{peak}), the electromyographic (EMG) amplitude at \(\dot{V}O_2\)\textsubscript{peak} (AMP\textsubscript{peak}), the EMG mean power frequency at \(\dot{V}O_2\)\textsubscript{peak} (MPF\textsubscript{peak}), and the rating of perceived exertion at \(\dot{V}O_2\)\textsubscript{peak} (RPE\textsubscript{peak}).
Table 2. The gas exchange threshold (GET) for each of the subjects for the critical heart rate minus 5 b·min\(^{-1}\) (CHR-5), CHR, and CHR plus 5 b·min\(^{-1}\) groups, the velocity associated with the GET, and the GET as a percentage of \(\dot{V}O_2\)\(_{\text{peak}}\). The -- indicates the data for that subject were excluded for that intensity (see Methods section for description of excluded subjects).

<table>
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<tr>
<th>Subject</th>
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<th>velocity</th>
<th>% (\dot{V}O_2)(_{\text{peak}})</th>
<th>(\dot{V}O_2)</th>
<th>velocity</th>
<th>% (\dot{V}O_2)(_{\text{peak}})</th>
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Note: \(\dot{V}O_2\) expressed in mL·kg\(^{-1}\)·min\(^{-1}\) and velocity expressed in km·h\(^{-1}\).
Table 3. The heart rate (HR) and percent of the HR at \( \dot{V}O_2 \text{peak} \) (HR\(_{\text{peak}}\)) for the critical heart rate minus 5 b·min\(^{-1}\) (CHR-5), CHR, and CHR+5 b·min\(^{-1}\) (CHR+5) groups. The -- indicates that the data for that subject were excluded for that intensity (see Methods section for description of excluded subjects).

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<th>HR (b·min(^{-1}))</th>
<th>% of HR(_{\text{peak}})</th>
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Table 4. The time to exhaustion ($T_{lim}$) for each subject at critical heart rate minus 5 b·min$^{-1}$ (CHR-5), CHR, and CHR+5 b·min$^{-1}$ (CHR+5) groups. The -- indicates that the data for that subject were excluded for that intensity (see Methods section for description of excluded subjects).

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Table 5. The \( \dot{V}_O_2 \), velocity, electromyographic (EMG) amplitude (EMG AMP), EMG mean power frequency (EMG MPF), and rating of perceived exertion (RPE) normalized values for the time critical heart rate minus 5 b·min\(^{-1}\) was reached (start) and at exhaustion (end) as well as the percent change (\(\%\Delta\)) for each variable.

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<th>%Δ</th>
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<th>End AMP</th>
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Table 6. The $\dot{V}O_2$ associated with the velocity at the termination of exercise at CHR-5 ($\dot{V}O_2^{\text{EST}}$) (see Methods section for a description), the actual $\dot{V}O_2$ measured at the termination of exercise ($\dot{V}O_2^{\text{END}}$), the velocity at the termination of exercise (END velocity), the metabolic efficiency ($E^{\text{EST}}$) determined from the $\dot{V}O_2^{\text{EST}}$/velocity ratio, the metabolic efficiency ($E^{\text{END}}$) determined from the $\dot{V}O_2^{\text{END}}$/velocity ratio.

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Note: $\dot{V}O_2$ expressed in L·min$^{-1}$ and velocity expressed in km·h$^{-1}$

* significantly greater (p < 0.05) than $\dot{V}O_2^{\text{EST}}$

# significantly greater (p < 0.05) than $E^{\text{EST}}$
Table 7. The $\dot{V}O_2$, velocity, electromyographic (EMG) amplitude (EMG AMP), EMG mean power frequency (EMG MPF), and rating of perceived exertion (RPE) normalized values for the time critical heart rate was reached (start) and at exhaustion (end) as well as the percent change ($\%\Delta$) for each variable.

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Table 8. The \( \dot{V}O_2 \) associated with the velocity at the termination of exercise at CHR (\( \dot{V}O_2^{\text{EST}} \)) (see Methods section for a description), the actual \( \dot{V}O_2 \) measured at the termination of exercise (\( \dot{V}O_2^{\text{END}} \)), the velocity at the termination of exercise (END velocity), the metabolic efficiency (\( E^{\text{EST}} \)) determined from the \( \dot{V}O_2^{\text{EST}}/\text{velocity} \) ratio, the metabolic efficiency (\( E^{\text{END}} \)) determined from the \( \dot{V}O_2^{\text{END}}/\text{velocity} \) ratio.

<table>
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<th>( \dot{V}O_2^{\text{EST}} )</th>
<th>( \dot{V}O_2^{\text{END}} )</th>
<th>END Velocity</th>
<th>( E^{\text{EST}} )</th>
<th>( E^{\text{END}} )</th>
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Note: \( \dot{V}O_2 \) expressed in L·min\(^{-1}\) and velocity expressed in km·h\(^{-1}\)

* significantly greater (\( p < 0.05 \)) than \( \dot{V}O_2^{\text{EST}} \)

° significantly greater (\( p < 0.05 \)) than \( E^{\text{EST}} \)
Table 9. The $\dot{V}O_2$, velocity, electromyographic (EMG) amplitude (EMG AMP), EMG mean power frequency (EMG MPF), and rating of perceived exertion (RPE) normalized values for the time critical heart rate plus 5 b·min$^{-1}$ was reached (start) and at exhaustion (end) as well as the percent change ($\%\Delta$) for each variable.

<table>
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<tr>
<th>Subject</th>
<th>$\dot{V}O_2$ start</th>
<th>$\dot{V}O_2$ end</th>
<th>%Δ</th>
<th>Velocity start</th>
<th>Velocity end</th>
<th>%Δ</th>
<th>AMP start</th>
<th>AMP end</th>
<th>%Δ</th>
<th>MPF start</th>
<th>MPF end</th>
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Table 10. The $\dot{V}O_2$ associated with the velocity at the termination of exercise at CHR+5 ($\dot{V}O_2^{\text{EST}}$) (see Methods section for a description), the actual $\dot{V}O_2$ measured at the termination of exercise ($\dot{V}O_2^{\text{END}}$), the velocity at the termination of exercise (END velocity), the metabolic efficiency ($E^{\text{EST}}$) determined from the $\dot{V}O_2^{\text{EST}}$/velocity ratio, the metabolic efficiency ($E^{\text{END}}$) determined from the $\dot{V}O_2^{\text{END}}$/velocity ratio.

<table>
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<th>Subject</th>
<th>$\dot{V}O_2^{\text{EST}}$</th>
<th>$\dot{V}O_2^{\text{END}}$</th>
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</table>

Note: $\dot{V}O_2$ expressed in L·min$^{-1}$ and velocity expressed in km·h$^{-1}$

* significantly greater (p < 0.05) than $\dot{V}O_2^{\text{EST}}$

# significantly greater (p < 0.05) than $E^{\text{EST}}$
Figure 1a. The relationship between the total distance (TD) and time limit ($T_{\text{lim}}$) is described by the linear equation $TD = a + b(T_{\text{lim}})$, where ($a$) is equal to the anaerobic running capacity (ARC) and ($b$) is critical velocity (CV). The equation $T_{\text{lim}} = \frac{ARC}{V - CV}$ describes the relationship between the imposed velocity ($V$) versus $T_{\text{lim}}$ for treadmill running and is derived from the linear relationship so that the asymptote is equivalent to ($b$ or CV). Figure 1b. Theoretically, exercise can be maintained indefinitely when the imposed velocity is $\leq CV$. The $T_{\text{lim}}$ can be predicted for any velocity $\geq CV$ from the hyperbolic relationship, $T_{\text{lim}} = \frac{ARC}{V - CV}$. 
Figure 2. An example of the mathematical model used to determine critical heart rate for one subject. Critical heart rate is the slope of the linear relationship between the total number of heartbeats ($HB_{lim}$) and time to exhaustion ($T_{lim}$).

$$HB_{lim} = 21 + 176(T_{lim})$$

$r^2 = 0.999$
Figure 3a. Results of the composite polynomial regression analyses at critical heart rate minus 5 b·min\(^{-1}\) for \(\dot{V}\text{O}_2\), heart rate (HR), electromyographic (EMG) amplitude (AMP), EMG mean power frequency (MPF), rating of perceived exertion (RPE), and velocity.

\(\dot{V}\text{O}_2\) – Quadratic decrease \((R^2 = 0.966)\)
HR – No relationship \((r^2 = 0.270)\)
AMP – Linear decrease \((r^2 = 0.674)\)
MPF – Linear Increase \((r^2 = 0.499)\)
RPE – Quadratic increase \((R^2 = 0.959)\)
Velocity – Quadratic decrease \((R^2 = 0.899)\)
Figure 3b. Results of the individual polynomial regression analyses at critical heart rate minus 5 b·min⁻¹ for \( \dot{V}O_2 \), heart rate (HR), electromyographic (EMG) amplitude (AMP), EMG mean power frequency (MPF), rating of perceived exertion (RPE), and velocity.

\( \dot{V}O_2 \) – Linear decrease \( (r^2 = 0.527) \)  
HR – No relationship \( (r^2 = 0.089) \)  
AMP – Quadratic decrease \( (r^2 = 0.523) \)  
MPF – No relationship \( (r^2 = 0.106) \)  
RPE – No relationship \( (r^2 = 0.250) \)  
Velocity – Quadratic decrease \( (R^2 = 0.930) \)
\( \dot{V}O_2 \) – Quadratic \( (R^2 = 0.933) \)  
HR – No relationship \( (r^2 = 0.050) \)  
AMP – No relationship \( (r^2 = 0.097) \)  
MPF – No relationship \( (r^2 = 0.001) \)  
RPE – No relationship \( (r^2 = 0.013) \)  
Velocity – Quadratic \( (R^2 = 0.923) \)
\( \dot{V}O_2 \) – Quadratic \( (R^2 = 0.769) \)

HR – No relationship \( (r^2 = 0.200) \)

AMP – No relationship \( (r^2 = 0.265) \)

MPF – No relationship \( (r^2 = 0.077) \)

RPE – Linear increase \( (r^2 = 0.903) \)

Velocity – Quadratic \( (R^2 = 0.722) \)
\( \dot{\text{V}}\text{O}_2 \) – Linear \((r^2 = 0.380)\)

HR – No relationship \((r^2 = 0.213)\)

AMP – Linear increase \((r^2 = 0.403)\)

MPF – Linear increase \((r^2 = 0.602)\)

RPE – Linear increase \((r^2 = 0.785)\)

Velocity – Quadratic \((R^2 = 0.878)\)
\( \dot{V}O_2 \) – Quadratic (\( R^2 = 0.814 \))
HR – No relationship (\( r^2 = 0.038 \))
AMP – No relationship (\( r^2 = 0.000 \))

MPF – No relationship (\( r^2 = 0.125 \))
RPE – Linear increase (\( r^2 = 0.933 \))
Velocity – Quadratic (\( R^2 = 0.796 \))
$\dot{V}O_2$ – Linear decrease ($r^2 = 0.684$)  
HR – No relationship ($r^2 = 0.023$)  
AMP – No relationship ($r^2 = 0.040$)  
MPF – No relationship ($r^2 = 0.087$)  
RPE – No relationship ($r^2 = 0.250$)  
Velocity – Quadratic ($R^2 = 0.915$)
\[ \dot{V}O_2 \text{ – Quadratic (} R^2 = 0.843 \) } \quad \text{MPF – Quadratic (} R^2 = 0.787 \) \\
HR – No relationship (} r^2 = 0.000 \) } \quad \text{RPE – No relationship (} r^2 = 0.054 \) \\
AMP – No relationship (} r^2 = 0.000 \) } \quad \text{Velocity – Quadratic (} R^2 = 0.839 \)
\[ \dot{V}_O_2 \text{ – Quadratic (} R^2 = 0.845 \) \]

\[ \text{HR} \text{ – No relationship (} r^2 = 0.139 \) \]

\[ \text{AMP} \text{ – Quadratic (} R^2 = 0.859 \) \]

\[ \text{MPF} \text{ – No relationship (} R^2 = 0.787 \) \]

\[ \text{RPE} \text{ – Quadratic (} r^2 = 0.847 \) \]

\[ \text{Velocity} \text{ – Quadratic (} R^2 = 0.922 \) \]
Subject 10

Normalized Values (% of peak values) vs. Normalized Time (% of $T_{\text{lim}}$)

- $\dot{V}O_2$ – Quadratic ($R^2 = 0.908$)
- HR – No relationship ($r^2 = 0.156$)
- AMP – Linear ($R^2 = 0.720$)
- MPF – Quadratic ($R^2 = 0.483$)
- RPE – Quadratic ($r^2 = 0.814$)
- Velocity – Quadratic ($R^2 = 0.872$)
\[ \dot{\text{V}}O_2 \] – Linear decrease \( (R^2 = 0.945) \)

HR – No relationship \( (r^2 = 0.017) \)

AMP – Quadratic \( (R^2 = 0.700) \)

MPF – No relationship \( (R^2 = 0.024) \)

RPE – No relationship \( (r^2 = 0.000) \)

Velocity – Quadratic \( (R^2 = 0.876) \)
\[ \dot{V}O_2 \] – Linear decrease (\( R^2 = 0.639 \))
HR – No relationship (\( r^2 = 0.023 \))
AMP – Linear (\( R^2 = 0.766 \))

MPF – Linear increase (\( R^2 = 0.620 \))
RPE – Quadratic (\( r^2 = 0.914 \))
Velocity – Quadratic (\( R^2 = 0.863 \))
\[ \dot{V}O_2 \] – Linear decrease \( (R^2 = 0.692) \)
HR – No relationship \( (r^2 = 0.233) \)
AMP – No relationship \( (r^2 = 0.144) \)

MPF – Linear increase \( (R^2 = 0.483) \)
RPE – Linear increase \( (r^2 = 0.863) \)
Velocity – Quadratic \( (R^2 = 0.835) \)
Figure 4a. Results of the composite polynomial regression analyses at critical heart rate for \( \dot{V}O_2 \), velocity, electromyographic (EMG) amplitude (AMP), EMG mean power frequency (MPF), and rating of perceived exertion (RPE).

\[ \dot{V}O_2 \text{ - Quadratic decrease } (R^2 = 0.979) \]
\[ \text{MPF – No relationship } (r^2 = 0.180) \]
\[ \text{HR – No relationship } (r^2 = 0.163) \]
\[ \text{RPE – Linear increase } (r^2 = 0.958) \]
\[ \text{AMP – Linear increase } (r^2 = 0.862) \]
\[ \text{Velocity – Quadratic decrease } (R^2 = 0.922) \]
Figure 4b. Results of the individual polynomial regression analyses at critical heart rate for \( \dot{V}O_2 \), heart rate (HR), electromyographic (EMG) amplitude (AMP), EMG mean power frequency (MPF), rating of perceived exertion (RPE), and velocity.

\[
\begin{align*}
\dot{V}O_2 & \quad \text{Linear decrease (} r^2 = 0.503) \\
HR & \quad \text{No relationship (} r^2 = 0.120) \\
AMP & \quad \text{Quadratic (} r^2 = 0.896) \\
MPF & \quad \text{Quadratic (} r^2 = 0.786) \\
RPE & \quad \text{Linear increase (} r^2 = 0.935) \\
\text{Velocity} & \quad \text{Quadratic (} R^2 = 0.957) \\
\end{align*}
\]
\( \dot{V}O_2 \) – Quadratic \( (R^2 = 0.935) \)
HR – No relationship \( (r^2 = 0.010) \)
AMP – No relationship \( (r^2 = 0.283) \)

MPF – Linear decrease \( (r^2 = 0.497) \)

RPE – No relationship \( (r^2 = 0.250) \)

Velocity – Quadratic \( (R^2 = 0.913) \)
\[ \dot{V}O_2 \text{ – Linear decrease (} r^2 = 0.887 \text{)} \]
\[ \text{HR – No relationship (} r^2 = 0.161 \text{)} \]
\[ \text{AMP – No relationship (} r^2 = 0.001 \text{)} \]
\[ \text{MPF – No relationship (} r^2 = 0.259 \text{)} \]
\[ \text{RPE – Quadratic (} R^2 = 0.928 \text{)} \]
\[ \text{Velocity – Quadratic (} R^2 = 0.866 \text{)} \]
Normalized Values (\% of peak values)

Normalized Time (% of T_{lim})

Subject 5

\(\dot{V}O_2\) – Linear decrease \((r^2 = 0.516)\)
HR – No relationship \((r^2 = 0.050)\)
AMP – Linear increase \((r^2 = 0.727)\)

MPF – Linear decrease \((r^2 = 0.654)\)
RPE – Linear increase \((r^2 = 0.781)\)
Velocity – Quadratic \((R^2 = 0.957)\)
$\dot{V}_O_2$ – Quadratic ($R^2 = 0.847$)  
HR – No relationship ($r^2 = 0.006$)  
AMP – Quadratic ($r^2 = 0.644$)  
MPF – No relationship ($r^2 = 0.119$)  
RPE – Quadratic ($r^2 = 0.813$)  
Velocity – Quadratic ($R^2 = 0.776$)
\[ \dot{V}O_2 \text{ – Quadratic (} R^2 = 0.935 \text{)} \]

\[ \text{HR – No relationship (} r^2 = 0.253 \text{)} \]

\[ \text{AMP – Linear decrease (} r^2 = 0.364 \text{)} \]

\[ \text{MPF – No relationship (} r^2 = 0.005 \text{)} \]

\[ \text{RPE – Linear decrease (} r^2 = 0.399 \text{)} \]

\[ \text{Velocity – Quadratic (} R^2 = 0.966 \text{)} \]
\[ \dot{V}O_2 \text{ – Quadratic (} R^2 = 0.926) \]
\[ \text{HR – No relationship (} r^2 = 0.007) \]
\[ \text{AMP – Linear increase (} r^2 = 0.781) \]
\[ \text{MPF – Linear decrease (} r^2 = 0.438) \]
\[ \text{RPE – Quadratic (} R^2 = 0.957) \]
\[ \text{Velocity – Quadratic (} R^2 = 0.910) \]
\[ \dot{V}O_2 \] – Quadratic \( (R^2 = 0.942) \)

HR – No relationship \( (r^2 = 0.107) \)

AMP – Linear decrease \( (r^2 = 0.603) \)

MPF – Linear increase \( (r^2 = 0.474) \)

RPE – Quadratic \( (r^2 = 0.705) \)

Velocity – Quadratic \( (R^2 = 0.833) \)
\( \dot{V}O_2 \) – Quadratic \( (R^2 = 0.955) \)  
HR – No relationship \( (r^2 = 0.026) \)  
AMP – No relationship \( (r^2 = 0.105) \)  
MPF – No relationship \( (r^2 = 0.289) \)  
RPE – Quadratic \( (R^2 = 0.832) \)  
Velocity – Quadratic \( (R^2 = 0.863) \)
\[ \dot{V}O_2 \] – Linear decrease \((r^2 = 0.835)\)

HR – No relationship \((r^2 = 0.063)\)

AMP – Linear increase \((r^2 = 0.727)\)

MPF – Linear increase \((r^2 = 0.412)\)

RPE – Quadratic \((R^2 = 0.949)\)

Velocity – Quadratic \((R^2 = 0.883)\)
\( \dot{V}O_2 \) – Linear decrease \( (r^2 = 0.684) \)
HR – No relationship \( (r^2 = 0.063) \)
AMP – Linear increase \( (r^2 = 0.779) \)
RPE – Linear increase \( (r^2 = 0.768) \)
Velocity – Quadratic \( (R^2 = 0.964) \)

MPF – No relationship \( (r^2 = 0.089) \)
Figure 5a. Results of the composite polynomial regression analyses at critical heart rate plus 5 b·min⁻¹ for $\dot{V}O_2$, heart rate (HR), electromyographic (EMG) amplitude (AMP), EMG mean power frequency (MPF), rating of perceived exertion (RPE), and velocity.

$\dot{V}O_2$ – Linear decrease ($r^2 = 0.960$)  
HR – No change ($r^2 = 0.004$)  
AMP – Quadratic decrease ($R^2 = 0.646$)  
MPF – Linear decrease ($r^2 = 0.572$)  
RPE – Linear increase ($r^2 = 0.961$)  
Velocity – Quadratic decrease ($R^2 = 0.955$)
Figure 5b. Results of the individual polynomial regression analyses at critical heart rate plus 5 b·min⁻¹ for \( \dot{V}O_2 \), heart rate (HR), electromyographic (EMG) amplitude (AMP), EMG mean power frequency (MPF), rating of perceived exertion (RPE), and velocity.

\[\dot{V}O_2 \text{ – Linear decrease (} r^2 = 0.536 \text{)}\]  
\[\text{HR – No relationship (} r^2 = 0.057 \text{)}\]  
\[\text{AMP – Linear increase (} r^2 = 0.373 \text{)}\]  
\[\text{MPF – Quadratic (} R^2 = 0.897 \text{)}\]  
\[\text{RPE – Quadratic increase (} R^2 = 0.974 \text{)}\]  
\[\text{Velocity – Quadratic (} R^2 = 0.978 \text{)}\]
\( \dot{V}O_2 \) – Quadratic \( (R^2 = 0.968) \) 
HR – No relationship \( (r^2 = 0.010) \) 
AMP – Linear decrease \( (r^2 = 0.686) \) 

MPF – No relationship \( (r^2 = 0.041) \) 
RPE – No relationship \( (r^2 = 0.250) \) 
Velocity – Quadratic \( (R^2 = 0.808) \)
Subject 5

\[ \dot{V}_O^2 \] – No relationship \((r^2 = 0.047)\)  
HR – No relationship \((r^2 = 0.150)\)  
AMP – Linear increase \((r^2 = 0.745)\)  
\[ \dot{V}_O^2 \] – No relationship \((r^2 = 0.163)\)  
RPE – Quadratic \((R^2 = 0.953)\)  
Velocity – Linear \((r^2 = 0.950)\)
\( \dot{V}O_2 \) – No relationship \( (r^2 = 0.297) \)
HR – No relationship \( (r^2 = 0.113) \)
AMP – Linear decrease \( (r^2 = 0.486) \)

\( r^2 = 0.000 \)

MPF – No relationship \( (r^2 = 0.147) \)
RPE – Quadratic increase \( (R^2 = 0.947) \)
Velocity – No relationship \( (r^2 = \)
Subject 8

\[ \dot{V}O_2 \] – Quadratic \( (R^2 = 0.988) \)

HR – No relationship \( (r^2 = 0.200) \)

AMP – Quadratic \( (r^2 = 0.772) \)

MPF – Linear decrease \( (r^2 = 0.391) \)

RPE – No relationship \( (r^2 = 0.054) \)

Velocity – Quadratic \( (R^2 = 0.920) \)
$\dot{V}O_2$ – Linear decrease ($r^2 = 0.872$)  
HR – No relationship ($r^2 = 0.038$)  
AMP – Linear decrease ($r^2 = 0.429$)  
MPF – No relationship ($r^2 = 0.194$)  
RPE – No relationship ($r^2 = 0.069$)  
Velocity – Quadratic ($R^2 = 0.948$)
$\dot{V}O_2$ – Quadratic ($R^2 = 0.973$)  
HR – No relationship ($r^2 = 0.040$)  
AMP – Quadratic ($r^2 = 0.481$)  

MPF – No relationship ($r^2 = 0.103$)  
RPE – Linear increase ($r^2 = 0.800$)  
Velocity – Quadratic ($R^2 = 0.930$)
Subject 12

\[ \dot{\text{VO}_2} \text{ – Quadratic} \quad (R^2 = 0.683) \]
\[ \text{HR} \text{ – No relationship} \quad (r^2 = 0.041) \]
\[ \text{AMP} \text{ – Linear decrease} \quad (r^2 = 0.594) \]
\[ \text{MPF} \text{ – No relationship} \quad (r^2 = 0.164) \]
\[ \text{RPE} \text{ – Linear increase} \quad (r^2 = 0.767) \]
\[ \text{Velocity} \text{ – Quadratic} \quad (R^2 = 0.927) \]
Subject 13

\[ \dot{V}O_2 \] – Linear decrease \( (r^2 = 0.535) \)
HR – No relationship \( (r^2 = 0.038) \)
AMP – No relationship \( (r^2 = 0.000) \)

Normalized Values (% of peak values)

\[ \text{Normalized Time} \ (% \ of \ T_{lim}) \]

\[ \dot{V}O_2 \] – Linear decrease \( (r^2 = 0.537) \)
MPF – No relationship \( (r^2 = 0.125) \)
RPE – Linear increase \( (r^2 = 0.751) \)
Velocity – Linear decrease \( (r^2 = \)
REFERENCES


86. Poole, DC, and Jones, AM. L-(+)-Lactate infusion into working dog gastrocnemius: no evidence lactate per se mediates $\dot{V}O_2$ slow component. J Appl Physiol. 76: 787-792, 1994.


101. Vanhatalo, A, Poole, DC, DiMenna, FJ, Bailey, SJ, and Jones, AM. Muscle fiber recruitment and the slow component of \( O_2 \) uptake: constant work rate vs. all-out sprint exercise. Am J Physiol Regul Integr Comp Physiol. 300: R700-R707, 2011.


## APPENDIX A

### GLOSSARY

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>AMP&lt;sub&gt;peak&lt;/sub&gt;</td>
<td>Electromyographic amplitude at $\dot{V}O_2$ peak</td>
</tr>
<tr>
<td>MPF&lt;sub&gt;peak&lt;/sub&gt;</td>
<td>Electromyographic mean power frequency at $\dot{V}O_2$ peak</td>
</tr>
<tr>
<td>ARC</td>
<td>Anaerobic running capacity</td>
</tr>
<tr>
<td>ATP</td>
<td>Adenosine triphosphate</td>
</tr>
<tr>
<td>AWC</td>
<td>Anaerobic work capacity</td>
</tr>
<tr>
<td>CHR</td>
<td>Critical heart rate</td>
</tr>
<tr>
<td>CHR-5</td>
<td>Critical heart rate minus 5 b·min&lt;sup&gt;-1&lt;/sup&gt;</td>
</tr>
<tr>
<td>CHR+5</td>
<td>Critical heart rate plus 5 b·min&lt;sup&gt;-1&lt;/sup&gt;</td>
</tr>
<tr>
<td>CP</td>
<td>Critical power</td>
</tr>
<tr>
<td>CV</td>
<td>Critical velocity</td>
</tr>
<tr>
<td>EMG</td>
<td>Electromyography</td>
</tr>
<tr>
<td>EMG AMP</td>
<td>Electromyographic amplitude</td>
</tr>
<tr>
<td>EMG MPF</td>
<td>Electromyographic mean power frequency</td>
</tr>
<tr>
<td>$E_{END}$</td>
<td>Actual metabolic efficiency</td>
</tr>
<tr>
<td>END velocity</td>
<td>The velocity at the termination of exercise at a constant heart rate</td>
</tr>
<tr>
<td>$E_{EST}$</td>
<td>Estimated metabolic efficiency</td>
</tr>
<tr>
<td>GET</td>
<td>Gas exchange threshold</td>
</tr>
<tr>
<td>HB&lt;sub&gt;lim&lt;/sub&gt;</td>
<td>Total number of heart beats</td>
</tr>
<tr>
<td>HR</td>
<td>Heart rate</td>
</tr>
<tr>
<td>HR&lt;sub&gt;peak&lt;/sub&gt;</td>
<td>Heart rate and $\dot{V}O_2$ peak</td>
</tr>
</tbody>
</table>
RCP  respiratory compensation point
RPE  rating of perceived exertion
RPE_{peak}  rating of perceived exertion at $\dot{V}O_2_{peak}$
TD  total distance
$T_{lim}$  time to exhaustion; time limit
$W_{lim}$  total amount of work accomplished; work limit
vCHR  the velocity associated with the critical heart rate
vCHR-5  the velocity associated with the critical heart rate minus 5 b·min$^{-1}$
vCHR+5  the velocity associated with the critical heart rate plus 5 b·min$^{-1}$
$\dot{V}O_2$  oxygen consumption rate
$\dot{V}O_2^{\text{EST}}$  the $\dot{V}O_2$ associated with the velocity at the termination of exercise
$\dot{V}O_2^{\text{END}}$  the $\dot{V}O_2$ at the end of exercise at a constant heart rate
$\dot{V}O_2_{\text{peak}}$  peak oxygen consumption rate
v$\dot{V}O_2_{\text{peak}}$  velocity associated with $\dot{V}O_2_{\text{peak}}$
Title of Research Study
Physiological responses at the critical heart rate during treadmill running

Invitation to Participate
You are invited to participate in this research study. The following is provided in order to help you make an informed decision whether or not to participate. If you have any questions, please do not hesitate to ask.

Basis for Subject Selection
You were selected as a potential subject because you are a moderately trained runner between the ages of 19 and 29 years and in good health. Moderately trained will be defined as running 15 to 30 miles per week, most weeks in the last six months. If you wish to participate you must fill out a health history questionnaire. You will be prevented from participating in this research study if there are indications from the questionnaire that you may have health risks or if you are a pregnant female. Such indications include symptoms suggestive of chest pain, breathing difficulties, irregular heart beat, kidney or liver problems, high blood pressure or cholesterol, and/or abnormal electrocardiogram (EKG). Muscle or skeletal disorders including previous or current ankle, knee, and/or hip injuries may also preclude you from participation in this study. If you have no muscle/skeletal disorders or disease that will prevent you from engaging in physical activity, you will be asked to perform the tests described below. Overall, there are numerous health-related issues that may preclude you from participation in this study and inclusion will be determined on a subject-by-subject basis.

Purpose of the Study
The critical heart rate (CHR) is the highest heart rate that can be maintained for an extended period of time during continuous exercise. The CHR model, however, has not been applied to treadmill running. The purposes of this study are to: 1) determine if the CHR model for cycle ergometry can be applied to treadmill running and 2) examine the oxygen consumption, perception of effort (RPE), and electrical activity of the muscle (electromyographic (EMG) amplitude and EMG mean power frequency) during three constant heart rate runs.

Initials
Explanation of Procedures
You will be asked to visit the Human Performance Laboratory located in Mabel Lee Hall (Room 141) on the UN-L campus on nine separate days, separated by 24 – 48 hours. Visit 1, (141 Mabel Lee Hall), will consist of reading the informed consent, filling out and completing a health history questionnaire, and deciding if you want to participate in the current study. You will then perform a maximal treadmill running test. Before the test begins, the skin on your thigh will be lightly scraped with emery paper at six locations. Six electrodes will then be then be taped to the scraped areas on your thigh for EMG analysis (to measure the electrical activity of your muscle). An electronic goniometer will also be taped to the side of your knee to measure the joint angle. In addition, you will be fitted with a heart rate monitor that will be secured around your chest by an elastic band. You will also be fitted with a mouthpiece, that will be used to collect expired gas samples throughout the test to measure oxygen consumption. You will be asked to give a rating of perceived exertion (how you are feeling) at the end of every minute during the test. Prior to the test, you will warm up for 3 min at 3 miles per hour and then rest for 3 min. The test will begin at 4 miles per hour and the velocity will be increased by 1 mile per hour every 2 min. Following the 9 mile per hour stage, the velocity will no longer be increased, however, the treadmill grade will be increased by 2% every 2 min until you can no longer maintain the running velocity and you grasp the handrails to signal exhaustion. During visits 2 through 6, you will perform 5 constant velocity, randomly ordered treadmill runs at a range of velocities that can be maintained between 3 and 20 min. Oxygen consumption, heart rate, perception of effort, the electrical activity of the muscle (EMG), and joint angle will be measured as previously described. During visits 7 through 9, you will be randomly assigned to run to exhaustion at three different heart rates (heart rate will be kept constant during the test by adjusting the velocity). The test will be terminated when you complete 60 min or you feel you can longer continue and grasp the handrails. Oxygen consumption, heart rate, perception of effort, the electrical activity of the muscle (EMG), and joint angle will be measured as previously described.

Total Time Commitment
The total time commitment for the 9 visits in this study will be approximately 5 to 6.5 hours with each visit lasting approximately 30 min to 60 min. Each visit will be separated by a minimum of 24 hours and all 9 visits must be completed within three weeks from the start of the first session. Visit 1: Orientation and Maximal Test (60 min), visit 2-6: Constant velocity run (30 min each), visit 7-9, Constant HR run (30 to 60 min each).

Potential Risks and Discomforts
The following are the potential risks and discomforts you may experience during this study:

- Electrode Preparation and Use – The use of electrodes and the preparation of the skin for their application may lead to the remote possibility of complications such as a rash or infection.
• Incremental Running Tests – Treadmill running can cause aching in your lower extremities, fatigue, sweating, shortness of breath and discomfort in the chest. However, you can stop running any time you want. Heavy exercise can cause high or low blood pressure, fainting, irregular heart rhythm, chest pain, and very rarely, heart attack, stroke or cardiac arrest. The need for hospital admission is reported in less than six of every 10,000 exercise tests. Cardiac arrest is reported in less than one of every 10,000 exercise tests.

Protection Against Risks
To minimize any potential risks and/or discomforts, you will be given instructions for special stretches, which may aid in the elimination of any muscle soreness as a result of the tests. In addition, you will be asked repeatedly during the tests how you feel in relation to your ability to continue the test. Throughout all the tests, you will be monitored by laboratory personnel trained in Cardiopulmonary Pulmonary Resuscitation (CPR) and use of an Automated External Defibrillator (AED). In addition, you will be asked repeatedly if you feel you can continue the tests. To minimize this risk of rash or infection from the electrode preparation, upon completion of each test, the scraped areas will be cleaned with isopropyl alcohol and an antibacterial salve will be applied to the electrode abrasion sites to prevent any possible infection.

Potential Benefits to Subjects
The main benefit from participating in this study will be feedback on your level of physical fitness. You will gain insight into your own neuromuscular function, an important component of health-related fitness. You will receive a data report of your results from the study. In addition you will have the opportunity to review your results with the principle investigator.

Subject Compensation
You will receive $50 stipend for completing the study. You will be paid when your participation is complete and will not receive compensation for filling out a health history questionnaire or informed consent but failing to participate. Completion of each visit is worth $5.55. If you withdraw after the first visit, the you would receive $5.55. You will receive payment for each completed session when the entire data collection portion of the study is complete.

In Case of Emergency Contact Procedures
If you are injured while you are in Mabel Lee hall during your participation in the study inform one of the investigators who will contact the University Health Center. If you experience an injury as a direct result of the study but are not in personal contact with an investigator please contact the University Health Center or your local health care provider. You may always contact any of the investigators listed at the end of this consent form if you have any questions.

Medical Care in Case of Injury
In the unlikely event that you should suffer an injury as a direct consequence of the research procedures described above, the acute medical care required to treat the injury
can be provided at the University of Nebraska Health Center from the hours of 8:00 a.m.–6:00 p.m. Monday through Friday, and 9:00 a.m.–12:30 p.m. Saturday (for urgent care needs only). The cost of such medical care will be the responsibility of the subject, whether at the University Health Center or at other local health care facilities. If the health center is unable to treat you, emergency care is available at local community health providers. In the case of an adverse event, you may be asked to sign a Private Health Information Authorization form allowing access to your related medical documents for review by the Institutional Review Board and associated personnel.

**Assurance of Confidentiality**

Any information obtained from this study which could identify you will be kept strictly confidential. The information may be published in scientific journals or presented at scientific meetings, but your identity will be kept strictly confidential. All data collected as a result of your participation will be kept in a locked cabinet in the office of the primary investigator (Room 141 Mabel Lee Hall). Your data will receive an identifying number and only the investigators will be able to identify you from your data. Your data will be compiled and only group data will be used for dissemination without identifying your name. For the purposes of future reference, your de-identified data will be stored for a minimum of 15 years.

**Rights of Research Subjects**
You may ask any questions concerning this research and have those questions answered before agreeing to participate in or during the study. Or you may call the investigator, Haley Bergstrom, at any time, office phone, (402) 472-2690, or after hours (308) 325-1363. You may also contact Dr. Terry Housh at his office phone, (402) 472-1160, or after hours (402) 477-6573. Please contact the investigator:

- if you want to voice concerns or complaints about the research
- in the event of a research related injury.

Please contact the University of Nebraska-Lincoln Institutional Review Board at (402) 472-6965 for the following reasons:

- you wish to talk to someone other than the research staff to obtain answers to questions about your rights as a research participant
- to voice concerns or complaints about the research
- to provide input concerning the research process
- in the event the study staff could not be reached.

**Voluntary Participation Withdrawal**

You are free to decide not to participate in this study, or to withdraw at any time without adversely affecting your relationship with the investigators or the University of Nebraska. Your decision will not result in any loss of benefits to which you are otherwise entitled.
You are voluntarily making a decision whether or not to participate in this research study. Your signature certifies that the content and meaning of the information on this consent form have been fully explained to you and that you have decided to participate having read and understood the information presented. Your signature also certifies that you have had all your questions answered to your satisfaction. If you think of any questions during this study, please contact the investigators. You will be given a copy of this consent form to keep.

Signature of Research Participant

Date

Printed name of Research Participant

My signature as witness certifies that the subject signed this consent form in my presence as his/her voluntary act and deed.

Signature of Investigator

Date

Investigators:

Haley Bergstrom
work phone (402) 472-2690
home phone (308) 325-1363

Terry Housh
work phone (402) 472-1160
home phone (402) 477-6573

Initials
PRE-EXERCISE TESTING HEALTH STATUS QUESTIONNAIRE

Subject ID#__________

A. JOINT-MUSCLE STATUS (✓ Check areas where you currently have problems)

Joint Areas
( ) Wrists
( ) Elbows
( ) Shoulders
( ) Upper Spine & Neck
( ) Lower Spine
( ) Hips
( ) Knees
( ) Ankles
( ) Feet
( ) Other__________________

Muscle Areas
( ) Arms
( ) Shoulders
( ) Chest
( ) Upper Back & Neck
( ) Abdominal Regions
( ) Lower Back
( ) Buttocks
( ) Thighs
( ) Lower Leg
( ) Feet
( ) Other_______________

Investigator initials ___

B. HEALTH STATUS (✓ Check if you previously had or currently have any of the following conditions)

( ) High Blood Pressure
( ) Heart Disease or Dysfunction
( ) Peripheral Circulatory Disorder
( ) Lung Disease or Dysfunction
( ) Arthritis or Gout
( ) Edema
( ) Epilepsy
( ) Multiple Sclerosis
( ) High Blood Cholesterol or Triglyceride Levels
( ) Loss of Consciousness
( ) Others That You Feel We Should Know About__________________

( ) Acute Infection
( ) Diabetes or Blood Sugar Level
( ) Anemia
( ) Hernias
( ) Thyroid Dysfunction
( ) Pancreas Dysfunction
( ) Liver Dysfunction
( ) Kidney Dysfunction
( ) Phenylketonuria (PKU)
( ) Allergic Reactions to Medication please describe__________________
( ) Allergic Reactions to Any Other substance please describe__________________

Investigator initials ___
C. PHYSICAL EXAMINATION HISTORY

Approximate date of your last physical examination________________________

Physical problems noted at that time______________________________

Has a physician ever made any recommendations relative to limiting your level of
physical exertion? _______ YES _______ NO

If YES, what limitations were recommended?________________________________

Have you ever had an abnormal resting electrocardiogram (ECG)?
_____ YES ____ NO

Investigator initials_____ 

D. CURRENT MEDICATION AND SUPPLEMENT USAGE (List the drug or
supplement name and the condition being managed)

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<th>CONDITION</th>
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<tr>
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<td>__________</td>
</tr>
</tbody>
</table>

Investigator initials_____ 

E. PHYSICAL PERCEPTIONS (Indicate any unusual sensations or perceptions.
✓Check if you have recently experienced any of the following during or soon after
physical activity (PA); or during sedentary periods (SED))

<table>
<thead>
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<th>PA</th>
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<td>( )</td>
<td>( )</td>
<td>Chest Pain</td>
<td>( )</td>
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<td>( )</td>
<td>( )</td>
<td>Heart Palpitations</td>
<td>“fast irregular heart beats”</td>
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<td>( )</td>
<td>( )</td>
<td>Unusually Rapid Breathing</td>
<td>( )</td>
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<tr>
<td>( )</td>
<td>( )</td>
<td>Overheating</td>
<td>( )</td>
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<td>( )</td>
<td>( )</td>
<td>Muscle Cramping</td>
<td>( )</td>
</tr>
<tr>
<td>( )</td>
<td>( )</td>
<td>Muscle Pain</td>
<td>( )</td>
</tr>
<tr>
<td>( )</td>
<td>( )</td>
<td>Joint Pain</td>
<td>( )</td>
</tr>
<tr>
<td>( )</td>
<td>( )</td>
<td>Other________________________</td>
<td>( )</td>
</tr>
</tbody>
</table>

Investigator initials_____ 

F. **FAMILY HISTORY** (✔ Check if any of your blood relatives . . . parents, brothers, sisters, aunts, uncles, and/or grandparents . . . have or had any of the following)

( ) Heart Disease  
( ) Heart Attacks or Strokes (prior to age 50)  
( ) Elevated Blood Cholesterol or Triglyceride Levels  
( ) High Blood Pressure  
( ) Diabetes  
( ) Sudden Death (other than accidental)

Investigator initials ____

G. **CURRENT HABITS** (✔ Check any of the following if they are characteristic of you current habits)

( ) Smoking. If so, how many per day? ________________________
( ) Regularly does manual garden or yard work
( ) Regularly goes for long walks  
   Hours per week? __________________
( ) Frequently rides a bicycle  
   Hours per week? ________________
( ) Frequently runs/jogs for exercise  
   Miles per week? ________________
( ) Participated in a weight training exercise program within the last 3 months.
( ) Engages in a sports program more than once per week. If so, what does the program consist of?

___________________________________________________________________

Investigator initials ____