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Core Temperature and Maximal Oxygen Uptake During Exercise in the Heat:

Implications for Exercise Prescription

(Under the direction of KIRK J. CURETON)

Previous studies have suggested that attainment of a critical core temperature may limit prolonged, exhaustive exercise in the heat. The aims of this study were to test the hypotheses that large ($> 10\%$) reductions in maximal oxygen uptake ($\dot{V}O_{2max}$) are associated with attainment of a critical core temperature, and that elevation in heart rate during submaximal exercise is related to reduced $\dot{V}O_{2max}$ in the heat. $\dot{V}O_{2max}$ and esophageal temperature at exhaustion (T_{ESOmax}), and oxygen uptake and heart rate during submaximal exercise, were measured in 11 male and 11 female runners under seven conditions designed to manipulate the T_{ESO} at which $\dot{V}O_{2max}$ was reached by varying environmental temperature and preheating. The conditions were: 1) $25^{\circ}C$, no preheating (control); 2-4 and 6) 25, 35, 40, and $45^{\circ}C$, with preheating by a 20-min walk at $\sim 33\%$ of control $\dot{V}O_{2max}$; 5) $45^{\circ}C$, no preheating; and 7) $45^{\circ}C$, with passive preheating raising T_{ESO} to the same degree as at the end of the 20-min walk in Condition 6. Compared to the control condition, $\dot{V}O_{2max}$ was reduced 3-9% under Conditions 2-5 when core temperature at $\dot{V}O_{2max}$ was below the apparent critical core temperature ($< 39.2^{\circ}C$ in men, $< 39.1^{\circ}C$ in women), but was reduced 17-19% under Conditions 6-7 when a critical core temperature appeared to have been attained ($39.6-39.7^{\circ}C$ in men, $39.3-39.4^{\circ}C$ in women). Reductions in time to exhaustion were strongly related to reductions in $\dot{V}O_{2max}$ ($r = 0.82-0.84$). The effects on heat on T_{ESOmax} , $\dot{V}O_{2max}$, and work capacity in men and women in the heat were almost identical. Heart rate and relative metabolic intensity ($\% \dot{V}O_{2max}$) during submaximal exercise increased in an identical fashion in the heat, and the elevations in heart rate were significantly related to the reductions in $\dot{V}O_{2max}$ ($r = 0.79$). It was concluded that large reductions in $\dot{V}O_{2max}$ and work capacity in severe, uncompensable heat stress are associated with attainment of a critical core temperature, and that the reductions in the two are strongly related. Men and women do not differ in T_{ESOmax} , or in the relation of T_{ESOmax} to the reductions in

$\dot{V}O_{2\max}$ and work capacity, in the heat. Elevation in heart rate during submaximal exercise in the heat is proportional to reduced $\dot{V}O_{2\max}$, and not indicative of dissociation between heart rate and % $\dot{V}O_{2\max}$.

INDEX WORDS: Critical core temperature, Maximal oxygen uptake, Work capacity, Heart rate, Relative metabolic intensity, Heat stress, Gender, Treadmill exercise, Submaximal exercise

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IMPLICATIONS FOR EXERCISE PRESCRIPTION

by

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DEDICATION

I dedicate this work to my future wife, Gunnhildur. For her willingness to relocate to another country far away from her family for many years. For her love, contagious laughter, and endless support throughout my studies. For all the things that make her “simply the best”.

Ég tileinka verk þetta tilvonandi eiginkonu minni, Gunnhildi. Fyrir viljann til að flytja til annars lands fjarri heimaslóðunum í mörg ár. Fyrir ást hennar, smitandi hlátur, og endalausán stuðning í gegnum mitt nám. Fyrir allt sem lætur hana bera af öllum öðrum.

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CHAPTER 1

INTRODUCTION

Maximal oxygen uptake ($\dot{V}O_{2\max}$) is the highest rate oxygen can be taken up into the body and sets the upper limit of energy supply through aerobic metabolism (10). It is frequently used as a measure of functional capacity of the cardiovascular system (188) and as an index of cardiorespiratory fitness (107). $\dot{V}O_{2\max}$ also is a good predictor of performance in prolonged, strenuous exercise (206). Since $\dot{V}O_{2\max}$ is such an important measure of cardiorespiratory capacity and performance, knowledge of the factors limiting $\dot{V}O_{2\max}$ is important.

It has been shown that pulmonary function does not limit oxygen transport in normal healthy untrained subjects (113,148), but it may be limiting in elite endurance athletes (56,177). Reduced partial pressure of oxygen during heavy exercise in athletes has been hypothesized to result from rapid blood transit time (178), ventilation perfusion inequalities (178), and high energy cost of breathing at high rates of oxygen uptake ($\dot{V}O_2$) (56,178). Similarly, muscle metabolic capacity does not limit $\dot{V}O_{2\max}$, because peak leg blood flow during one-leg exercise is much higher than during two-leg exercise (2), alteration of the oxygen transport capacity of the blood increases $\dot{V}O_{2\max}$ (50,79,118), and oxidative enzyme activity in muscle exceeds the maximal rate of oxygen uptake per kilogram of the muscle (83). The muscle metabolic capacity only limits $\dot{V}O_{2\max}$ or peak $\dot{V}O_2$ ($\dot{V}O_{2\text{peak}}$) during small muscle mass activity (41,181,223) and during hypoxemia (103).

In normal healthy subjects under thermoneutral conditions, $\dot{V}O_{2\max}$ is most likely limited by the capacity of the circulatory delivery of oxygen (9,188), since cardiac output reaches a maximum as $\dot{V}O_{2\max}$ is attained and does not increase further at higher

work rates (169,259). In addition, studies have shown that addition of muscle groups to those already eliciting $\dot{V}O_{2\max}$ does not increase $\dot{V}O_2$ in proportion to the extra muscle mass activated (125,239). Furthermore, reduction in plasma or blood volumes decrease $\dot{V}O_{2\max}$ (79,118).

In a hot environment, other factors might limit $\dot{V}O_{2\max}$. Since many people live, exercise, and compete (perform) in hot climates, the body's response to exercise in the heat has been extensively studied. Some studies suggest that high ambient temperature might reduce $\dot{V}O_{2\max}$ slightly (3-6%) (124,197,204,244), whereas other studies reported no effect (193,200,259). Pirnay et al. (174) was the first to observe a marked decrease (25% or 985 ml/min) in $\dot{V}O_{2\max}$ when measured in the heat. Their protocol differed from those employed in the earlier studies in that $\dot{V}O_{2\max}$ was measured following low-intensity exercise that elevated core temperature (preheating). Small (6-8%) to moderate (16%) decreases in $\dot{V}O_{2\max}$ following preheating were later observed by others (167,208,215).

Endurance performance also is compromised by high environmental temperatures (1,23,45,71,77,130,140,161,197,204,208,225). In general, the greater the duration, the more the performance is affected. Considerable data indicate that hyperthermia (high core temperature) may limit exercise performance in the heat. In studies that have manipulated initial core temperature or the rate of rise in temperature (76,87,130,254), termination of exercise occurred at an identical core temperature under all experimental conditions. Termination of exercise at a critical level of hyperthermia has been observed by others (37,38,43,46,71,93,128,141,142,150,161,165-167,214,225, 243,247,263). Studies involving lowering of core temperature prior to exercise (precooling) have found enhanced performance in neutral or warm ($\sim 30^\circ\text{C}$) temperatures (19,25,100,129,168,217), but no studies have been conducted on the effect of precooling and performance in uncompensably hot environment.

A common explanation for the possible reduction in $\dot{V}O_{2\max}$ and the reduction in performance in the heat is an hypothesized reduction in muscle blood flow, due to pooling of blood in the skin (190). Exercise in a hot environment causes increased skin blood flow to dissipate heat (201). Stroke volume decreases due to pooling of blood in the skin, partly due to less effective muscle pump (190) and partly because of withdrawal of vaso- and venoconstrictor outflows to skin (195,201). During mild exercise, the decreased stroke volume is compensated for through higher heart rate resulting in slightly higher cardiac output (156,163,199). The tachycardia observed at a given submaximal absolute intensity in the heat compared to a thermoneutral environment (22,59,73,156,172,193,197,200,219,234,259), suggests that there is dissociation between heart rate and submaximal relative metabolic intensity ($\% \dot{V}O_{2\max}$), if $\dot{V}O_{2\max}$ is not reduced in the heat (193,200,208,259). During heavy exercise, on the other hand, heart rate is close to maximum and, therefore, cannot compensate for reduced stroke volume, resulting in lower cardiac output (86,200), lower skeletal muscle blood flow, and lower $\dot{V}O_{2\max}$. Basically, the cardiovascular system is forced to its functional limits by the heat stress at submaximal levels of $\dot{V}O_2$ (200), but with blood pressure maintained, probably through vasoconstriction in active muscle and skin (20,134,156). Such cardiovascular strain associated uncompensable heat stress would lower work capacity (37,38,46,128,141-143,150,214,216,225,247).

More recently, a different explanation for the reduction in performance in the heat has been proposed. It is that muscle blood flow is not reduced, but the high core temperature attained during exercise in the heat causes premature fatigue (71,84,85,87, 159-161,165,166,213). Savard et al. (213) measured leg blood flow directly during moderate exercise under heat stress and found it unaltered. That finding was later replicated during severe exercise (161,165,166). Savard et al. (213) also found that the maximal skin blood flow was the same in one-legged and two-legged exercise, but the two-legged exercise resulted in much higher core temperature, presumably because of

vasoconstriction in cutaneous vessels during two-legged exercise, due to the higher metabolic demand of that exercise. Such plateauing of or slight drop in skin or forearm blood flow or its rate of rise has been demonstrated by others (20,87,156,161,166,246, 256,259). These findings suggest that under circumstances of high metabolic and thermoregulatory demands, venoconstriction in the skin maintains muscle blood flow at the expense of thermoregulation, resulting in a rise in core temperature.

Further support for hyperthermia limiting exercise performance comes from studies that have found exhaustion to occur at similar core temperature regardless of ambient/water or skin temperatures (43,46,87,130,225), initial core temperature (87), rise in core temperature (87), exercise duration (37,38,87,141,150,161,166,167,214, 225,247), exercise intensity (38,141,150,214), rest periods during exercise (214), climate (150), acclimatization stage (37,161,166), state of hydration (37,38,128), fluid ingestion (38,141), stage in menstrual cycle (247), melatonin intake (142), or carbohydrate availability (71). The critical core temperature can vary with subject characteristics, exercise, dehydration, and the environmental conditions (92). The critical temperature is higher in trained than untrained subjects (37) and is lower during protocols involving prolonged, low intensity exercise to exhaustion in uncompensable heat stress (37,38,142,143,150,216,225,247), dehydration (216), full protective clothing (150), very humid environment (143), and in the morning (142). High core temperature also may limit $\dot{V}O_{2\max}$ during exercise in the heat if it is linked to a maximum cardiovascular response and causes termination of exercise at a work rate below that needed to elicit $\dot{V}O_{2\max}$ under thermoneutral conditions.

The mechanisms underlying the reduction in performance, and possibly in $\dot{V}O_{2\max}$, as a result of high core temperature have not been identified. The high core temperature is thought to affect central nervous system and mental functioning, and the discomfort from heat storage may counteract motivation and reduce the drive to exercise (25,161,165). These mechanisms may prevent rise in core temperature to

levels that cause heat injury and jeopardize cell functions (30,76,87,112,254). In accordance with that hypothesis, most animals and humans terminate exercise at a core temperature around 40° C (76,84,85,87,130,161,165,166,213,254), whereas heat injury results from sustained exposure to temperatures above this (75,93,109,110).

Alternatively, the cardiovascular strain in uncompensable heat stress could be the cause of the reduction in performance and $\dot{V}O_{2max}$ (37,38,46,128,141-143,150,214,216,225, 247). Heat stress can divert blood from the working muscles to the skin for cooling, resulting in lower stroke volume and $\dot{V}O_{2max}$ (190,200).

Almost all of the evidence supporting the thermal limit for performance (37,38,46,71,87,128,130,141,142,150,161,166,167,214,225,247), and possibly $\dot{V}O_{2max}$ (59,167,174,204,208,215,219), has been obtained in males. Whether the effect of high core temperature on $\dot{V}O_{2max}$ and performance in women is qualitatively or quantitatively different from that in men is unknown.

Statement of the Problem

The problem of this study was to determine whether large (> 10%) reductions in $\dot{V}O_{2max}$ during exercise in the heat are associated with attainment of a critical core temperature in endurance-trained men and women. A second problem was to determine whether the elevation heart rate resulting from sustained exercise in the heat is related to reduced $\dot{V}O_{2max}$.

Hypotheses

It was hypothesized that during exercise in the heat:

- 1) Large (> 10%) reductions in $\dot{V}O_{2max}$ in endurance-trained men and women are obtained only under conditions in which a critical core temperature is reached.
- 2) Reduced physical work capacity is related to reduced $\dot{V}O_{2max}$.
- 3) There is no gender difference in the effect of core temperature on $\dot{V}O_{2max}$.

4) The higher relative metabolic intensity resulting from a reduced $\dot{V}O_{2\max}$ is associated with the elevation in heart rate during sustained submaximal exercise in the heat.

Significance of the Study

The study contributes new information to the literature on the mechanism through which $\dot{V}O_{2\max}$ is reduced in the heat. A few studies have reported $\dot{V}O_{2\max}$ to be reduced under hot environmental conditions (59,124,174,175,197,204,208,215,219,244), but only two (167,174) have showed a marked decrease. The basis for the large reduction observed by Pirnay et al. (174) is unknown. It is hypothesized that the prior submaximal exercise in their study elevated core temperature so that the critical level of hyperthermia that causes fatigue was reached prior to attainment of the work rate that elicited $\dot{V}O_{2\max}$ under thermoneutral conditions. Studies also have reported that exhaustion in sustained, aerobic exercise occurs at a critical core temperature regardless of environmental conditions (37,38,43,46,71,87,128,130,141,142,150,161,166,167,214,225,247), but no one has investigated the effects of the critical core temperature on $\dot{V}O_{2\max}$. In addition, almost all of the studies that have investigated the effect of environmental heat on $\dot{V}O_{2\max}$ and the core temperature at exhaustion have used men, so whether the effects of environmental and core temperature on $\dot{V}O_{2\max}$ and performance in women are the same as in men is unknown. This study systematically manipulated core temperature through exercise and passive heating under different environmental conditions such that the critical core temperature was reached under some conditions, but not others.

The study also has practical significance, since many people live, exercise, and compete in hot climates and they are interested in the effects of environmental temperature on $\dot{V}O_{2\max}$ and performance. If $\dot{V}O_{2\max}$ is limited by attainment of a critical core temperature in the heat, it suggests that once people exercise for a moderate time in the heat, even at low intensities, and elevate core temperature, work capacity is reduced

and performance is negatively affected. This suggests that actions preventing a rise in core temperature during warm-up, exercise, or work in the heat, such as precooling, would potentially enhance performance. The data obtained provides new information and improve understanding of the physiological factors that limit performance and contribute to premature fatigue in the heat.

Furthermore, this study has practical implications for exercise prescription in the heat. Generally, it has been recommended to exercise at the same heart rate in the heat as in a thermoneutral environment, because heart rate is sensitive to heat stress and provides an index of the overall physiological strain (108). Since heart rate at a submaximal intensities is elevated in the heat, exercise intensity must be reduced to maintain the same heart rate as in thermoneutral environment, resulting in lower absolute $\dot{V}O_2$. If $\dot{V}O_{2max}$ is unchanged in the heat (193,200,208,259), the relative intensity of exercise in the heat is lower and the training stimulus for an increase in $\dot{V}O_{2max}$ is decreased (220,257). However, if the findings of this study demonstrate a relation between relative exercise intensity and elevation in heart rate during submaximal exercise, then one might be working at a similar relative intensity in the heat when exercising at the same heart rate as in a thermoneutral environment. That finding could mean that the training stimulus for the increase in $\dot{V}O_{2max}$ in the heat and in neutral environment is the same. Furthermore, such findings would provide a scientific basis for the recommendation to exercise at the same heart rate in the heat as in a thermoneutral environment.

CHAPTER 2

REVIEW OF THE RELATED LITERATURE

In this section, the literature relevant to the study is discussed. It starts with a discussion of maximal oxygen uptake ($\dot{V}O_{2\max}$). Then, effects of environmental and core temperature on $\dot{V}O_{2\max}$ and performance are reviewed. Finally, factors that limit $\dot{V}O_{2\max}$ and performance under hot environmental conditions are addressed.

Maximal Oxygen Uptake

Measurement of maximal oxygen uptake. Maximal oxygen uptake is the highest rate that oxygen can be taken up into the blood during physical work (10). It reflects the highest rate that energy can be supplied through aerobic metabolism. It is generally accepted as the best measure of the functional limit of the cardiovascular system (147,158,188,245) and is commonly interpreted as an index of cardiorespiratory fitness (107). The actual value of $\dot{V}O_{2\max}$ depends upon the nature of the physical activity (9,18,116,151,239).

The most widely accepted criterion for attainment of $\dot{V}O_{2\max}$ is the demonstration of a plateau or that there is no further increase in oxygen uptake despite an increase in workload (10,221,244). Oxygen uptake ($\dot{V}O_2$) increases linearly as function of exercise intensity until $\dot{V}O_{2\max}$ is reached; after that, $\dot{V}O_2$ increases only slightly, remains the same, or decreases. An increase in $\dot{V}O_2$ less than 150 ml/min or 2.1 ml/kg/min for a 2.5% increase in grade has commonly been used to determine that $\dot{V}O_{2\max}$ has been reached based on the work of Taylor et al. (244). More stringent criteria for plateauing have been used (50-60 ml/min) (148), but these approach the limit of the ability to measure a real difference in $\dot{V}O_2$ (107). Other indicators of maximal effort have been used to address the likeliness of $\dot{V}O_{2\max}$ attainment and they

are: 1) blood lactate concentrations above 7 mmol/l (10), 2) heart rate within 1 standard deviation of age-predicted maximal heart rate (221), 3) respiratory exchange ratio (RER) between 1.0-1.15 (17,111,188,221) and 4) ratings of perceived exertion (RPE) of 19 or 20. These are indicators of maximal effort, however, and do not provide direct evidence about whether $\dot{V}O_{2max}$ was attained.

The success rate in reaching a plateau ranges typically from 50-100% of the subjects in most reports in the literature (68,120,148,176,244) even though lower success rates (7-35%) have been reported (47,74). Children, low-fit people and the elderly have more difficult time in reaching a plateau and the criterion represents a larger fraction of their $\dot{V}O_{2max}$. An intermittent protocol increases the number of subjects reaching a plateau, and motivated subjects are more likely to reach a plateau (47,49,107,222,226,244). Wyndham et al. (262) also cautioned that a plateau could underestimate $\dot{V}O_{2max}$ and suggested an alternate statistical curve fitting the oxygen uptake-work load relationship. That statistical fitting is impractical, though, because of the number of observations needed (17).

Most studies report that $\dot{V}O_{2max}$ is highly reliable. Using increases in $\dot{V}O_2$ less than 2.1 ml/kg/min with 2.5% increase in grade as a criterion for plateau, Taylor et al. (244) report a reliability coefficient of 0.95 with standard error of measurement 0.84 ml/min or 2.4% of the overall mean. Furthermore, they state that less than 1% of the variance in $\dot{V}O_{2max}$ was accounted for by day-to-day variability. Kasch et al. (119) report the greatest difference between test-retest determination of bench stepping $\dot{V}O_{2max}$ was 1.6 ml/kg/min. Other studies have reported reliability coefficients in excess of 0.9 during test-retest of $\dot{V}O_{2max}$ during cycling, running, swimming, rowing, and ice skating (48,72,131,136,137,148) and Wyndham et al. (262) report a coefficient of variation for $\dot{V}O_{2max}$ over four-month period of 4.3%. Despite such a high reliability, Glassford et al. (78) reported higher $\dot{V}O_{2max}$ values in their subjects when retested after the subjects had reached the criterion for attainment of $\dot{V}O_{2max}$ on a different day.

Reliability coefficients are lower (0.71-0.76) in inexperienced subjects (48) and children (49) and depended upon demonstration of a plateau; without a plateau the reliability coefficient was low (49). However, Froelicher et al. (74) report a coefficient of variation of 4.1% despite only 33% of their subjects demonstrated a plateau using the Taylor et al. (244) protocol.

Various protocols and tasks have been used to measure $\dot{V}O_{2max}$. Generally, the task should involve larger muscle groups such as the legs or arms and legs (at least 50% of the muscle mass), work loads should be measurable and reproducible, duration of work should be at least 2-3 min but not much longer than 12 min, the mechanical efficiency should be similar in the population tested, and the test should be appropriate for those tested (10,188,221). Most of the tests used to measure $\dot{V}O_{2max}$ involve graded or progressive exercise (9,33,36,49,66,68,74,78,99,106,116,119,131,136,138,139,148, 176,237,244,260,262), although a single maximal or supramaximal work bout has been used (48,66,82,106,131,133,149,158,185,232). Longer work bouts may be needed in supramaximal protocols, since the half-time of the $\dot{V}O_2$ response at the onset of exercise increases with increasing intensity (91). These tests can be administered continuously or discontinuously with rest periods, with little difference in $\dot{V}O_{2max}$ between the two (132,136,151,221,230,260).

The highest oxygen uptake that can be measured during exercise depends upon the exercise mode. The most common exercise modes used for testing are treadmill walking and running, cycling, and bench stepping, but $\dot{V}O_{2max}$ has also been measured during swimming, skiing, arm cranking, ladder climbing, combined arm and leg work, and other forms of exercise (8,9,18,43,48,82,116,131,218,228,239,244).

Generally, the highest oxygen uptake is obtained during uphill running (100%) followed by skiing, level running, bench stepping, and simultaneous arm and leg exercise (95-97%); cycling and walking (90-95%); rowing and swimming (85-90%); supine cycling (82%); and arm cranking (65-70%) (8,9,18,36,48,68,78,98,99,116,119,

136,138,139,149,151,176,221,228,237,239,244,260). The speed of running or walking does not affect $\dot{V}O_{2\max}$ values as long as normal running and walking strides are used (237,244).

The differences in $\dot{V}O_{2\max}$ during various exercise modes is probably due to different amount of muscle utilized in the exercise. That has been demonstrated by an increase in oxygen uptake when adding arm exercise to leg exercise such as simultaneous arm and leg cycling (82,218). Not everyone has found such increase when adding arm exercise (9,239), but these studies did not increase the total work rate in simultaneous exercise above that used for leg exercise alone. Higher values for $\dot{V}O_{2\max}$ were found during ladder climbing than during uphill running in women but not in men, presumably due to lower stepping rate in women (116). Similarly, lower step rate in uphill running compared to horizontal running has been suggested as one of the reasons for lower $\dot{V}O_{2\max}$ values in horizontal running (116).

Task familiarity may also play a role in the differences in oxygen uptake observed between various exercise modes. It has been demonstrated that championship cyclists can reach just as high $\dot{V}O_{2\max}$ values on the bike as during running (158) and that the difference between cycling and running is less in a population more accustomed to cycling (78). Furthermore, highly competitive swimmers can reach just as high oxygen uptake during swimming as during uphill running, whereas less skilled subjects demonstrated up to a 25% decrement in $\dot{V}O_{2\max}$ (131).

Finally, local fatigue has been suggested as a reason for lower $\dot{V}O_{2\max}$ in cycling and bench stepping (221,231). Shephard et al. (221) concluded on clinical grounds (based on visual and verbal inspection of the subjects) that the factor limiting treadmill exercise was a central exhaustion but cycle exercise was limited by local exhaustion in the legs (pain in the quadriceps). Bench stepping was thought to be limited by a combination of local and central exhaustion. Also, increases in workload during bench

stepping by increasing the height of the bench results in greater local muscular fatigue than if step rate is increased (119).

The measurement errors associated with $\dot{V}O_{2\max}$ result from error in measuring minute ventilation, and the oxygen (O_2) and carbon dioxide (CO_2) fractions of expired air (107). In addition, the barometric pressure, gas temperature, and water vapor pressure of the gas enter into the calculation of $\dot{V}O_{2\max}$, but these variables contribute little to the measurement errors, since very large errors (7-8 mmHg or 3° C) are needed for 1% change in $\dot{V}O_{2\max}$ (107). Maximal error in the gas fractions are generally less than 1.5-2% and, therefore, the maximal errors of combined effects of random error in volumes and gas fractions should be no more than 3% of $\dot{V}O_{2\max}$ or ~120-130 ml/min (107,120). These are only the inaccuracies that would result from random errors in the measuring instruments, but such errors are generally less than 10% of the total error of measurements (120,241), with biological variability accounting for 90% or more of the total within-subject variability in $\dot{V}O_{2\max}$ (120). During high steady state levels of $\dot{V}O_2$, the minute variation should rarely exceed 100 ml/min and the coefficient of variation has been found to range from 2-5% at such levels (107). Despite such a small variation, it must be kept in mind that the Fick principle on which the calculation is based, is strictly only applicable during steady state, which $\dot{V}O_{2\max}$ is not, resulting in higher absolute error and variability. Nevertheless, studies have found coefficients of variation around 4% for repeated measures of $\dot{V}O_{2\max}$ (74,120,255,262), which is very similar to the values reported for steady state exercise (107,255).

Sampling time also may introduce error in the measurement of $\dot{V}O_{2\max}$ (107,153). Myers et al. (153) found that the standard deviation (SD) of $\dot{V}O_2$ was highest during breath-by-breath sampling (4.5 ml/kg/min) and lowest during 60-s sampling (0.8 ml/kg/min). Other sampling intervals falling in between the two had SD of 1.3-3 ml/kg/min. Collection periods shorter than 60 s magnify the random errors of the collection interval (107). In addition, the inherent variability in the non-steady state

condition and the common use of the largest value as $\dot{V}O_{2\max}$, which introduces a systematic error with bias towards to the most extreme value, can result overestimation of $\dot{V}O_{2\max}$ of 200 ml/min or more (107). However, despite the 60-s sampling period having the least variability, it may be too imprecise for evaluating certain interventions, therefore, a 30-s sampling period (with SD of ~1.3 ml/kg/min) offers high precision and might be considered reasonable for standard incremental testing (153).

Limitations of Maximal Oxygen uptake. Limitations for $\dot{V}O_{2\max}$ could be of central (pulmonary or cardiovascular limitations) or peripheral (muscle) origin. Pulmonary factors impose no limitation on oxygen transport in normal healthy subjects (10,57,113,148,207). That is probably because of the completeness and efficiency of ambient air to arterial blood oxygen transfer (57,148,207). Even at maximal levels of exercise below 5000 feet, partial pressure of oxygen in arterial blood (PaO_2) does not decrease much, resulting in an arterial blood oxygen saturation (% SaO_2) similar to rest (113). Furthermore, the peak ventilation at exhaustive exercise is only 70-80% of the functional capacity of the ventilatory apparatus (55,57) and ventilation continues to increase as intensity is increased beyond $\dot{V}O_{2\max}$, indicating that the maximal voluntary ventilation is not reached at $\dot{V}O_{2\max}$ (10). The respiratory muscles deprive the working muscles of less than 7% of the total oxygen intake at maximal exercise by one estimate (145) and others (54) have estimated that the cost of respiratory work at $\dot{V}O_{2\max}$ is about 8-10%, and never higher than 15%, of the total oxygen cost. Since peak perfusion of limb locomotor muscle has been estimated to be 80-85% of cardiac output (4,54,191,253), respiratory work does not seem to limit $\dot{V}O_{2\max}$ in normal healthy subjects.

However, in some elite endurance athletes, pulmonary function may limit $\dot{V}O_{2\max}$. In these types of athletes, during heavy exercise and at $\dot{V}O_{2\max}$, PaO_2 and % SaO_2 can be markedly reduced, indicating that pulmonary gas exchange is inadequate (55,56,177,203). When these athletes breathe 23.9% O_2 , the PaO_2 is restored to values

normally seen at maximal exercise (56). The decrease in PaO_2 is most likely due to extremely rapid transit time through the lungs so that adequate oxygenation of the blood is impossible, resulting in attainment of $\dot{V}\text{O}_{2\text{max}}$ prematurely (178). At an oxygen uptake higher than 4.5 liters and at maximal pulmonary capillary blood volume, mean red blood cell transit time could be as low as 0.25 s (178), far below the minimum time (~ 0.35 s) required for complete oxygenation of arterial blood (114,178) and nearly 25% of the distribution of transit times would be less than 0.2 s (178). Ventilation perfusion inequalities may also be responsible for part of the widening of the alveolar-arterial oxygen tension difference, since ventilation perfusion inequality has been shown to increase with rising oxygen consumption (178). Furthermore, breathing at extremely high metabolic rates may be approaching the mechanical limits of airflow velocity and/or exceeding the optimal linear range of the pressure-flow relationship (178). Dempsey et al. (56) found that tidal volumes of highly trained runners exceed flow rates of 5-6 l/min, which matches or exceeds the limits of resting flow volume curve resulting in hypoventilation. They also found that breathing a normoxic helium-oxygen gas mixture increases PaO_2 , probably due to unloading of the respiratory muscles. On the other hand, Bender and Martin (13) concluded that respiratory muscle fatigue does not occur in short-term heavy exercise.

The circulatory transport of oxygen most likely limits $\dot{V}\text{O}_{2\text{max}}$ in most healthy athletes and non-athletes (9,33,188). Cardiac output plateaus and reaches a maximum as $\dot{V}\text{O}_{2\text{max}}$ is attained and does not increase further at higher work rates (169,259). Also, addition of extra muscle groups, such as in simultaneous arm and leg cycling, to those already eliciting $\dot{V}\text{O}_{2\text{max}}$ does not increase oxygen uptake (9,33,116) or the increase is small and not in proportion to the extra muscle mass activated (9,82,125,218,239,244). Since most of these studies are conducted on simultaneous arm and leg cycling and since, as stated above, the $\dot{V}\text{O}_{2\text{max}}$ during cycling is around 10% lower than during running, a 10% rise in $\dot{V}\text{O}_{2\text{max}}$ when arm work is added to leg work would be expected.

The cardiac output cannot be increased through additional muscle vasodilation because recruitment of more muscle mass at $\dot{V}O_{2max}$ causes vasoconstriction in active muscle to maintain arterial pressure (218). Furthermore, following training when both legs are trained separately, total body $\dot{V}O_{2max}$ and single-leg $\dot{V}O_{2max}$ increase to much greater extent during single-legged cycling than during two-legged cycling (50,51,125,210).

In addition, manipulations of plasma or blood volumes through detraining, and blood withdrawal or infusion that change stroke volume and cardiac output, change $\dot{V}O_{2max}$ (44,79,117,118), suggesting that oxygen delivery to the muscle and not the utilization by the muscle is the limiting factor. However, expansion of plasma volume in trained people with normal blood volume will not increase $\dot{V}O_{2max}$ (44,79,117,118), because any increases in cardiac output are offset by the decrease in hemoglobin concentration ([Hb]) (117). When whole blood is infused under these normal conditions to maintain [Hb] as blood volume expands, cardiac output may not increase due to increased viscosity of the blood, resulting in unaltered $\dot{V}O_{2max}$ (184,211). Nevertheless, it is clear that reductions in blood volume will decrease cardiac output and $\dot{V}O_{2max}$ (44,79,117,118,249). Finally, training data shows that large muscle activity increases cardiac output and $\dot{V}O_{2max}$, but training with limited muscle mass such as the arms or one leg increases peak oxygen consumption ($\dot{V}O_{2peak}$) during that form of exercise, but not in exercise involving larger muscle mass such as two-legged cycling or during exercise with the untrained leg (41,51,81). Thus, small muscle mass exercise elicits peripheral, but not central, adaptations resulting in unchanged $\dot{V}O_{2max}$.

If $\dot{V}O_{2max}$ was limited by the muscles' metabolic capacity, then an individual's $\dot{V}O_{2max}$ would be a function of muscle mass and not be reached until maximum use of all synergistic muscles was achieved (188). The same would be true for maximal cardiac output and arterial-venous oxygen saturation difference ($A-VO_{2diff}$) (188). Stenberg et al. (239) have shown that while arm cranking $\dot{V}O_{2max}$ is only 66% of two-legged cycling one, the cardiac output during maximal arm cranking is 80% of that

during two-legged cycling. Similar differences between percentages of cardiac output and $\dot{V}O_2$ of maximum when comparing small and large muscle mass exercise have been reported by others (33,181).

Muscle blood flow capacity does not seem to limit $\dot{V}O_{2max}$. Measurements of peak muscle blood flow per gram of active muscle during one-legged exercise indicate that only one-third of the muscle mass would be needed for the cardiac output to reach its upper limit, suggesting that muscle blood flow capacity is not the limiting factor in exercise involving large muscle mass (2,223). Furthermore, the partial pressure (PvO_2) and saturation (% SvO_2) of oxygen in venous blood are 10 mmHg and 10% higher in one-legged exercise than the values normally seen with maximal two-legged exercise, supporting the notion that flow is not limiting for muscle oxygen uptake (2). In addition, during hypoxemia (breathing 10-11% oxygen), muscle blood flow is increased above that observed during peak small muscle activity, resulting in the same oxygen uptake (202). Others have also reported somewhat higher leg blood flow during one-legged compared to two-legged exercise or during leg work compared to simultaneous arm and leg work (125,218).

The muscle oxidative capacity does not appear to limit $\dot{V}O_{2max}$ in large muscle activity. When the partial pressure or concentration of inspired oxygen is increased, $\dot{V}O_{2max}$ increases (50,63,67,133,261), suggesting that once the muscles receive more oxygen from the circulation, they can extract and use it. Similar changes in $\dot{V}O_{2max}$ have been reported when oxygen delivery to the muscle is altered through changes in hemoglobin concentration (26,62,64,79,118,183,249,250). In addition, it has been demonstrated from studies of enzyme activity in skeletal muscle that the metabolic capacity of both untrained and trained muscle normally exceeds the maximal rate of oxygen uptake per kilogram of the muscle, and the increase with conditioning by far exceeds the increase in $\dot{V}O_{2max}$ (83). Neither capillarization nor the concentration of mitochondrial enzymes are limiting to whole body aerobic power in healthy men; the

capacity of muscle to utilize oxygen far exceeds the capacity of the circulation to deliver it (209).

In general $\dot{V}O_{2\max}$ does not seem to be limited peripherally in large muscle activity. However, there is some evidence from studies in dogs that during hypoxemia, the capillary-to-mitochondria diffusion gradient might be limiting, since $\dot{V}O_{2\max}$ was reduced in direct proportion to decreasing PvO_2 in hypoxemia (103). Despite this fall in $\dot{V}O_{2\max}$, oxygen extraction was 10% higher during hypoxemia. The authors argue that if oxygen delivery was limiting $\dot{V}O_{2\max}$, oxygen extraction should be complete as $\dot{V}O_{2\max}$ declined during hypoxemia and that oxygen delivery is only important in determining partial pressure of oxygen in the capillaries (driving pressure) (103). Unfortunately, no data were collected during hyperoxemia.

Under normoxic conditions, the only time the metabolic factors, and not blood flow, limit $\dot{V}O_{2\max}$ is during use of a small muscle mass (41,181,223). Peak cardiac output in activities using a small muscle mass is lower than the maximal cardiac output measured during maximal exercise with a large muscle mass (81,181). Compared to large muscle mass exercise, the cardiac output can be raised to a higher relative extent (66-87%) than $\dot{V}O_{2\text{peak}}$ (60-77%) during small muscle mass exercise, suggesting that cardiac output can be increased without an increase in $\dot{V}O_{2\text{peak}}$, hence a peripheral limitation (81,181,239). Also, in hypoxemia the cardiac output can rise to even higher peak values without apparent limit when the mass of the active muscle is too small to overwhelm the pumping capacity of the heart (202). In addition, training can increase $\dot{V}O_{2\text{peak}}$ in activities involving limited muscle mass without increasing maximal cardiac output or whole body $\dot{V}O_{2\max}$ (41,51). Finally, the $\dot{V}O_{2\text{peak}}$ in small muscle mass activities is linearly related to the active muscle mass, and reducing oxygen delivery (breathing 12% O_2) has much smaller effect on $\dot{V}O_{2\text{peak}}$ in activities involving small muscle mass than large muscle mass, indicating that the $\dot{V}O_{2\text{peak}}$ for these activities is limited peripherally (223).

In summary, “true” $\dot{V}O_{2\max}$ is most likely obtained during uphill running of greater than 4 min in duration due to the large muscle mass used in that type of activity. The measurement of $\dot{V}O_{2\max}$ is reliable, and the variability in the measure is mainly biological in nature. In normal healthy individuals utilizing large muscle mass activity, $\dot{V}O_{2\max}$ is typically limited by the capacity of the circulatory system to deliver O_2 to the working muscles.

Effects of Environmental Temperature on Maximal Oxygen Uptake

Since many people live, work, and exercise in hot climates, there has been considerable interest in the effects of environmental heat on $\dot{V}O_{2\max}$. The first to report any effect of heat on $\dot{V}O_{2\max}$ were Taylor et al. (244) who found a small but significant decrease in $\dot{V}O_{2\max}$ (150 ml/min or 4%) in 32° C compared to their control condition of 25.5° C. However, studies that followed, investigating the effects of hot environment (36-43° C) on $\dot{V}O_{2\max}$, found no reduction (193,200,259).

Other studies conducted shortly thereafter, reported that $\dot{V}O_{2\max}$ might be reduced (3-5%) under hot conditions, but these studies had design limitations (124,197). Klausen et al. (124) found a significant negative correlation between air temperature and $\dot{V}O_{2\max}$ over a series of measures in the desert, but their control tests (in 25° C) were not done until four months later. Furthermore, the desert portion of the study was conducted at modest altitude while the control tests were at sea level, so the observed differences between the control and the desert conditions could be due to the altitude (28,236). Other problems with these studies were lack of subjects, and work prior to the $\dot{V}O_{2\max}$ test in the heat, resulting in failure to reach work rates sufficient to elicit control $\dot{V}O_{2\max}$ (197).

The short maximal protocol used in some of these early studies could be the reason for the lack of decrease in $\dot{V}O_{2\max}$ in the heat. The high workload from the beginning would favor a redistribution of blood flow to the working muscle and prevent pooling of blood in the skin, and the short protocol would not allow core temperature to

rise much. Using preheating, Pirnay et al. (174) were the first investigators to show a marked decrease in $\dot{V}O_{2\max}$ due to ambient temperature. They compared $\dot{V}O_{2\max}$ values in 46° C following 20 min of low-intensity exercise in the same temperature, and 23° C without any preheating (different group of subjects). A 25% (985 ml/min) reduction was observed following preheating, whereas there was only a 7% (225 l/min) reduction without preheating in a different group of subjects. The preheating may have allowed sufficient time for vasodilation and blood accumulation in the skin prior to the $\dot{V}O_{2\max}$ test or, alternatively, allowed core temperature to rise. Core temperature was almost 2° C higher at the start of the $\dot{V}O_{2\max}$ following preheating. Similarly, Saltin et al. (208) found 7% (350 ml/min) reduction in $\dot{V}O_{2\max}$ in 40° C following 15 min of exercise at 50% of $\dot{V}O_{2\max}$ that elevated esophageal temperature 0.7° C. They used a constant-rate maximal protocol to elicit $\dot{V}O_{2\max}$ and without the preheating, no reduction was observed in $\dot{V}O_{2\max}$ probably due to vasoconstriction of the skin because of the high work load (190).

The elevation in core temperature observed following preheating (174,208) and warm-up (197) suggests that attainment of a critical core temperature during a $\dot{V}O_{2\max}$ test causing a decrement in $\dot{V}O_{2\max}$ might depend upon the core temperature at the start of the test. Nevertheless, Bergh and Ekblom's (15) manipulations of initial core temperature (34.9-38.4° C) through low-to-moderate-intensity water exercise resulted in lower $\dot{V}O_{2\text{peak}}$ values only at low initial core temperatures. $\dot{V}O_{2\text{peak}}$ was significantly correlated with esophageal temperature, suggesting that high body temperatures are needed to elicit high $\dot{V}O_2$ values. However, these $\dot{V}O_{2\text{peak}}$ tests were conducted in a comfortable environment (20-22° C) and brief supramaximal work bouts were utilized, both of which would limit rise in core temperature, and the development of high cardiovascular strain.

Nybo et al. (167) recently investigated this issue of the effects of initial core temperature on possible attainment of a critical core temperature during a $\dot{V}O_{2\max}$ test.

They elicited $\dot{V}O_{2\max}$ by a maximal constant-load cycle exercise and found 16% (750 ml/min) reduction in $\dot{V}O_{2\max}$ after elevating core temperature 1° C without apparent attainment of the critical core temperature. The reduction in $\dot{V}O_{2\max}$ was attributed to a reduction in stroke volume due to the heat stress, suggesting that $\dot{V}O_{2\max}$ is not limited by hyperthermia. However, their subjects did both $\dot{V}O_{2\max}$ tests on the same day separated by an hour, and the control test always preceded the hot condition. Reversing the order of the tests (dehydration portion of their study) resulted in only 11% difference in $\dot{V}O_{2\max}$ between the hot and neutral conditions, indicating that the order of the tests affected $\dot{V}O_{2\max}$. Their subjects also cycled 2 h prior to the maximal test at 50% of control $\dot{V}O_{2\max}$, which is closer to 60% of the $\dot{V}O_{2\max}$ actually found in the heat. The higher metabolic strain in the heat could account for some of the reduction in $\dot{V}O_{2\max}$. Furthermore, the hyperthermic condition in their study was created by using water perfused jackets (44° C), but most of the heat production during cycling occurs in the legs, and about 40% of it is dissipated directly to the environment instead of being transferred to the core (85). Such a direct heat dissipation would retard the rise of core temperature, which combined with the short protocol (<3 min) used, could explain lack of critical core temperature attainment.

Sawka et al. (215) used passive preheating in hot environment to investigate the effects of heat on $\dot{V}O_{2\max}$, and thereby eliminated the effects of any possible fatigue due to active preheating (167,174,197,208). They found about 7% (250 ml/min) reduction in $\dot{V}O_{2\max}$ in the heat, both prior to and after acclimatization. The same power output was reached in hot (49° C) and comfortable (25° C) conditions (215), so the reduction was not due to lower work rate in the heat. The same rate of work suggests that more energy was derived aerobically in the heat, but neither lactate nor any other measure of anaerobic metabolism was reported. After acclimatization, fit subjects showed a greater percentage reduction than unfit subjects as was observed by Rowell et al. (197).

The studies finding reduction in $\dot{V}O_{2\max}$ in the heat have used variety of ambient temperatures ranging from 32° C to 49° C, but only one study has systematically investigated the effects of ambient temperature on $\dot{V}O_{2\max}$ (204). They measured $\dot{V}O_{2\max}$ at temperatures ranging from 5° C to 50° C in 15° C increments. $\dot{V}O_{2\max}$ values differed only by 6% (165 ml/min), with the highest value at 35° C and the lowest at 50° C. This is the only fairly well controlled study to find a significant decrease in $\dot{V}O_{2\max}$ as result of hot environmental temperature without preheating.

Some of the studies cited above have been conducted in hot dry (RH < 40%) environments (193,200,215), whereas others have used hot humid (RH ≥ 50%) environment (174,204,259). Saltin et al. (208) compared hot humid and dry environments using a maximal protocol without preheating, and found $\dot{V}O_{2\max}$ to be identical in these environments. On the other hand, Sen Gupta et al. (219) found, using a graded maximal test, that hot and very hot humid environments (55% RH) resulted in greater reductions in $\dot{V}O_{2\max}$ (10% at 36.5° C and 14% at 41° C) than the hot and very hot dry (20-23%RH) environments (5% at 44° C and 11% at 49° C), despite higher temperatures in the dry heat. The effective temperature was the same between the hot and humid environments (31° C in hot and 34° C in very hot). The greater reduction in humid heat could be due to thermal discomfort associated with lack of heat dissipation through evaporation (3). They (59) later replicated the hot and very hot humid heat conditions (60% RH), but found slightly lower reductions in $\dot{V}O_{2\max}$ (7 and 11%, respectively). Unfortunately, core temperature was not measured in these studies (59,219), and the authors do not describe their protocol clearly (unclear whether preheating was employed). Furthermore, the values for $\dot{V}O_{2\max}$ were suspiciously low for men or ~2.6 (219) and ~2.1 l/min (59).

Several studies have been conducted in water to assess the effect of different water temperatures on $\dot{V}O_{2\max}$. Studies using water temperatures from 18° C to 34° C during swimming have not found decreased $\dot{V}O_{2\max}$ with increasing water temperature

(43,105). The short work time (3 min) might account for the lack of decrease in $\dot{V}O_{2\max}$, since the exposure time was probably not long enough for blood pooling in the skin, as a result of vasodilation from heat, or increase in core temperature (43). However, 20 min of swimming prior to the $\dot{V}O_{2\max}$ test did not alter the results (105).

Water temperature of 34° C is probably not enough heat stress to cause a reduction in $\dot{V}O_{2\max}$. Pirnay et al. (175) found that $\dot{V}O_{2\max}$ measured following 15 min of preliminary exercise at the same temperatures, increased with increasing water temperature from 20° C to 35° C, but decreased at 40° C. There was a 10% increase in $\dot{V}O_{2\max}$ for every 1° C in muscle temperature for water temperatures up to 35° C, but at higher water temperatures, $\dot{V}O_{2\max}$ decreased despite increased in muscle temperature (175). The decrease in $\dot{V}O_{2\max}$ observed at the highest temperature could be due to circulatory strain or to attainment of critical core temperature, because of the uncompensable heat stress.

A few studies have investigated the effect of cold ambient temperature on $\dot{V}O_{2\max}$. These studies have used temperatures from -20° C to 5° C compared to a thermoneutral environment. Some studies did not find any effect of cold on $\dot{V}O_{2\max}$ (171,205), whereas others found $\dot{V}O_{2\max}$ to be decreased in the cold (179,186). Precooling in cold air had no effect on $\dot{V}O_{2\max}$ measured under thermoneutral conditions (217). In contrast, a 2° C drop in rectal temperature through swimming in cold water reduced $\dot{V}O_{2\max}$ in thermoneutral environment, but fatigue from the 30 min of swimming prior to the $\dot{V}O_{2\max}$ test might have accounted for some of the reduction in $\dot{V}O_{2\max}$ (53).

It can be concluded from the studies discussed in this section that $\dot{V}O_{2\max}$ is reduced in a hot environment following preheating. Without preheating, $\dot{V}O_{2\max}$ is not reduced or only slightly reduced. Preheating most likely reduces $\dot{V}O_{2\max}$ in one of the following ways: 1) by increasing skin blood flow and blood pooling in the skin resulting in lower stroke volume and cardiac output or 2) by increasing core

temperature resulting in less room for heat storage and attainment of a critical core temperature associated with maximal circulatory strain, causing termination of exercise prior to attainment of $\dot{V}O_{2\max}$ of thermoneutral environment.

Effects of Environmental Temperature on Exercise Performance

The effects of environmental temperature on endurance performance has been extensively investigated, although in many of these investigations the effect of temperature has been confounded by the detrimental effect of dehydration or the effect of fuel supplementation. However, a few studies have compared performance in thermoneutral and hot environments. Generally, endurance performance decreases with increasing ambient temperature (1,23,45,71,77,130,140,161,197,204,208,225). Endurance time also has been reported to be lower in a cold compared to a neutral environment (171,179).

MacDougal et al. (130) conducted the first study that suggested that endurance performance in the heat could be limited by attainment of a high (critical) core temperature. They manipulated heat storage by a water-perfused vest during exercise to exhaustion at 70% of $\dot{V}O_{2\max}$ at 23°C. The control condition was conducted with no perfusion, whereas hyperthermic and hypothermic conditions were created by perfusing the vest with water temperature equal to that of the rectum or 18°C water, respectively. In all conditions, exhaustion occurred at similar esophageal temperature (~39.5°C) despite large differences in performance time. Reduced stroke volume was observed in the control and hyperthermic conditions, and pooling of blood in cutaneous vessels, resulting in reduced ventricular filling pressure, was suggested as the reason for the shorter endurance time. However, in the hypothermic condition, a drop in stroke volume was not observed, suggesting that the cause of fatigue was not inadequate cardiac output, but the attainment of a critical core temperature.

Then, after series of studies in which leg blood flow was directly measured during exercise, Nielsen and colleagues proposed that the high core temperature

attained during heat stress and exercise might in itself cause the fatigue and inability to continue to exercise (84,85,87,159-161,165,166). Support for that claim comes from the many studies that have found exhaustion to occur at similar core temperature regardless of ambient/water or skin temperatures (43,46,87,130,225), initial core temperature (76,87,254), rise in core temperature (76,87), exercise duration (37,38,76, 87,93,141,150,161,166,167,214,225,243,247,263), exercise intensity (38,141,150,214), rest periods during exercise (214), climate (150), acclimatization stage (37,161,166), state of hydration (37,38,128), fluid ingestion (38,141), stage in menstrual cycle (247), melatonin intake (142), or carbohydrate availability (71).

The studies measuring leg blood flow have found it to not be reduced during mild-to-exhaustive exercise, and suggest that exercise is terminated because of attainment of a critical core temperature. Savard et al. (213) measured leg blood flow during one-legged (25% of $\dot{V}O_{2\max}$) and two-legged (50-60% of $\dot{V}O_{2\max}$) exercise at skin temperatures of 31, 35, and 38°C, respectively. They found no differences between conditions in $\dot{V}O_2$, cardiac output, stroke volume, $A-\dot{V}O_{2\text{diff}}$, mean arterial pressure, lactate, leg blood flow, leg oxygen uptake, or leg vascular conductance. Skin blood flow was higher at higher skin temperatures and was met by increased cardiac output (2 l/min) and the normal reduction of splanchnic and renal blood flow of 0.6-0.8 l/min (190). Interestingly, the maximal level of skin blood flow at the hottest skin temperature was the same in one-and two-legged exercise despite different esophageal temperatures (37.9 vs 39.4°C, respectively), suggesting that vasoconstriction took place in two-legged exercise. This vasoconstriction, which is necessary for maintenance of blood pressure (20,156,190), was probably, in part, responsible for the rise in esophageal temperature, but the cardiac output was still able to meet the muscle blood flow demands of the exercise. On the other hand, during one-legged exercise the central circulation was able to meet blood flow demands of both skin and muscle, because of much lower metabolic rate, retarding the rise in esophageal temperature.

A similar plateau in skin blood flow once esophageal temperature reached 38° C was reported by Gonzalez-Alonso et al. (87). They used water immersion to preheat/precool subjects to esophageal temperatures of 35.9, 37.4, and 38.2° C, followed by exercise to exhaustion at 60% of $\dot{V}O_{2max}$ in 40° C. Exhaustion occurred at similar esophageal (40.1-40.2° C) and muscle temperatures despite endurance time being the longest in the precooling condition. They also controlled the rate of heat storage (0.05 vs 0.1° C/min) by perfusing a jacket in contact with the skin on the trunk and arms. Similarly, exhaustion occurred at similar esophageal (40.1-40.3° C) and muscle temperatures despite a markedly different skin temperature (35.6 vs 38.4° C, respectively) and endurance time. These results suggest that hyperthermia caused fatigue in the heat, and are in accordance with the suggestion that once cardiac output cannot meet both metabolic and thermoregulatory demands, skin blood flow is reduced or the rate of rise in skin blood flow decreases (20,156,157,246,256,259), resulting in rise in core temperature.

Not only is leg blood flow adequate at exhaustion during submaximal exercise in the heat, but altered metabolism or substrate depletion also appear to not cause fatigue. During exhaustive exercise, Nielsen et al. (165) found no differences between hot and cold environment in leg blood flow, oxygen uptake, $A-\dot{V}O_{2diff}$; lactate; or blood glucose and plasma free-fatty acid concentrations. Glycogen was not completely depleted and the rate of glycogen utilization was actually lower in the heat. The authors concluded that neither flow limitations to the working muscles nor altered metabolism or substrate depletion were the causes of fatigue. Instead, the high core temperature (39.3° C) was the most likely cause of fatigue.

During heat acclimatization in hot dry (42° C 15% RH) and hot humid (35° C 87% RH) conditions, very similar results were reported (161,166). Acclimatization did not affect VO_2 ; leg blood flow; oxygen consumption, and $A-\dot{V}O_{2diff}$; or blood glucose, free-fatty acid, lactate, and hormonal concentrations. However, a plateau or slight drop

in forearm blood flow was observed as the exercise progressed (161,166). This plateau may reflect a limit for the amount of blood flow available for heat transfer to the skin despite the increasing hyperthermia. Exhaustion prior to, during, and after acclimatization was not due to limitations in muscle blood flow, or substrate utilization, but rather to the high core temperature (39.8°C) attained (161,166). Similarly, no difference in forearm blood flow during light-to-moderate exercise was found between 25°C and $65\text{--}70^{\circ}\text{C}$ environments (235), and muscle blood flow measured by microspheres was not reduced during exhaustive exercise in the heat in animals (5,93).

Carbohydrate ingestion did not improve performance time compared to a placebo in a mild heat stress (33°C , RH 20-30%) during cycling at 70% of $\dot{V}\text{O}_{2\text{max}}$ until exhaustion (71). Hyperthermia was hypothesized as the cause of fatigue, since during every trial, exhaustion occurred at rectal temperature above 39°C . In contrast, carbohydrate ingestion improved performance in 5°C (RH ~50%) environment, and rectal temperature was below 39°C at exhaustion.

Termination of exercise at a critical core temperature also has been reported in studies of animals. Fuller et al. (76) found that rats exercising to exhaustion in 33°C , 38°C , and 38°C following passive preheating elevating core temperature, voluntarily terminated exercise at the same hypothalamic ($40.1\text{--}40.2^{\circ}\text{C}$) and abdominal temperatures in all conditions. The mean run times to exhaustion in the different conditions were 29, 22, and 14 min, respectively. Similarly, rapidly preheated rats by microwaving to hypothalamic temperatures of 41.5 , 42.5 , and 43.5°C , stopped running after 30, 19, and 7 min, respectively, whereas they ran 36-38 min without microwaving (254). Termination of exercise occurred at similar hypothalamic ($41.9\text{--}42.2^{\circ}\text{C}$) and rectal temperatures ($42.2\text{--}42.5^{\circ}\text{C}$), and rats that had higher initial hypothalamic temperature than 42.5°C did not even attempt to run. Shorter performance with increasing initial temperature in the heat has been reported by others as well (45,162,167). Furthermore, studies of other animal species have reported exhaustion

occurring at a similar core or brain temperatures (5,31,93,243,263), and endurance time inversely related to the heat storage, i.e. the time it took to reach those temperatures (93,243,263).

Despite many studies observing exhaustion occurring at a constant (critical) core temperature between 39.3^o C and 40.5^o C (31,76,84,85,87,130,161,165,166,213, 243), findings of fatigue at different core temperatures in hot and neutral environments have been reported (77,234), but whether the heat stress in these studies was uncompensable is questionable. Still, under uncompensable heat stress, exhaustion has been reported to occur at a lower body temperature than 39.3^o C (37,38,46,128,141-143,150,214,216,225,247). The core temperature at exhaustion has been found to be lower subjects who were untrained (37,75), dehydrated (216), exercised in humid environment (143), exercised in the morning (142), and exercised in full chemical and/or protective clothing (150). Training has been found to increase thermal tolerance. Fruth and Gisolfi (75) coerced rats to run to exhaustion, and found that trained rats could run longer in the heat, sustain greater thermal loads, and were less susceptible to work-induced thermal fatality than untrained rats. The better heat tolerance by trained rats could be due to induction of heat shock proteins that increase cell thermotolerance (152,229).

Some of the human studies reporting exhaustion at lower core temperature than 39.3^o C, (37,38,142,143,150,216,225,247) have used very prolonged (2-3 h), low-intensity exercise protocols, which could potentially lower work capacity and cause exhaustion due to cardiovascular strain prior to attainment of core temperatures in populations susceptible to such strain (untrained, dehydrated, and fully clothed subjects). The protective clothing and the excessive dehydration conditions used in these studies could also increase the thermal load (core temperature above certain level times work time) on the subjects which can cause fatigue or interfere with exercise without attainment of a critical core temperature (30,110). However, it is also possible

that the protective clothing and the excessive dehydration in these conditions interact with the uncompensable heat stress such that the critical core temperature for termination of exercise is lowered (92).

It is unclear whether it is muscle, core, or brain temperature that causes fatigue. The animal studies that have measured hypothalamic temperature, have found it to be slightly higher (0.3°C) than abdominal temperature (76), similar to rectal temperature (93,254) or lower than rectal temperature (31,242) at exhaustion. This could be due to species differences because some species (i.e. goats, gazelles) can selectively cool the brain while their body temperature keeps increasing (30,31,242). This selective brain cooling suggests that the brain or the central nervous system is especially susceptible to damage from high temperatures (27,144,251). Furthermore, the subjective reports of dizziness, confusion and being hot in the head, make high brain temperature appealing as the limiting factor for exercise. On the other hand, skeletal muscle is the locus of heat production, so high skeletal muscle temperatures could also be the limiting factor for exercise in the heat. Skeletal muscle temperature at exhaustion has been reported to be slightly higher (0.6°C) than esophageal core temperature (87). Finally, a critical level of skin temperature does not seem to cause fatigue as was eloquently demonstrated by Gonzales Alonso et al. (87) where subjects exhausted at same core and muscle temperatures but at markedly different skin temperatures; a finding supported by others (130).

The mechanisms for the reduced $\dot{V}\text{O}_{2\text{max}}$ and performance as a result of reaching a critical core temperature have not been identified. The high core temperature is thought to affect the central nervous system and mental functioning (25,161,165). Muscular and circulatory discomforts in addition with thermal discomfort from heat storage and lack of heat dissipation may counteract motivation and thus gradually reduce the drive to exercise with increasing body temperature (25). Heat stressed participants are often dizzy and confused (165,166), which demonstrates the central

nervous system susceptibility to high temperatures. Some studies (165,166) have reported that subjects, in addition to being dizzy, discontinued to exercise due to inability to move their legs or to a feeling of being extremely hot in the head despite maintenance in muscle circulation and adequate substrate availability and turnover. Therefore, it may be that extremely high core temperatures reduce the functions of the motor centers and the ability to recruit motor units through an effect on motivation for motor performance (165). Nielsen et al. (161) found, however, no differences in the ability to recruit motor units at exhaustion.

The adaptive value of a mechanism that causes termination of exercise at a critical core temperature could be to protect the body to from reaching temperatures that cause heat injury and jeopardize cell functions (30,76,87,112,254). Rats that run to voluntary exhaustion at 33° C, 38° C, and 38° C following preheating all terminated exercise at the same brain and abdominal temperatures despite differences in endurance time (longest in the coldest condition) (76). None of the animals displayed signs of heat illness following exercise or died, which is contrast to studies that have coerced rats or sheep to run to collapse (unable to right themselves when placed on their backs) through electrical shock (75,93,109). Such protocols can result in heat stroke and death (75,93,109,110). Electrical shock has been used to motivate untrained rats to run until exhaustion (still able to walk at reduced speeds) without any mortality, and the rats stopped exercise at the same hypothalamic and rectal temperatures despite differences in initial temperature or endurance time (254). The difference in mortality rates between the studies using electrical shock lies in the thermal load (rectal temperature in ° C multiplied by minutes with rectal temperature above 40° C). The rats in the study of Walters et al. (254) sustained three times lower thermal load than the untrained rats that died in the study of Fruth and Gisolfi (75), whereas the survivors in that study sustained a thermal load similar to that in the study of Walters et al. (254). The thermal load is an important determinant of heat casualties (75,109,110,252), and despite a

critical core temperature beyond which exercise cannot continue, it seems relatively insensitive to thermal load and cannot protect against damage or lethality in all circumstances (254), because the effect of heat is determined by both its intensity and duration (29,224). The thermal load could explain reports of heat strokes occurring during prolonged endurance events or exercise in the heat (34,94), because persons with high critical core temperature (i.e. 40.5°C) could potentially exercise for prolonged time below that temperature ($40\text{-}40.2^{\circ}\text{C}$) but sustain a considerable thermal load. Basically, a lethal thermal load can occur during submaximal exercise in the heat before the critical core temperature is attained (110).

The same phenomenon is observed in humans as reviewed above (37,38,43,46, 71,87,128,130,141,142,150,161,166,214,225,247). Most humans fatigue (terminate exercise) at a core temperature of approximately 40°C (84,85,87,130,161,166). Volitional exhaustion in these and other studies occurred at the same critical core temperature, regardless of external conditions. None of the studies report any adverse effect of the high core temperature supporting the notion that exercise is terminated through physiological mechanisms preventing the subjects from injuring themselves (37,38,43,46,71,87,128,130,141,142,150,161,166,214,225,247). The level of core temperature that becomes limiting for performance may depend on factors such as dehydration (92,216), fitness (37,75,87,92), clothing (37,38,46,128,141-143,150,214, 216,225,247), circadian rhythm (142) nutrition, and motivation (92,161). It has been found to be lower in untrained (37,75), dehydrated (216), and fully clothed (150) subjects exercising in the morning (142).

The effects of environmental temperature on other types of performance also has been investigated. Isometric force/torque is not affected by temperature (14,16,52,238) but isometric, like dynamic, endurance is shorter at high and low temperatures (40,61,173). Moderate heat ($30\text{-}33^{\circ}\text{C}$) or increasing esophageal or muscle temperatures, generally have a positive effect on short maximal performance

(7,11,14,16,32,39,40,65,130,212), although a few studies report no difference between thermoneutral and hot environments (52,60,102,238), and two studies report increases in short maximal performance with decreasing muscle temperature (7,182). Repeated short maximal performance to exhaustion is, like endurance performance, negatively affected by hot environment (90,135).

Some attention has been given to the effect of precooling on performance. An early series of studies demonstrated that repeated short-duration performances were enhanced when cold water or ice was applied to the subjects during the recovery between exercise bouts compared to a control condition (95,187,227). Then, Brück and colleagues (24,25,100,168,217) published a series of studies in which subjects cycled at 18° C after precooling at 0-5° C until shivering or after resting in 18° C. They (217) found no difference during short graded maximal exercise in $\dot{V}O_{2\text{peak}}$ or maximal work, probably due to lack of heat stress. However, sweating started at a higher work rate after precooling which corresponded with lower mean body temperature. Delayed sweating, but starting at a lower body temperature, also has been reported by others comparing cold and thermoneutral environments (127). When the subjects performed as much work as possible in 60 min (100) or cycled for 60 min at a heart rate of 120 beats/min (24), the work rate and $\dot{V}O_2$ were higher after precooling. Mean body temperature and heart rate were lower during the first 10-20 min and sweat rate was lower for the first 30 min (100). Similar results were reported for endurance time to exhaustion at 80% of $\dot{V}O_{2\text{max}}$ (168); sweat rate, forearm and skin blood flows, and mean body temperature were found to be less with precooling.

Very similar results were reported by Lee and Haymes (129) for subjects running in 24° C at 82% of $\dot{V}O_{2\text{max}}$ following a rest for 30 min at 5° C or 24° C. Run duration increased with precooling, and rectal, skin, and mean body temperatures were lower after precooling, resulting in greater rectal-skin temperature gradient during most of the run. That lead to greater heat storage after precooling, and sweat rate and heart

rate were lower. Similarly, Booth et al. (19) found that distance run in 30 min in 31.6° C was greater after precooling. Skin and rectal temperatures were lower throughout the test, heat storage was greater, and heart rate was lower for the first 10 min. The sweat rate was not lower following precooling, probably due to more work done (greater distance covered in the same time), as was reflected in higher blood lactate concentration. The increased heat storage reported in these studies permitted greater metabolic heat production, and delayed decline of cardiovascular and thermoregulatory functions prior to fatigue.

Only a short period of precooling is needed for performance enhancement. Myler et al. (154) found that, after warm-up in 30° C, only 5 min application of ice packed in damp towels to subjects' skin prior to 6 min all out rowing performance, increased performance equivalent to a margin of 4-5 s in a 2000 m race. Such a short cooling reduced tympanic temperature at the start, potentially increasing heat storage. Skin temperature also was lower before and after the test, resulting in greater skin to core temperature gradient and supposedly greater heat dissipation. In addition, heart rate was lower and pH higher during the test, which might reflect increased muscle blood flow (greater removal of lactate).

The results of these studies suggest that hot or cold environments negatively affects dynamic exercise endurance performance. Precooling generally has positive effects on performance in a thermoneutral environment by lowering initial body temperature and heart rate, allowing greater room for heat storage, less pooling of blood in the skin, and lowering the sweating threshold in relation to core temperature. The effects of precooling on exercise in an uncompensably hot environment has not been studied.

Limitations of Maximal Oxygen Uptake and Performance in a Hot Environment

A hot environment may increase anaerobic metabolism and glycogen breakdown (70,73,126), possibly due to elevated epinephrine (170), which could

quicken fatigue in the heat because of metabolite accumulation or glycogen depletion. Increased muscle temperature may also induce structural and functional alteration in various proteins involved in skeletal muscle contractions and energy metabolism (69,96). Mitochondrial phosphorylative efficiency is reduced at muscle temperatures above 40° C (21) and exercise in the heat increases oxidative stress (146), which may detrimentally affect skeletal muscle metabolic function (69,96). Despite these potential effects of heat on skeletal muscle metabolism, circulatory strain or hyperthermia associated with circulatory strain are the two main hypotheses offered as an explanation for the reduction in endurance performance, and possible reduction in $\dot{V}O_{2\max}$, observed in hot environments. The circulatory strain hypothesis focuses on reductions in $\dot{V}O_{2\max}$ as a result of reduced cardiac output, due displacement of blood volume to the skin for heat dissipation. The core temperature hypothesis emphasizes the rapid increase in core temperature due to the heat stress, and the attainment of a critical core temperature associated with maximal circulatory strain, resulting in termination of exercise at a work rate below that which elicits $\dot{V}O_{2\max}$ in a thermoneutral environment.

The circulatory strain hypothesis. During severe exercise in a hot environment the need for thermoregulation imposes a stress on the cardiovascular system in addition to the metabolic demand of the muscles. In essence, skin and active muscle compete for the available blood flow and their needs can exceed the pumping capacity of the heart. Eventually, either work must stop due to inadequate muscle blood flow and increased anaerobic metabolism, or hyperthermia occurs (155,190). In addition, cutaneous vasodilation results in pooling of blood in the cutaneous veins and lowers stroke volume. In normal or cool environments, cutaneous veins refill slowly after each muscle contraction and average venous pressure and volume are reduced (97). During exercise in the heat, this muscle pump is less effective because the cutaneous veins refill so rapidly that their average pressure and volume remain elevated and cause reduced filling pressure (190). Redistribution of blood from active skeletal muscle to

the skin could also be responsible for the increase in skin blood flow, but that has not been shown to occur in humans (190) unless sympathetic activity is increased above what is normal during exercise (240). The pooling of blood in the skin could be responsible for the reduced $\dot{V}O_{2\max}$ and performance reported in a hot environment, since lower central blood volume would lead to lowered cardiac filling pressure, resulting in lowered stroke volume and eventually lowered cardiac output. The cutaneous vasodilation reduces stroke volume so maximal heart rate and $A\text{-}\dot{V}O_{2\text{diff}}$ are reached at a lower $\dot{V}O_2$, resulting in lower muscle blood flow because maximal cardiac output is not reached, with the end result being lower $\dot{V}O_{2\max}$. Further evidence for the blood volume displacement on central circulatory changes was obtained during exercise in air at 45° C and in water at 35° C at the same oxygen uptake and skin temperature, but cardiac output and stroke volume were higher in the water exercise because of less blood pooling in the skin (164).

Heart rate increases fairly linearly with increased absolute and relative exercise intensity in thermoneutral environment (10,190). During heat stress, the heart rate is higher at a given absolute intensity (22,59,73,156,172,193,197,200,219,234,259). Therefore, studies (193,200,259) finding no reduction in $\dot{V}O_{2\max}$ in the heat despite increased heart rate during submaximal exercise at the same absolute intensity, suggest that there is dissociation in the relationship between heart rate and relative exercise intensity. This apparent dissociation and elevated heart rate in the heat has been explained by increased sympathetic activity (123,196) increased rate of sinoatrial node firing in the heat (115), and feedback from increased core and skin temperature to the heart (42,248).

Furthermore, increased skin blood flow for thermoregulation could play a role for the elevated heart rate in the heat. Skin blood flow during submaximal exercise generally starts at around core temperature of 37.2° C (121,246,256). This increase in skin blood flow in the heat is partially met by reduced splanchnic (189) and renal (180)

blood flows and partially by elevated cardiac output during mild exercise (156,163,199). The cardiac output can be increased 2-3 l/min due to tachycardia during prolonged mild exercise in the heat (156,163,199) to maintain both the elevated metabolic rate during mild exercise and the increased skin blood flow for thermoregulation.

Although cardiac output is elevated during mild exercise in the heat, during heavy exercise in the heat, it is not increased despite heart rate reaching maximal values (86,200), because of a marked decrease in stroke volume (17-21%) at the highest workloads that could not be compensated for by increase in heart rate (200). These results support the findings of earlier work (6,58) but are in contrast with the results found by Williams et al. (259), who found no reduction in cardiac output in the heat despite the fact that maximal heart rate was reached prior to $\dot{V}O_{2max}$; apparently stroke volume was restored at the highest workload. In thermoneutral environment, or during low-intensity exercise in heat, up to 3 l/min of blood (188) can be redistributed away from splanchnic and renal regions, non-exercising muscle, and adipose tissue (35,88,89, 101,104,122,180,189,192-194,198,233,258) meeting the blood flow of both skin and muscle. However, during hard exercise in severe heat stress, the cardiovascular system is forced to its functional limits at submaximal levels of oxygen uptake (200). Nevertheless, once the flow requirements of skin and muscle exceed the cardiac pumping capacity, blood pressure is still maintained possibly through vasoconstriction in both active muscle and skin (20,134). The vasoconstrictor stimuli affects skin blood flow which has been reported to be reduced at high core temperature (20,156,157,246,256) and during exhaustive exercise (12,259). Furthermore, the plateauing of cardiac output has been related to the point at which vasoconstrictor stimuli for blood pressure begin to overwhelm the vasodilator influence of increasing core temperature (20,156,201). The vasoconstrictor stimuli overrides the vasodilation when stroke volume falls to about 100 ml/beat (156) and prevents further fall in stroke

volume, stabilizing the cardiac output and the central circulation at the expense of core temperature, which increases.

During brief periods of maximal exercise in the heat, $\dot{V}O_{2\max}$ may not be reduced, most likely because of skin vasoconstriction (190), permitting the normal 85% of cardiac output to be directed towards the working muscle (4,54,191,253). However, at some core temperature or a combination of core and skin temperatures, vasoconstriction in the skin is overwhelmed and $\dot{V}O_{2\max}$ falls as a consequence of cutaneous vasodilation (190).

The core temperature hypothesis: Although attainment of critical core temperature alone can cause termination of submaximal endurance exercise as reviewed above, high core temperature during maximal exercise is usually associated with maximal heart rate indicating a maximal level of circulatory strain (167,174,208). Therefore, it is plausible that a combination of circulatory strain and high core temperature contribute to a reduction in $\dot{V}O_{2\max}$ in the heat. Under severe heat stress, blood volume displacement to the skin, reducing stroke volume and cardiac output, may occur at the same time as a critical core temperature is attained. Stroke volume could also be lowered due to increased vascular resistance caused by an increase in sympathetic activity in proportion to increased core temperature (123). Furthermore, attainment of a critical core temperature by itself may prevent attainment of a maximal stroke volume, which has been found to increase progressively up to $\dot{V}O_{2\max}$ in trained individuals (80). Regardless of the mechanism for lowered stroke volume associated with high core temperature, the lower cardiac output and hyperthermia would decrease $\dot{V}O_{2\max}$, despite no reduction in A- $\dot{V}O_{2\text{diff}}$ (200). Because $\dot{V}O_{2\max}$ in the heat would be reached at lower work rate than in thermoneutral environment, muscle blood flow compared to the same work rate in a thermoneutral environment, would not be reduced, regardless of lower cardiac output. However, higher work rates could not be sustained,

due to the simultaneous attainment of critical core temperature and maximal cardiac output.

Reduction in $\dot{V}O_{2\max}$ due to lower central blood volume (circulatory strain) would not occur with a maximal or supramaximal protocol without preheating. Skin vasoconstriction (190) due to the high workload would permit the normal 85% of cardiac output to be directed towards the working muscle (4,54,191,253) as was demonstrated by Saltin et al (208). Similarly, during regular exercise at 60% of $\dot{V}O_{2\max}$ in the heat, the active muscles do not need the blood that has pooled in the skin, and therefore, no reduction in leg blood flow is observed (161,166). These studies report that $\dot{V}O_2$ (87,161,166) and heart rate (161,166) at exhaustion are well below maximal, suggesting that circulatory strain is not the cause of fatigue. In these studies, a plateau was reached in forearm blood flow, which would maintain leg blood flow and allow core temperature to rise to critical levels. However, during very prolonged exercise in the heat, circulatory strain or combination of circulatory strain and high core temperature has been shown to be the cause of fatigue, especially if subjects were untrained (37), dehydrated (216), or wore protective clothing that interfered with heat dissipation (38,46,128,141-143,150,214,225,247).

In conclusion, the most plausible reason for reduced $\dot{V}O_{2\max}$ and performance during exercise in the heat following preheating is attainment of a critical core temperature. The preheating elevates core temperature and reduces the ability for heat storage. At certain point, cardiac output cannot meet both the thermoregulatory and metabolic demands, which leads to a plateau or a decrease in skin blood flow, further escalating the rise in core temperature. Once the core temperature reaches a critical level, subjects terminate exercise. Depending on the intensity of exercise, that critical level of core temperature may or may not be associated with maximal level of circulatory strain. Under extremely hot temperatures, it is hypothesized that attainment

of this critical core temperature will occur prior to attainment of work rates sufficient to elicit $\dot{V}O_{2\max}$ in thermoneutral environments. Hence, $\dot{V}O_{2\max}$ will be reduced.

CHAPTER 3
MAXIMAL OXYGEN UPTAKE IN THE HEAT:
ROLE OF CORE TEMPERATURE¹

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Abstract

Previous studies have suggested that attainment of a critical core temperature ($\sim 40^{\circ}\text{C}$) can limit performance in prolonged, exhaustive exercise in the heat. To test the hypothesis that large ($> 10\%$) reductions in maximal oxygen uptake ($\dot{V}\text{O}_{2\text{max}}$) in the heat are associated with attainment of a critical core temperature, $\dot{V}\text{O}_{2\text{max}}$ was measured in 11 male and 11 female runners under 7 conditions designed to manipulate the esophageal temperature (T_{ESO}) at which $\dot{V}\text{O}_{2\text{max}}$ was reached by varying environmental temperature (at 50% RH) and preheating. The conditions were: 1) 25°C , no preheating (control); 2-4 and 6) 25, 35, 40, and 45°C , with preheating by a 20-min walk at $\sim 33\%$ of control $\dot{V}\text{O}_{2\text{max}}$; 5) 45°C , no preheating; and 7) 45°C , with passive preheating raising T_{ESO} to the same degree as at the end of the 20-min walk in Condition 6. Compared to $\dot{V}\text{O}_{2\text{max}}$ (l/min) in the control condition (4.52 ± 0.46 in men, 3.01 ± 0.45 in women), $\dot{V}\text{O}_{2\text{max}}$ in the heat was reduced by 3-9% under Conditions 2-5 when core temperature at $\dot{V}\text{O}_{2\text{max}}$ was below the apparent critical core temperature ($< 39.2^{\circ}\text{C}$ in men, $< 39.1^{\circ}\text{C}$ in women), but was reduced 17-19% under Conditions 6-7 when a critical core temperature appeared to have been attained ($39.6\text{-}39.7^{\circ}\text{C}$ in men, $39.3\text{-}39.4^{\circ}\text{C}$ in women). Percentage reductions in time to exhaustion were larger (7-36%) than for $\dot{V}\text{O}_{2\text{max}}$ and were strongly related to reductions in $\dot{V}\text{O}_{2\text{max}}$ ($r = 0.82\text{-}0.84$). The effect of heat on $\dot{V}\text{O}_{2\text{max}}$ and work capacity in men and women were almost identical. We conclude that large reductions in $\dot{V}\text{O}_{2\text{max}}$ and work capacity in men and women in severe, uncompensable heat stress are associated with attainment of a critical core temperature. The strong relation between reductions in $\dot{V}\text{O}_{2\text{max}}$ and in work capacity in the heat appears to depend, in part, on the effect of attainment of a critical core temperature on both measures. Men and women do not differ in their body temperature responses, or in the relation of the core temperature, to the reductions in $\dot{V}\text{O}_{2\text{max}}$ and work capacity in the heat.

INDEX WORDS: Critical core temperature, Maximal oxygen uptake, Endurance performance, Heat stress, Gender, Treadmill exercise.

Introduction

A hot compared to a cool environment compromises physical work capacity (28,33,40,42). However, the literature is conflicting as to whether the reduction in performance is related to reduced maximal oxygen uptake ($\dot{V}O_{2\max}$) in the heat. Some studies suggest that high ambient temperature reduces $\dot{V}O_{2\max}$ slightly (3-6%) (25,40,42,52), whereas other studies reported no effect (39,41,55). Pirnay et al. (37) were the first to observe a marked decrease (25%) in $\dot{V}O_{2\max}$ when measured in the heat. Their protocol differed from those employed in the earlier studies in that $\dot{V}O_{2\max}$ was measured following low-intensity exercise that elevated body core temperature (preheating). Small (6-8%) to moderate (16%) decreases in $\dot{V}O_{2\max}$ following preheating were later observed by others (36,43,46).

A common explanation for the reduction in work capacity and the possible reduction in $\dot{V}O_{2\max}$ in the heat is an hypothesized reduction in muscle blood flow due to peripheral displacement of blood to the skin (38). However, data suggest that high core temperature may limit exercise performance in the heat (16,20-22,28,33-35,44) independent of an effect on muscle blood flow (33-35). A thermal limit to performance of prolonged, strenuous exercise is suggested by studies that have found exhaustion to occur at a similar core temperature regardless of ambient or skin temperatures (13,22,28,49), initial core temperature (22), rate of rise in core temperature (22,28), exercise duration (22,33,35), exercise intensity (32,45), climate (32), acclimatization stage (11,33,35), state of hydration (11,26), fluid ingestion (12), stage in menstrual cycle (53), melatonin intake (30), or carbohydrate availability (16). High core temperature also could limit $\dot{V}O_{2\max}$ during high-intensity exercise in the heat if it is linked to a maximum cardiovascular response and causes termination of exercise at a work rate below that needed to elicit $\dot{V}O_{2\max}$ under thermoneutral conditions.

Almost all of the evidence supporting a thermal limit for performance (11-13,16,22,26,28,30,32,33,35,45,49,53), and possibly $\dot{V}O_{2\max}$ (14,36,37,42,43,46,48), has

been obtained in men. Whether the effect of high core temperature on $\dot{V}O_{2\max}$ and performance in women is qualitatively or quantitatively different from that in men is unknown.

The purpose of this study was to determine whether large ($> 10\%$) reductions in $\dot{V}O_{2\max}$ during exercise in the heat are associated with attainment of a critical core temperature in endurance-trained men and women. We hypothesized that: 1) large ($> 10\%$) reductions in $\dot{V}O_{2\max}$ are obtained only under conditions in which a critical core temperature is reached; 2) reduced work capacity is related to reduced $\dot{V}O_{2\max}$ in the heat; and, 3) there is no gender difference in the effect of core temperature on $\dot{V}O_{2\max}$ and physical work capacity.

Methods

Subjects: Twenty-eight healthy, endurance-trained male ($n=15$) and female ($n=13$) runners and triathletes were recruited as subjects. Three males and 2 females withdrew from the study after 1-4 tests and incomplete data were obtained from 1 male subject. Therefore, all analyses are reported on 11 men and 11 women. Their physical characteristics are presented in Table 3.1. The men and women had averaged 72.4 ± 42.6 and 59.5 ± 39.5 km/week of running, respectively, for at least 6 wk and were accustomed to exercising in a hot environment. Each subject was tested at the same time of the day to minimize the effects of circadian rhythms on heart rate and body temperature, and a minimum of 2 days intervened between tests of the same subject. All subjects wore shorts and running shoes, and the women wore a sport bra. Participation was voluntary and subjects were paid upon completion of the study. The study was approved by the University's Institutional Review Board and written consent was obtained before testing.

Table 3.1: Physical characteristics of the subjects

Measures	Age yr	Height cm	Mass kg	$\dot{V}O_{2\max}$ l/min	$\dot{V}O_{2\max}$ ml/kg _{BM} /min	$\dot{V}O_{2\max}$ ml/kg _{FFM} /min	Fat %
Males (n = 11)	23.1± 4.7	178.2± 4.4	70.1 ±8.8	4.52 ±0.46	64.7 ±5.3	70.5 ±4.5	8.2 ±3.0
Females (n = 11)	23.8± 3.8	164.8± 5.7	56.0 ±4.9	3.01 ±0.45	53.9 ±7.5	65.8 ±8.0	18.3 ±3.3

$\dot{V}O_{2\max}$ = maximal oxygen uptake, kg_{BM} = kilograms of body mass, kg_{FFM} = kilograms of fat-free mass.

The number of subjects needed to have adequate power to detect a meaningful decrease in $\dot{V}O_{2\max}$ was estimated according to Lipsey (27). A decrease in $\dot{V}O_{2\max}$ of 0.3 l/min was considered meaningful, since 0.3 l/min is about twice the value generally used as a criterion for demonstration of attainment of $\dot{V}O_{2\max}$ in men (increase in $\dot{V}O_2$ less than 0.15 l/min with standard increase in work rate (52)) and is similar in magnitude to the smallest significant decreases in $\dot{V}O_{2\max}$ reported as a result of preheating in the literature (43,46). Assuming a standard deviation (SD) of 0.5 l/min and a correlation of 0.7 for repeated determinations of $\dot{V}O_{2\max}$ under the conditions of this study (37), an adjusted effect size of 1.1 was expected. Based on these assumptions, it was estimated that 11 male subjects were sufficient to obtain a power of 0.8 utilizing a one-tailed α of 0.05. Despite lower absolute $\dot{V}O_{2\max}$ in the women, 11 female subjects also were sufficient to obtain a power of 0.8 (one-tailed $\alpha = 0.05$) to detect a decrease in $\dot{V}O_{2\max}$ of 0.2 l/min assuming the same correlation (0.7) for repeated determinations of $\dot{V}O_{2\max}$ as in men, because of a smaller assumed SD of $\dot{V}O_{2\max}$ (0.33 l/min) in women.

Experimental Design: A repeated-measures experimental design in which subjects served as their own control was used. The dependent variables were $\dot{V}O_{2\max}$, esophageal temperature (T_{ESO}) at the time of fatigue (T_{ESOMax}), and performance time during a continuous, progressive running test to exhaustion. The independent variables were environmental temperature and pretest physical activity (or lack thereof), designed to manipulate core temperature and time to fatigue. Other metabolic, cardiorespiratory,

and perceptual measures were assessed during exercise to help interpret the effects of manipulations of the independent variables on the dependent variables.

Treatments: The study was conducted in an environmental chamber at 50% relative humidity under the following seven conditions in which environmental and pretest core temperature were varied: 1) 25° C with no warm-up (25N); 2) 25° C with a 20-min walking warm-up at ~33% of control $\dot{V}O_{2\max}$ (25W); 3) 35° C with a 20-min walking warm-up at ~33% of control $\dot{V}O_{2\max}$ (35W); 4) 40° C with a 20-min walking warm-up at ~33% of control $\dot{V}O_{2\max}$ (40W); 5) 45° C with no warm-up (45N); 6) 45° C with a 20-min walking warm-up at ~33% of control $\dot{V}O_{2\max}$ (45W); and 7) 45° C with passive heating prior to the test for a period of time sufficient to elevate the core temperature to the same extent as after the 20-min walk in condition 6 (45P). Condition 1 was completed first and served as a control (baseline) condition. Then, Conditions 2-6 were performed in random order, with Condition 7 following. Conditions 2-4 and 6 were designed to elevate the core temperature to different degrees using active preheating prior to the $\dot{V}O_{2\max}$ test, and to alter the rate of rise in core temperature during the test. Condition 5 was designed to investigate whether there was any reduction in $\dot{V}O_{2\max}$ in the heat without preheating. Finally, Condition 7 was designed to determine whether there was any effect of prior exercise on $\dot{V}O_{2\max}$ beyond the effect of heat. Based on pilot data, we expected that a critical (same within a subject) core temperature at the point of fatigue on the graded exercise test would be attained in 40W, 45W, and 45P. We also expected that some individuals might reach their critical core temperature in 35W. Because walking for 20-min at 25° C had no significant effect on any of the measures, reproducibility of the measures was assessed using data from 25N and 25W.

Test Protocol: Subjects reported to the laboratory following a 3-h fast, but well hydrated. They were instructed not to consume alcohol or drugs 48 h prior to testing, not to consume caffeine 12 h prior to testing, and to drink water and other non-

caffeinated beverages liberally. On the morning of the test, subjects filled out a 24-h history questionnaire designed to determine adherence to pretest instructions. Then, skinfold thickness measures were taken for estimation of body fat (in 25N only) and subjects measured their nude body weight. Next, subjects inserted rectal and esophageal thermistors for measurement of core temperature, thermistors for measurement of skin temperature were attached, and a strap containing the electrodes and transmitter for a heart rate monitor was placed around the chest. Finally, the subjects ingested water to compensate for the estimated sweat loss that would occur.

Metabolic, cardiorespiratory, temperature, and perceptual measures were obtained every 5 min prior to the graded running test during the preheating in 25W, 35W, 40W, 45W, and 45P (no metabolic data collected during preheating in 45P). During the passive preheating in Condition 7 (45P), body weight was monitored and fluid was ingested to compensate for sweating. The subjects then completed a graded running test to exhaustion during which oxygen uptake ($\dot{V}O_2$), other metabolic and perceptual variables, heart rate, and performance were measured. Rectal, esophageal, and skin temperatures were continuously monitored throughout the test and recorded every 2 min. Three min following the test, a fingerstick blood sample was obtained and analyzed for lactate. Then, subjects dried off and measured their nude body weight to determine the amount of weight loss (dehydration).

$\dot{V}O_{2max}$ Test Procedures: To elicit $\dot{V}O_{2max}$, subjects ran to exhaustion on a treadmill at a constant speed, with the grade increasing 2% every 2 min. A speed was chosen to exhaust subjects in 6-15 min of exercise. During the test, a Sensormedics Vmax 29 metabolic cart was used to measure the metabolic variables over a sampling period of 30 s. Two consecutive 30-s values were averaged for all metabolic measures. The metabolic cart was calibrated using standard gases with concentrations verified to 0.02%.

Attainment of $\dot{V}O_{2\max}$ in the control condition (25N) was assured by using a modification of the plateauing criterion described by Taylor et al. (52). The criterion for determining a plateau was an increase in $\dot{V}O_2$ (ml/kg_{BM}/min) between the last two stages of less than 50% of the expected increase, based on the American College of Sports Medicine metabolic equation (29). The criterion varied depending on treadmill speed and ranged from 1.3 ml/kg_{BM}/min (5.5 mph) to 2.2 ml/kg_{BM}/min (9 mph). After completion of the graded test, all subjects (regardless of plateau attainment during the graded test) rested for 20 min and then ran until exhaustion at a grade 2% higher than the grade at the end of the graded test. This process was repeated until a plateau was obtained. Using this protocol, all subjects demonstrated plateau in $\dot{V}O_2$ either during the graded exercise test and/or after the first rest period.

Because we hypothesized that a plateau in $\dot{V}O_2$ might not be evidenced in the heat if performance was limited by hyperthermia, and because it was not possible to do follow-up tests in the heat due to the threat of heat injury, this procedure only was followed in the control condition. For the other conditions, peak $\dot{V}O_2$ (average of the two highest consecutive 30-s values) was used if heart rate was within 5 bpm of the control condition, or if the $\dot{V}O_2$ obtained was the same (within the plateauing criterion) as the $\dot{V}O_{2\max}$ in the control condition. If this criterion was not met, the test was repeated on another day. Five tests in males were repeated because of inability to meet the criterion, and 2 tests in males and 1 test in females were repeated because of equipment failure. The SD of replicate measures (25N and 25W) in 22 subjects for $\dot{V}O_{2\max}$ (l/min), $\dot{V}O_{2\max}$ (ml/kg_{BM}/min), and RER at exhaustion was 1.36, 0.085, and 0.02, respectively.

Rectal temperature was measured with an YSI thermister (model 4491E) inserted 12 cm beyond the anal sphincter. T_{ESO} was measured using an YSI thermister model (4491E) inserted through the nasal cavity into the esophagus a distance equal to one-fourth of the standing height. Mean skin temperature was calculated according to

the formula of Burton (8) from measurements of forearm, back, and thigh skin temperatures with YSI thermistors (model 409B). All temperature probes were connected to YSI telethermometers (models 44TD or 4600). The accuracy of the thermistors was verified by checking against a mercury thermometer prior to use. The SD of replicate measures (25N and 25W) in 22 subjects for rectal, esophageal, and skin temperatures at exhaustion was 0.25, 0.17, and 0.44^o C, respectively.

Heart rate was measured using a Polar Vantage XL Heart Rate Monitor (model 145900). The SD of replicate measures (25N and 25W) in 22 subjects for heart rate at exhaustion was 2.6 bpm. Ratings of perceived exertion (RPE) and motivation to continue were measured by having the subjects holding up the appropriate number of fingers corresponding to the Borg 15-point category scale (4), and a 5-point scale evaluating “motivation to continue” (1 corresponding to “very weak” and 5 corresponding to “very strong”). The SD of replicate measures (25N and 25W) in 22 subjects for RPE and motivation to continue at exhaustion was 0.7 and 0.9, respectively. Performance was measured as the running time to volitional exhaustion during the graded exercise test. The SD of replicate measures (25N and 25W) in 22 subjects for performance time was 0.51 min. Blood lactate was measured with a YSI 2300 STAT plus lactate/glucose analyzer. The SD of replicate measures (25N and 25W) in 22 subjects was 1.1 mmol/l. Finally, body weight was measured with an electronic scale (A&D Co. Ltd., model FW-150KA1). The SD of replicate measures (25N and 25W) in 22 subjects for pre-exercise nude body weight and weight loss was 0.66 and 0.18 kg, respectively.

Statistical Analysis: Statistical analyzes were done with SPSS 10 for Windows (SPSS Inc. Chicago, IL). Data are reported as means \pm SD. A two-way (Gender x Condition) mixed-model repeated-measures ANOVA was used to determine the significance of differences among the measures under the different environmental

conditions, and to test for the presence of an interaction. Planned contrasts (paired samples *t*-test) were used to determine differences between conditions.

A two-way (Gender x Condition) mixed-model repeated-measures ANCOVA with resting temperature held constant was used to determine the significance of differences among the core temperature measures under the different environmental conditions, and to test for the presence of an interaction. Planned contrasts (paired samples *t*-test) were used to determine differences between conditions.

A two-tailed α level of 0.05 was used for all significance tests except for the measures of $\dot{V}O_{2\max}$ and performance, for which a one-tailed α level of 0.05 was used. The significance level was adjusted using the modified Bonferonni adjustment for the family of contrasts performed.

Results

Core and skin temperature measurements at the start of the graded exercise test and at exhaustion are presented in Table 3.2. There was a significant Gender x Condition interaction for all rectal and esophageal temperature measurements, but the pattern of differences among conditions was the same in men and women with one exception. In men, rectal temperature at exhaustion tended to be higher in 45P compared to 45W, whereas in women, this difference was reversed. The manipulations in core temperature (no warm-up, walking, passive preheating) prior to the $\dot{V}O_{2\max}$ test were successful in altering T_{ESO} at the onset of the graded exercise test ($T_{\text{ESOinitial}}$), and the different environmental temperatures produced different rates of increase in T_{ESO} . $T_{\text{ESOinitial}}$ was higher in 45W and 45P than in the other conditions, but did not differ between 45W and 45P by design, which allowed comparison of the effects of passive and active preheating (Table 3.2). $T_{\text{ESOinitial}}$ also was lower in the conditions with no warm-up (25N and 45N) than in conditions with warm-up, allowing us to assess the effects of preheating. The lack of differences in $T_{\text{ESOinitial}}$ in 25W, 35W, and 40W allowed for comparison of the effects of different rates of rise in core temperature

during the $\dot{V}O_{2\max}$ test (0.13 ± 0.03 and $0.13 \pm 0.02^\circ \text{C/min}$ in 25W, 0.16 ± 0.03 and $0.15 \pm 0.01^\circ \text{C/min}$ in 35W, and 0.18 ± 0.03 and $0.16 \pm 0.02^\circ \text{C/min}$ in 40W in men and women, respectively).

Table 3.2: Core and skin temperatures at the start of the graded exercise test and at exhaustion

Males							
Measures	25N	25W	35W	40W	45N	45W	45P
$T_{\text{ESOinitial}}^\circ \text{C}$	$36.4 \pm 0.4^\dagger$	$36.9 \pm 0.4^\dagger \ddagger$	$36.8 \pm 0.2^\dagger$	$37.2 \pm 0.5^\dagger$	$36.6 \pm 0.4^\dagger \ddagger$	37.9 ± 0.3	37.8 ± 0.3
$T_{\text{ESOmax}}^\circ \text{C}$	$38.6 \pm 0.6^\dagger$	$38.6 \pm 0.6^\dagger$	$38.7 \pm 0.6^\dagger$	$39.2 \pm 0.4^\dagger \ddagger$	$39.2 \pm 0.5^\dagger *$	39.7 ± 0.4	39.6 ± 0.4
$T_{\text{RECTALinitial}}^\circ \text{C}$	$36.8 \pm 0.4^\dagger$	$37.2 \pm 0.3^\dagger \ddagger$	$37.2 \pm 0.3^\dagger$	$37.2 \pm 0.3^\dagger$	$36.7 \pm 0.5^\dagger \ddagger$	37.6 ± 0.3	37.6 ± 0.4
$T_{\text{RECTALmax}}^\circ \text{C}$	$37.9 \pm 0.4^\dagger$	$38.2 \pm 0.4^\dagger \ddagger$	$38.3 \pm 0.4^\dagger$	$38.4 \pm 0.4^\dagger$	$38.2 \pm 0.6^\dagger$	38.8 ± 0.5	39.1 ± 0.6
$T_{\text{SKINinitial}}^\circ \text{C}$	$32.3 \pm 0.6^\dagger$	$32.3 \pm 0.7^\dagger$	$35.0 \pm 0.4^\dagger \ddagger$	$36.0 \pm 0.4^\dagger \ddagger$	$34.6 \pm 0.6^\dagger \ddagger *$	37.6 ± 0.3	37.5 ± 0.4
$T_{\text{SKINmax}}^\circ \text{C}$	$32.1 \pm 0.8^\dagger$	$31.9 \pm 0.6^\dagger$	$35.0 \pm 0.5^\dagger \ddagger$	$36.6 \pm 0.4^\dagger \ddagger$	$38.0 \pm 0.4^\dagger \ddagger *$	38.5 ± 0.4	$38.1 \pm 0.4^\dagger$
Females							
$T_{\text{ESOinitial}}^\circ \text{C}$	$36.8 \pm 0.4^\dagger$	$37.3 \pm 0.4^\dagger \ddagger$	$37.2 \pm 0.4^\dagger$	$37.3 \pm 0.3^\dagger$	$36.9 \pm 0.3^\dagger \ddagger$	37.7 ± 0.2	37.6 ± 0.2
$T_{\text{ESOmax}}^\circ \text{C}$	$38.8 \pm 0.3^\dagger$	$38.9 \pm 0.3^\dagger$	$39.0 \pm 0.4^\dagger$	$39.1 \pm 0.4^\dagger$	$39.1 \pm 0.3^\dagger *$	39.4 ± 0.4	39.3 ± 0.5
$T_{\text{RECTALinitial}}^\circ \text{C}$	$37.0 \pm 0.4^\dagger$	$37.3 \pm 0.4^\dagger \ddagger$	$37.3 \pm 0.4^\dagger$	$37.3 \pm 0.3^\dagger$	$37.1 \pm 0.3^\dagger \ddagger$	37.6 ± 0.3	37.6 ± 0.2
$T_{\text{RECTALmax}}^\circ \text{C}$	$38.1 \pm 0.5^\dagger$	$38.3 \pm 0.4^\dagger \ddagger$	$38.3 \pm 0.4^\dagger$	$38.4 \pm 0.4^\dagger$	$38.1 \pm 0.4^\dagger \ddagger$	38.7 ± 0.6	38.4 ± 0.5
$T_{\text{SKINinitial}}^\circ \text{C}$	$32.4 \pm 0.4^\dagger$	$32.1 \pm 0.7^\dagger$	$35.2 \pm 0.4^\dagger \ddagger$	$36.3 \pm 0.3^\dagger \ddagger$	$35.1 \pm 0.6^\dagger \ddagger *$	37.5 ± 0.2	37.6 ± 0.4
$T_{\text{SKINmax}}^\circ \text{C}$	$31.4 \pm 0.6^\dagger$	$31.4 \pm 0.9^\dagger$	$34.9 \pm 0.7^\dagger \ddagger$	$36.4 \pm 0.4^\dagger \ddagger$	$37.9 \pm 0.2^\dagger \ddagger *$	38.3 ± 0.4	$38.0 \pm 0.4^\dagger$

$T_{\text{ESOinitial}}$ = esophageal temperature at the start of the graded exercise test, T_{ESOmax} = esophageal temperature at exhaustion, $T_{\text{RECTALinitial}}$ = rectal temperature at the start of the graded exercise test, $T_{\text{RECTALmax}}$ = rectal temperature at exhaustion, $T_{\text{SKINinitial}}$ = Skin temperature at the start of the graded exercise test, T_{SKINmax} = skin temperature at exhaustion, 25N = 25°C without preheating, 25W = 25°C with active preheating, 35W = 35°C with active preheating, 40W = 40°C with active preheating, 45N = 45°C without preheating, 45W = 45°C with active preheating, 45P = 45°C with passive preheating, $^\dagger = p < 0.05$ from 45W, $^\ddagger = p < 0.05$ compared to the cell left of the value, $*$ = $p < 0.05$ 45N vs. 25N.

T_{ESOmax} was significantly higher in 45W, than in all conditions except 45P (Table 3.2). The similar means in 45W and 45P, and high absolute values, suggest that the critical core temperature was probably reached in 45W and 45P. Similarly, the rectal

temperature at exhaustion was significantly higher in 45° C after preheating (45W and 45P) than in the other conditions. The skin temperature at exhaustion, on the other hand, was higher in 45W than in all the other conditions including 45P and 45N. The significant difference among the three 45° C conditions in skin temperature indicates that exhaustion was not associated with a constant (critical) skin temperature.

Data on $\dot{V}O_{2\max}$; the other metabolic, cardiorespiratory, and perceptual measures assessed at exhaustion, and performance are contained in Table 3.3. There was a significant Gender x Condition interaction in $\dot{V}O_{2\max}$ (l/min) and oxygen (O_2) pulse, but the pattern of differences among conditions was the same in men and women. As expected, a significant main effect was found for these variables with men having higher values. Compared to control, $\dot{V}O_{2\max}$ was decreased under all conditions except 25W. There was a significant, small reduction in 35W (3-5%); a modest reduction in 40W and 45N (~9%); and a large reduction in 45W and 45P (17-19%). Similarly, O_2 pulse at exhaustion was lower in the heat compared to the thermoneutral environment.

Table 3.3: Metabolic, cardiorespiratory, perceptual, and performance variables at exhaustion

Males							
Measures	25N	25W	35W	40W	45N	45W	45P
VO _{2max} , l/min	4.52± 0.46	4.52± 0.47	4.30± 0.43*	4.12± 0.44*	4.13± 0.51*†	3.75± 0.48*	3.70± 0.42*
O ₂ pulse ml/beat	24.0± 3.7	24.5± 3.7	23.1± 3.4*	21.9± 3.6*	22.1± 4.1*†	19.6± 3.4*	19.4± 3.3*
HR, bpm	189± 12	186± 9*	187± 10*	190± 12	189± 11†	193± 12*	192± 10*
RER	1.16± 0.03	1.15± 0.03	1.13± 0.03*	1.12± 0.03*	1.15± 0.03†	1.09± 0.06*	1.09± 0.04*
RPE	19.3± 1.0	18.9± 1.1	18.8± 1.6	19.3± 1.2	19.0± 1.1	19.2± 1.0	18.9± 1.3
Motivation to continue	3.1± 1.5	2.8± 1.4	2.7± 1.6	3.0± 1.4	3.0± 1.3	3.1± 1.2	2.7± 1.4
Lactate, mmol/l	8.98± 1.60	9.40± 2.82	7.81± 1.51*	8.51± 1.77	7.89± 2.18*†	5.96± 2.07*	5.24± 1.58*
Wt. loss, kg	0.36± 0.33	0.05± 0.11*	0.28± 0.28	0.38± 0.32	0.31± 0.25	0.45± 0.24	0.78± 0.34*†
P _{TIME} , min	13.01± 1.18	13.15± 1.41	11.76± 1.22*	11.33± 1.59*	11.51± 1.15*†	8.77± 1.31*	8.30± 1.25*
Females							
VO _{2max} , l/min	3.01± 0.45	3.01± 0.44	2.91± 0.47*	2.75± 0.42*	2.75± 0.44*†	2.52± 0.40*	2.44± 0.40*
O ₂ pulse ml/beat	15.9± 2.4	15.9± 2.4	15.4± 2.6*	14.4± 2.2*	14.5± 2.3*†	13.1± 2.0*	12.8± 2.0*
HR, bpm	190± 8	189± 7	189± 7	190± 8	190± 7	192± 8*	190± 7
RER	1.18± 0.03	1.16± 0.04	1.14± 0.05*	1.15± 0.04*	1.14± 0.05*	1.10± 0.04*	1.06± 0.07*
RPE	19.4± 0.7	19.4± 0.5	19.1± 0.8	19.5± 0.5	19.4± 0.7	19.1± 0.8	19.3± 0.6
Motivation to continue	2.2± 1.1	2.8± 1.3	2.9± 1.2	2.8± 1.5	2.8± 1.5	2.5± 0.9	2.2± 1.1
Lactate, mmol/l	8.13± 1.23	7.45± 1.68	7.21± 1.87*	6.54± 1.58*	6.60± 1.70*†	5.39± 1.60*	4.75± 1.51*
Wt. loss, kg	0.32± 0.22	0.14± 0.11*	0.22± 0.20	0.18± 0.18	0.15± 0.13*	0.40± 0.24	0.48± 0.31
P _{TIME} , min	13.00± 1.13	12.64± 1.45	12.10± 1.82*	10.99± 1.48*	11.38± 1.43*†	9.05± 1.79*	8.32± 1.91*

VO_{2max} = maximal oxygen uptake, P_{TIME} = performance time, O₂ pulse = oxygen pulse, HR = heart rate, RER = respiratory exchange ratio, RPE = ratings of perceived exertion, Wt. loss = weight loss, 25N = 25° C without preheating, 25W = 25° C with active preheating, 35W = 35° C with active preheating, 40W = 40° C with active preheating, 45N = 45° C without preheating, 45W = 45° C with active preheating, 45P = 45° C with passive preheating, * = p < 0.05 from 25N, † = p < 0.05 45W vs 45N or 45P.

The identical $\dot{V}O_{2\max}$ in 25N and 25W indicates that, in absence of environmental heat stress and a large increase in $T_{\text{ESOinitial}}$, the 20-min walk prior to the graded exercise test had no effect on $\dot{V}O_{2\max}$. In contrast, the significantly lower $\dot{V}O_{2\max}$ in 45W than in 45N indicates that the 20-min walk prior to the graded exercise test, which was accompanied by an increase in $T_{\text{ESOinitial}}$, and resulted in significantly higher T_{ESOmax} , did contribute to a reduction in $\dot{V}O_{2\max}$. The lack of a significant difference in $\dot{V}O_{2\max}$ in 45W and 45P conditions, in which the increase in $T_{\text{ESOinitial}}$ and T_{ESOmax} were not different, strongly suggests that the significantly lower $\dot{V}O_{2\max}$ under these two condition was related to the higher T_{ESOmax} and not to some other effect of prior exercise. Heat stress by itself also reduced $\dot{V}O_{2\max}$ without attainment of a critical core temperature, as was evidenced by the lower $\dot{V}O_{2\max}$ in 45N compared to 25N.

There is strong evidence that $\dot{V}O_{2\max}$ was attained in each of the conditions. Physiological and perceptual indicators of effort were similar across conditions at exhaustion, except for blood lactate (Table 3.3). Blood lactate was lower in the heat, except in 40W in men, in which it was not different from control. A significant heart rate Gender X Condition interaction at exhaustion was found, but the pattern of differences among conditions was the same for men and women.

Dehydration also did not appear to contribute to differences in $\dot{V}O_{2\max}$ among conditions. Weight loss during the test was small, or less than 0.9% of body weight under all conditions, except in 45P in men in which it was 1.1% (Table 3.3). Weight loss was not greater in the heat compared to the control condition, except in 45P in men. Weight loss responses were somewhat different in men and women, resulting in a significant Gender x Condition interaction. Men tended to have greater weight loss in 40W compared to 25N and in 45N compared to 35W, whereas the pattern in women for these conditions was the opposite.

Significant decreases in performance time were observed in the heat in men and women (Table 3.3). The percentage reductions in performance were larger than the

percentage reductions in $\dot{V}O_{2\max}$. In 35W, the reduction in run time was 7-10 %; in 40W, 13-15%; in 45N, ~12%; in 45W, 30-33%; and in 45P, ~36%.

The relation between the reduction in $\dot{V}O_{2\max}$ from control and the difference in T_{ESOmax} from T_{ESOmax} in 45W, which we operationally defined as the critical core temperature and used as the upper-limit reference for T_{ESOmax} , is shown in Figures 3.1A and B. When T_{ESOmax} was well below T_{ESOmax} in 45W, $\dot{V}O_{2\max}$ did not differ much from the control value. On the other hand, when T_{ESOmax} approached the T_{ESOmax} in 45W, larger reductions in $\dot{V}O_{2\max}$ were observed. The same relation between the reductions in performance time from control and the difference in T_{ESOmax} from T_{ESOmax} in 45W is depicted in Figures 3.1C and D.

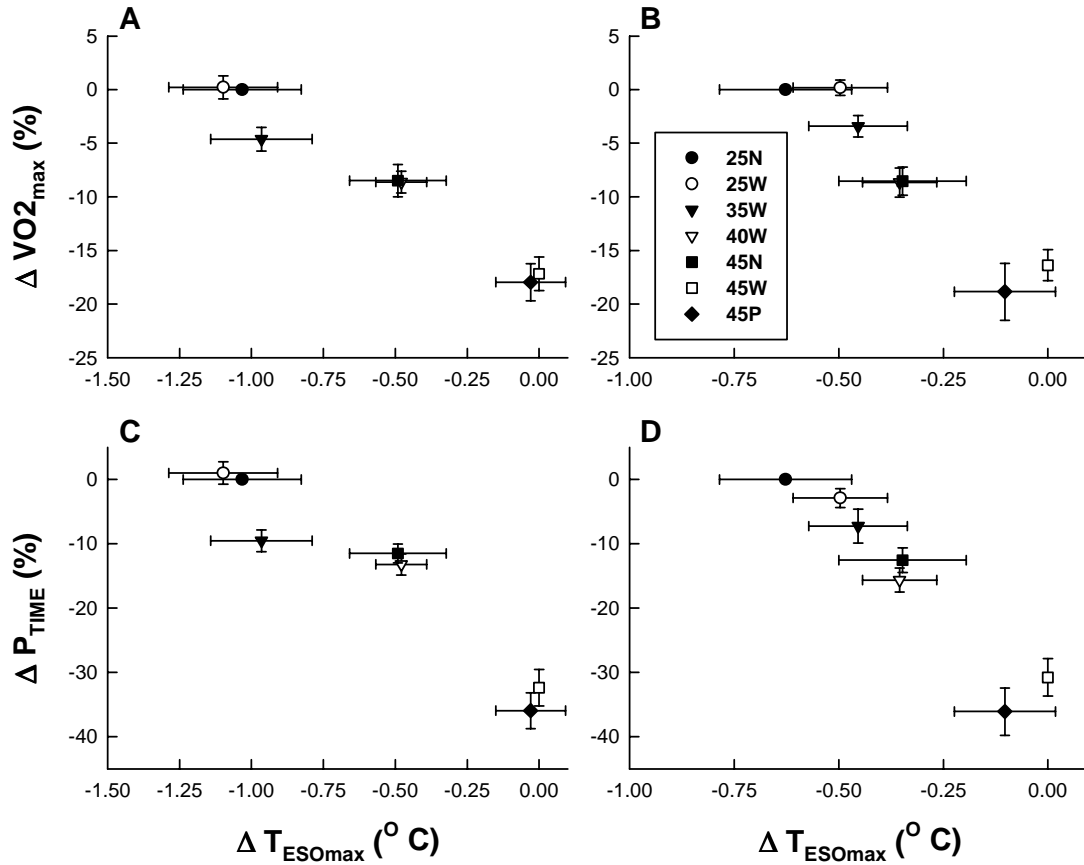


Figure 3.1: The relationship between reductions in $\dot{V}O_{2\max}$ and performance time, and deviations in T_{ESOmax} from T_{ESOmax} in 45W. Panels A and C are men; panels B and D are women. $\Delta \dot{V}O_{2\max}$ = reduction in maximal oxygen uptake from 25N, ΔP_{TIME} = reductions in performance time from 25N, ΔT_{ESOmax} = deviations in the esophageal temperature at exhaustion from the esophageal temperature in 45W, 25N = 25 $^{\circ}\text{C}$ without preheating, 25W = 25 $^{\circ}\text{C}$ with active preheating, 35W = 35 $^{\circ}\text{C}$ with active preheating, 40W = 40 $^{\circ}\text{C}$ with active preheating, 45N = 45 $^{\circ}\text{C}$ without preheating, 45W = 45 $^{\circ}\text{C}$ with active preheating, 45P = 45 $^{\circ}\text{C}$ with passive preheating. Values are means and SE.

There were considerable individual differences in the environmental conditions at which attainment of critical core temperature (T_{ESOmax} in 45W \pm 0.2) was first observed (Figures 3.2A-D). Two individuals reached the critical core temperature in 25N, 1 in 25W, 4 in 35W, 2 in 40W, and 1 in 45N; almost half of the subjects appeared to reach their critical core temperature before the highest level of heat stress (45W and 45P). Some individuals demonstrated clear attainment of a critical core temperature even at low ambient temperatures as illustrated in a representative subject in Figures 3.2A and

C. In 25W, 35W, and 45N, this individual had no reduction in $\dot{V}O_{2\max}$ (Figure 3.2A), because the work intensity that elicited control $\dot{V}O_{2\max}$ was attained before critical core temperature was reached. This lack of reduction in $\dot{V}O_{2\max}$ was reflected in minor reductions in performance time (Figure 3.2C). At higher levels of heat stress, $\dot{V}O_{2\max}$ was markedly reduced, apparently because exercise was terminated at the critical core temperature, which was reached early in the protocol (large reductions in performance time), because of higher $T_{\text{ESOinitial}}$ (45W, 45P) or more rapid rise in T_{ESO} (40W). Other individuals did not attain the critical core temperature until the highest level of heat stress (45W, 45P) as illustrated in a representative subject in Figures 3.2B and D. Consequently, this individual demonstrated no reduction in $\dot{V}O_{2\max}$ until in 45W and 45P (Figure 3.2B), and performance time was not affected much until these conditions (Figure 3.2D). The magnitude of the reductions were small because of the length of time it took to reach the critical core temperature, which permitted exercise to continue almost to the same work intensity as that which elicited the control $\dot{V}O_{2\max}$.

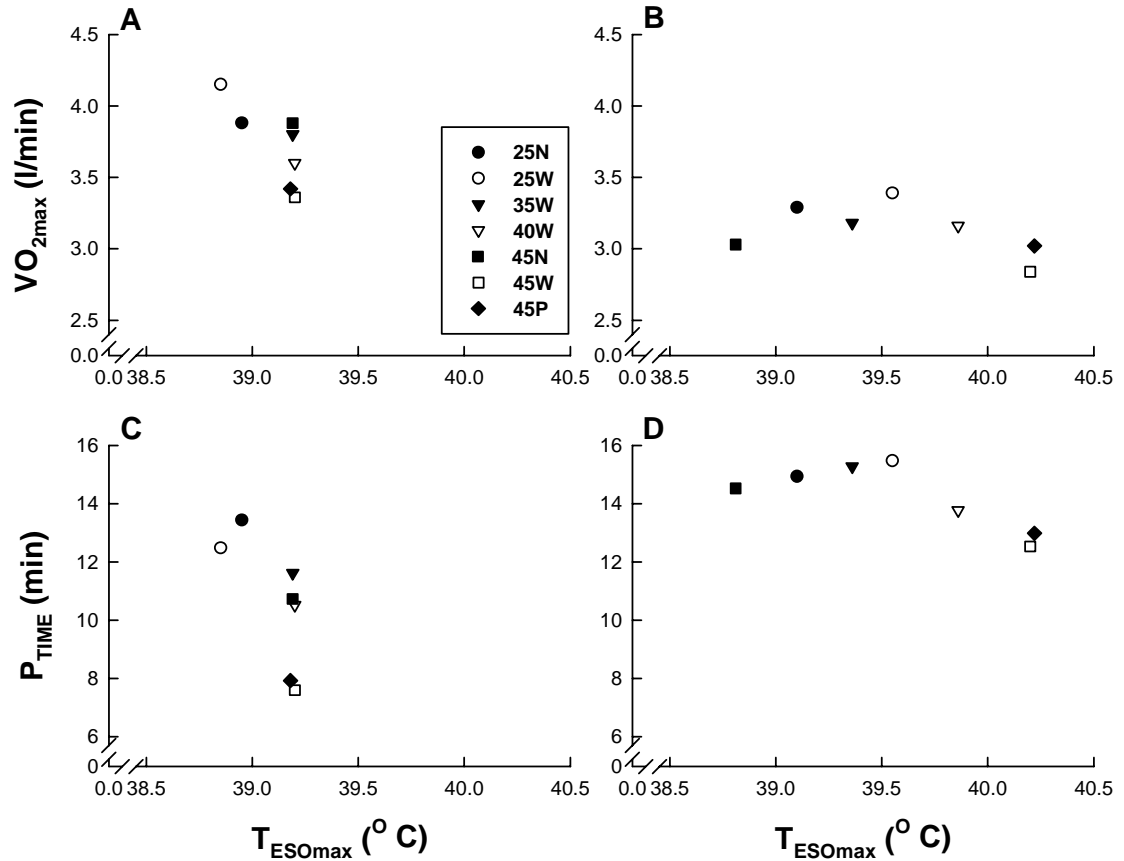


Figure 3.2: Representative data from two subjects illustrating variability in the relation of T_{ESOmax} to $\dot{V}O_{2max}$ and performance time. A) Illustrates reduction in $\dot{V}O_{2max}$ with clear attainment of a critical core temperature. B) Illustrates lack of reduction in $\dot{V}O_{2max}$ without attainment of a critical core temperature. C) Illustrates reduction in performance time with a clear attainment of a critical core temperature. D) Illustrates lack of reduction in performance time without attainment of a critical core temperature. $\dot{V}O_{2max}$ = maximal oxygen uptake, P_{TIME} = performance time, T_{ESOmax} = esophageal temperature at exhaustion, 25N = 25 $^{\circ}C$ without preheating, 25W = 25 $^{\circ}C$ with active preheating, 35W = 35 $^{\circ}C$ with active preheating, 40W = 40 $^{\circ}C$ with active preheating, 45N = 45 $^{\circ}C$ without preheating, 45W = 45 $^{\circ}C$ with active preheating, 45P = 45 $^{\circ}C$ with passive preheating.

There was a strong linear relationship ($r = 0.84$) between the decrease in $\dot{V}O_{2max}$ and the decrease in performance time in the heat in men (Figure 3.3A). Essentially the same relationship ($r = 0.82$) was observed in women (Figure 3.3B).

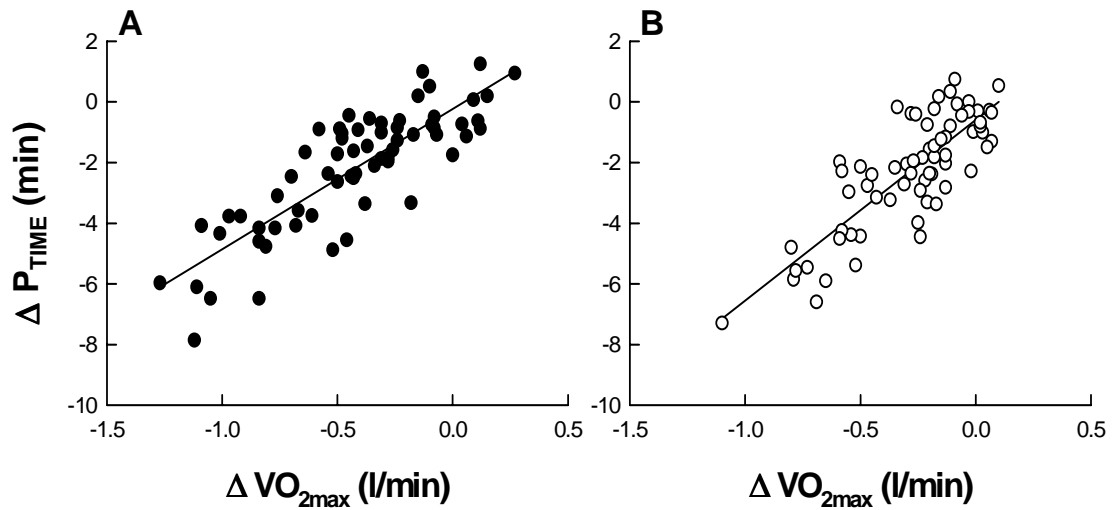


Figure 3.3: The relation between the reduction in performance time and reduction in $\dot{V}O_{2\max}$ in A) men ($r = 0.84$, SEE 1.08 min) and B) women ($r = 0.82$, SEE 1.09 min). ΔP_{TIME} = reductions in performance time from 25N, $\Delta \dot{V}O_{2\max}$ = reduction in maximal oxygen uptake from 25N.

Discussion

The major finding of this study was that $\dot{V}O_{2\max}$ was markedly reduced under environmental conditions (45W and 45P) in which critical core temperature was reached at an exercise intensity below that needed to elicit $\dot{V}O_{2\max}$ under thermoneutral conditions. This finding suggests that a critical level of hyperthermia can limit $\dot{V}O_{2\max}$. Secondly, decreases in performance were related to the reductions in $\dot{V}O_{2\max}$, suggesting that reduced $\dot{V}O_{2\max}$ can be a cause for reduced performance of prolonged, strenuous exercise in the heat. A final important finding was that the relations of $\dot{V}O_{2\max}$ and performance to T_{ESOmax} were very similar in men and women, indicating that the effect of hyperthermia on $\dot{V}O_{2\max}$ and performance was not related to gender.

To our knowledge, this study is the first to assess the effects of environmental and core temperatures on $\dot{V}O_{2\max}$ and performance by systematically altering $T_{\text{ESOinitial}}$ and by altering the rate of rise in core temperature during the test. The manipulations caused T_{ESOmax} to be reached at different work intensities, permitting us to study the effect of attainment of critical core temperature on $\dot{V}O_{2\max}$ and performance. Previous

studies have not had the data necessary to determine the effect of attainment of a critical core temperature on $\dot{V}O_{2\max}$ (14,25,36,37,39-43,46,48,52,55).

Considerable data suggest that attainment of critical core temperature in prolonged, strenuous exercise may limit work capacity or endurance performance in humans (11-13,16,20-22,26,28,30,32-35,45,49,53) and animals (1,10,18,23,51,54,56). In these studies, people and animals fatigued at similar core temperatures despite markedly different exercise times to exhaustion. In several studies, most alternative causes of fatigue such as lactate accumulation, glycogen depletion, or glucose or free-fatty acid availability were ruled out and cardiovascular strain was not maximal. In these studies, fatigue at a critical (constant) core temperature for an individual occurs regardless of ambient or skin temperatures (13,22,28,49) initial core temperature (18,22,54), rate of rise in core temperature (18,22), exercise duration (10,18,22,23,33, 51,54,56) exercise intensity (32,45), climate (32), acclimatization stage (11,33,35), state of hydration (11,26), fluid ingestion (12), stage in menstrual cycle (53), melatonin intake (30), or carbohydrate availability (16). In most human studies, exhaustion occurred at a mean core temperature between 39.3 and 40.2° C (20-22,28,33-35,44), although others have reported exhaustion at lower body temperatures at exhaustion in uncompensable heat stress (11-13,26,30,32,45,47,49,53). The core temperature at exhaustion has been found to be lower in subjects who were untrained (11), dehydrated (47), and exercised in full chemical and/or protective clothing (32) early in the morning (30). Some of these studies (11,12,30,32,47,49,53) have used very prolonged (2-3 h), low-intensity exercise, which could increase the thermal load (core temperature above a certain temperature level multiplied by the time above that level) and cause exhaustion due to cardiovascular strain. The protective clothing, extensive dehydration, and lower intensity exercise conditions used in these studies also could lower the critical core temperature. In addition, in many of these studies exercise was terminated

once rectal temperature reached 39.3-39.5° C, preventing some subjects from reaching their critical core temperature, resulting in lower mean rectal temperature at exhaustion.

In our study, T_{ESOmax} in men and women in 45W and 45P was higher than in the other conditions and similar to core temperature at exhaustion in uncompensable heat stress in many other studies (20-22,28,33-35,44), suggesting that critical core temperature was attained under these conditions. Based on the very high level of heat stress, and the high T_{ESOmax} values obtained, we believe that all subjects reached their individual critical core temperature in 45W and/or 45P conditions. We operationally defined the T_{ESOmax} in 45W as the critical core temperature, and used it as an upper-limit temperature reference for data analysis, because on average, T_{ESOmax} was slightly higher in that condition than in 45P. Reasons for terminating exercise support our assumption that a critical core temperature causing fatigue was probably attained in 45W and 45P. When asked why they stopped running in 45W or 45P, the subjects reported being too hot or dizzy to continue in 65% of cases. Complaints of being extremely dizzy or hot have been reported by subjects at exhaustion due to attainment of a critical core temperature in other studies (34,35). In the other conditions, subjects complained of being too hot or dizzy to continue in only 9% of the cases.

There was considerable variability in the minimum environmental temperature at which individual critical core temperatures (T_{ESOmax} in 45W \pm 0.2) appeared to have been attained. Individuals who did not appear to reach their critical core temperature until 45W and 45P usually had higher critical core temperatures. The $\dot{V}\text{O}_{2\text{max}}$ test was probably too short (10-15 min) for them to reach their critical core temperature in lower ambient temperatures. Therefore, the mean T_{ESOmax} was lower in all conditions compared to 45W and 45P.

This study is the first to test the hypothesis that attainment of a critical core temperature may limit $\dot{V}\text{O}_{2\text{max}}$ in the heat during uphill treadmill running, which generally results in the highest $\dot{V}\text{O}_2$ values attainable (2). Results from studies on the

effect of heat stress on $\dot{V}O_{2\max}$ have been equivocal. Some studies report no reduction (39,41,43,55), whereas others report very small (150-300 ml/min or 3-8%), often trivial, reductions (14,25,37,40,42,43,46,48,52). The first study to find a marked reduction in $\dot{V}O_{2\max}$ in the heat was that of Pirnay et al. (37). They found a 985 ml/min or 25% reduction after 20 min of light walking (active preheating) in 46° C. Without preheating, only a 7% (225 ml/min) reduction was observed in a different group of subjects. Similarly, Nybo et al. (36) recently reported that $\dot{V}O_{2\max}$ was reduced 16% (750 ml/min) after elevating core temperature 1° C prior to the test. The findings of the present study are consistent with these two studies and help to reconcile the differences between these studies and others that have found lesser decreases in $\dot{V}O_{2\max}$. In our study, under hot environmental conditions in which the initial core temperature was not very high (45N, 35W), or the rate of rise in core temperature was not very fast (35W, 40W), $\dot{V}O_{2\max}$ was reduced 3-9% (100-400 ml/min), or on the same order as reported by most other studies. In 45W and 45P, in which the initial core temperature and its rate of rise were higher, much greater reductions of 17-19% (500-820 ml/min) were observed. The larger reductions are similar in absolute magnitude to the reductions found by Nybo et al. (36) but smaller than reported by Pirnay et al. (37).

Attainment of a critical core temperature is the most likely explanation for the larger reduction in $\dot{V}O_{2\max}$ and performance observed in 45W and 45P in this study. The attainment of the critical core temperature appears to cause subjects to terminate exercise before $\dot{V}O_2$ reaches levels comparable to the $\dot{V}O_{2\max}$ attained in a neutral environment (25N and 25W). Lack of effort was not a likely cause for the large reductions in $\dot{V}O_{2\max}$ based on the physiological and perceptual data collected. RER was ~1.1 or higher under all conditions, and similar means for heart rate and RPE in all conditions, indicate that a maximal effort was given. Blood lactate accumulation was lower in the heat, but that more likely reflects reduced work rate at $\dot{V}O_{2\max}$ rather than

lack of effort. Weight loss was similar in all the conditions, suggesting that the reductions were not due to dehydration.

This study is the first to demonstrate a relation between attainment of a critical core temperature and reduction in $\dot{V}O_{2\max}$. Previous studies (40,43,46) have not found a relationship between core temperature at exhaustion and $\dot{V}O_{2\max}$ or reduction in $\dot{V}O_{2\max}$. There are several reasons for the difference. First, other studies have not specifically investigated the relation of attainment of a critical level of core temperature and reduction in $\dot{V}O_{2\max}$. Second, most of these studies used rectal temperature as a measure of core temperature. Rectal temperature responds more slowly than esophageal temperature and does not reflect the rapid change in core temperature that occurs during a short $\dot{V}O_{2\max}$ test, as reflected by the difference in T_{ESOmax} and rectal temperature at exhaustion observed in our study (Table 3.2). We also found no relation between rectal temperature at exhaustion and $\dot{V}O_{2\max}$ ($r = 0.07$). Only when reductions in $\dot{V}O_{2\max}$ from control (25N) were plotted against the differences in T_{ESOmax} from T_{ESOmax} in 45W (operationally defined as the critical core temperature), was an inverse relation evident (Figures 3.1A and B). Large reductions in $\dot{V}O_{2\max}$ were observed when T_{ESOmax} was near the apparent critical core temperature, whereas smaller reductions were observed when T_{ESOmax} was much below the critical core temperature. Third, a critical core temperature that causes fatigue would be expected during a relatively brief $\dot{V}O_{2\max}$ test only if core body temperature is elevated initially. Despite similar rates of rise in core temperature in the 45°C conditions (0.23 ± 0.04 and $0.19 \pm 0.03^\circ \text{C/min}$ in 45N, 0.20 ± 0.03 and $0.19 \pm 0.02^\circ \text{C/min}$ in 45W, and 0.22 ± 0.05 and $0.21 \pm 0.03^\circ \text{C/min}$ in 45W in men and women, respectively) in the present study, the T_{ESOmax} was lower in the 45N than in 45W and 45P, because the $T_{\text{ESOinitial}}$ was lower in 45N. The only studies to report reductions in $\dot{V}O_{2\max}$ in the heat larger than 15%, have preheated subjects prior to the $\dot{V}O_{2\max}$ test (36,37). Without preheating, there is insufficient time for the critical core body temperature to be attained. Nybo et al. (36), however, reported a 16%

reduction in $\dot{V}O_{2\max}$ after elevating initial core temperature, apparently without attainment of the critical core temperature, although the T_{ESOMax} in their study was high (39.0°C) and higher than during the control tests ($38.5\text{--}38.7^{\circ}\text{C}$). Because higher T_{ESOMax} ($39.5\text{--}40.2^{\circ}\text{C}$) were obtained in prior prolonged submaximal studies in their laboratory (22,33,35), they attributed the decrease in $\dot{V}O_{2\max}$ to lower stroke volume and not to attainment of a critical core temperature. Our data essentially agree with theirs (36), and our values for T_{ESOMax} are within the range of the values reported in their earlier studies (22,33,35). The different levels of T_{ESOMax} obtained in the present study and that of Nybo et al. (36), could be due to the duration of the $\dot{V}O_{2\max}$ test (10-15 min vs < 3 min, respectively).

Physical work capacity also was decreased in the heat in this study confirming the findings of others (13,16,22,28,33,36,40,42,43,49). Performance time was affected much more than $\dot{V}O_{2\max}$, but the strength of the relation between T_{ESOMax} and decrease in performance time was similar to the relation between T_{ESOMax} and reduction in $\dot{V}O_{2\max}$. The reductions in performance in men and women ranged from 7-10% in 35W to 36% in 45P. The reason for greater percentage reductions in performance time than $\dot{V}O_{2\max}$ may be explained by the nature of the $\dot{V}O_{2\max}$ test used in this study. Persons that demonstrated a good plateau in $\dot{V}O_{2\max}$ (2-4 min) in a temperate (25N and 25W) environment still reached (or were close to) that $\dot{V}O_{2\max}$ in slightly hotter ambient temperature (35W), despite their performance time being 2-4 min shorter.

The strong relation between the reductions in $\dot{V}O_{2\max}$ and performance time in this study is a unique finding, since others have reported reductions in performance without reductions in $\dot{V}O_{2\max}$ (40,41,43). The strong relation found in this study is probably because of the preheating and the different environmental temperatures, which caused attainment of the critical core temperature. Attainment of the critical core temperature caused larger decreases in $\dot{V}O_{2\max}$ and performance than in previous studies, and appears to explain the concomitant large reductions in the hot test

environment. The strong relation between reductions in $\dot{V}O_{2\max}$ and work capacity observed in this study is important because it suggests that after a modest duration of sustained exercise in the heat that elevates core temperature, $\dot{V}O_{2\max}$ may be reduced, which in turn, is associated with reduced performance.

Traditionally, the reduction in performance (and $\dot{V}O_{2\max}$) as result of heat stress has been explained by reduction in stroke volume and cardiac output due increased blood flow to the skin for thermoregulation, resulting in peripheral displacement of blood volume (38,41) and possibly lower muscle blood flow (circulatory strain). In addition, glycogen breakdown (17) and oxidative stress are increased in the heat (31), and mitochondrial phosphorylative efficiency is reduced at muscle temperatures above 40° C (6), all of which may detrimentally affect skeletal muscle metabolic function (15).

More recently though, it has been shown that neither leg blood flow nor leg $\dot{V}O_2$ is reduced in the heat, even during exhaustive exercise in men (20,21,33-35,44) and animals (1,23). Furthermore, other metabolic causes of fatigue such as lactate accumulation, glycogen depletion, or availability of glucose or free-fatty acids have been ruled out as causes of premature fatigue in the heat (21,33-35). Excluding most other causes of fatigue, these studies have generally attributed exhaustion during prolonged strenuous exercise in the heat to attainment of a high core temperature.

The rise in core temperature to critical levels in the heat could be explained by reduction in skin blood flow at the expense of thermoregulation. Skin blood flow has been reported to be reduced at high core temperatures (5,23) and during exhaustive exercise (23,55). In addition, Savard et al. (44) demonstrated that the maximal level of skin blood flow, which occurred at skin temperature of 38° C, was the same in one-and two-legged exercise despite very different esophageal temperatures (37.9 vs 39.4° C, respectively), suggesting that skin vasoconstriction occurred in two-legged exercise. These findings indicate that during one-legged exercise the central circulation was able

to meet blood flow demands of both skin and muscle, because of much lower metabolic rate, retarding the rise in esophageal temperature, whereas in two-legged exercise, muscle blood flow was maintained at the expense of skin blood flow and rise in core temperature.

The mechanisms underlying the reduced $\dot{V}O_{2\max}$ and performance associated with attainment of a critical core temperature have not been identified. The high core temperature is thought to affect the central nervous system and mental functioning (7,33,34). Muscular discomfort and circulatory strain in addition to thermal discomfort from heat storage and lack of heat dissipation, may counteract motivation and thus gradually reduce the drive to exercise with increasing body temperature (7). However, previous studies have not attempted to quantify the effect of different environmental conditions on motivation to continue. We found no differences among conditions in “motivation to continue” assessed at the time of fatigue using a 5-point scale, and could, therefore, not confirm this hypothesis. Similarly, Nielsen et al. (33) found no difference in the ability to recruit motor units in a maximal contraction before and after exercise to exhaustion that appeared to be limited by attainment of a critical core temperature.

It also is unclear whether it is muscle, core, or brain temperature that is associated with fatigue. Brain temperature as the limiting factor for exercise is appealing given the subjective reports of dizziness, confusion, and being too hot to continue at exhaustion. Selective brain cooling with increasing body temperature that occurs in some animal species (e.g., goats and gazelles) (9,10,50) suggests that protecting the brain from high temperature is important. On the other hand, the skeletal muscles are the origin of increased heat production during exercise, and Febbrario (15) has argued that high skeletal muscle temperatures causing dysfunction in metabolic processes could be the limiting factor for exercise in the heat. Skeletal muscle temperature at exhaustion has been reported to be slightly higher (0.6°C) than T_{ESOmax}

(22). A critical level of skin temperature does not seem to cause fatigue as was eloquently demonstrated by Gonzalez-Alonso et al. (22), who found that subjects fatigued at same core and muscle temperatures, but at markedly different skin temperatures. The findings of the present study and others (28) support their findings.

An alternate mechanism through which attainment of the critical core temperature may have limited $\dot{V}O_{2\max}$ and work capacity in the present study is that the high (critical) core temperature was associated with the upper limit in cardiovascular capacity, as suggested by the similar maximal heart rates in all of the conditions in our study. Assuming that the arterio-venous O_2 difference was not reduced (41), the lower O_2 pulse in 45W and 45P would reflect reduced stroke volume. Thus $\dot{V}O_{2\max}$ would have been attained at a lower maximal cardiac output under these conditions, which is consistent with the mechanism proposed by Rowell (38). Because of the higher exercise intensity, the conditions in our study were different than in other studies in which fatigue at a critical core temperature did not appear to coincide with a maximal level of circulatory strain (33,35). In these studies, fatigue during prolonged, constant-rate exercise occurred when heart rate was submaximal and when there was no reduction in cardiac output or muscle blood flow compared to early in the exercise. In our study, the exercise intensity was increased progressively and fatigue occurred at the maximal heart rate. It is possible that sympathetic nervous system activity increased in proportion to core body temperature (24), increasing vascular resistance and decreasing stroke volume. However, there is a possibility that attainment of a critical core temperature in 45W and 45P prevented maximal stroke volume attainment, which has been found to increase progressively up to $\dot{V}O_{2\max}$ in endurance trained individuals (19), and to be restored at maximal level of exercise in the heat (55).

This study is the first to report T_{ESOmax} from strenuous exercise, and the effect of heat on $\dot{V}O_{2\max}$ and work capacity, in women. We found that T_{ESOmax} in men and women was similar, and the effects of heat and T_{ESOmax} on $\dot{V}O_{2\max}$ and performance were

essentially the same in men and women. Our findings suggest that men and women are subject to the same limitations in $\dot{V}O_{2\max}$ and work capacity during exercise in the heat. These data are consistent with studies involving lower intensity exercise that have concluded that thermoregulation and exercise tolerance in the heat is similar in men and women (3).

We conclude that large reductions in $\dot{V}O_{2\max}$ and work capacity in men and women in severe, uncompensable heat stress are associated with attainment of a critical core temperature. The strong relation between reductions in $\dot{V}O_{2\max}$ and in work capacity in the heat appears to depend, in part, on the effect of attainment of a critical core temperature on both measures. Men and women do not differ in their maximal body temperature responses, or in the relation of the core temperature to the reductions in $\dot{V}O_{2\max}$ and work capacity in the heat.

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References

1. Armstrong, R. B., M. D. Delp, E. F. Goljan, and M. H. Laughlin. Progressive elevations in muscle blood flow during prolonged exercise in swine. *J.Appl.Physiol.* 63: 285-291, 1987.
2. Astrand, P.-O. and B. Saltin. Maximal oxygen uptake and heart rate in various types of muscular activity. *J.Appl.Physiol.* 16: 977-981, 1961.
3. Avellini, B. A., E. Kamon, and J. T. Krajewski. Physiological responses of physically fit men and women to acclimation to humid heat. *J.Appl.Physiol.* 49: 254-261, 1980.
4. Borg, G. The perception of physical performance. In Shephard, R. J., ed., *Frontiers of Fitness*. Springfield, IL, Charles C Thomas Publisher. 1971, 280-294.
5. Brengelmann, G. L., J. M. Johnson, L. Hermansen, and L. B. Rowell. Altered control of skin blood flow during exercise at high internal temperature. *J.Appl.Physiol.* 43: 790-794, 1977.
6. Brooks, G. A., K. J. Hittelman, J. A. Faulkner, and R. E. Beyer. Temperature, skeletal muscle mitochondrial functions, and oxygen debt. *Am.J.Physiol.* 220: 1053-1059, 1971.
7. Bruck, K. and H. Olschewski. Body temperature related factors diminishing the drive to exercise. *Can.J.Physiol.Pharmacol.* 65: 1274-1280, 1987.
8. Burton, A. C. Human calorimetry II. The average temperature of the tissues of the body. *J.Nutr.* 9: 61-280, 1935.
9. Caputa, M., G. Feistkorn, and C. Jessen. Effects of brain and trunk temperatures on exercise performance in goats. *Pflugers Arch.* 406: 184-189, 1986.
10. Caputa, M., W. Kadiela, and J. Narebski. Cerebral temperature regulation in resting and running guinea-pigs (*cavia porcellus*). *J.Therm.Biol.* 8: 265-272, 1983.

11. Cheung, S. S. and T. M. McLellan. Heat acclimation, aerobic fitness, and hydration effects on tolerance during uncompensable heat stress. *J.Appl.Physiol.* 84: 1731-1739, 1998.
12. Cheung, S. S. and T. M. McLellan. Influence of hydration status and fluid replacement on heat tolerance while wearing NBC protective clothing. *Eur.J.Appl.Physiol.* 77: 139-148, 1998.
13. Craig, F. N., H. W. Garren, H. Frankel, and W. V. Blevins. Heat load and voluntary tolerance time. *J.Appl.Physiol.* 6: 634-644, 1954.
14. Dimri, G. P., M. S. Malhotra, J. Sen Gupta, T. S. Kumar, and B. S. Aora. Alterations in aerobic-anaerobic metabolic proportions of metabolism during work in heat. *Eur.J.Appl.Physiol.* 45: 43-50, 1980.
15. Febbraio, M. A. Does muscle function and metabolism affect exercise performance in the heat? *Exerc.Sport Sci.Rev.* 28: 171-176, 2000.
16. Febbraio, M. A., P. Murton, S. E. Selig, S. A. Clark, D. L. Lambert, D. J. Angus, and M. F. Carey. Effect of CHO ingestion on exercise metabolism and performance in different ambient temperatures. *Med.Sci.Sports Exerc.* 28: 1380-1387, 1996.
17. Fink, W. J., D. L. Costill, and W. J. VanHandle. Leg muscle metabolism during exercise in the heat and cold. *Eur.J.Appl.Physiol.* 34: 183-190, 1975.
18. Fuller, A., R. N. Carter, and D. Mitchell. Brain and abdominal temperatures at fatigue in rats exercising in the heat. *J.Appl.Physiol.* 84: 877-883, 1998.
19. Gledhill, N., D. Cox, and R. Jamnik. Endurance athletes' stroke volume does not plateau: major advantage is diastolic function. *Med.Sci.Sports Exerc.* 26: 1116-1121, 1994.
20. Gonzalez-Alonso, J., J. A. L. Calbet, and B. Nielsen. Muscle blood flow is reduced with dehydration during prolonged exercise in humans. *J.Physiol.* 513: 895-905, 1998.

21. Gonzalez-Alonso, J., J. A. L. Calbet, and B. Nielsen. Metabolic and thermodynamic responses to dehydration-induced reductions in muscle blood flow in exercising humans. *J.Physiol.* 520: 577-589, 1999.
22. Gonzalez-Alonso, J., C. Teller, S. L. Andersen, F. B. Jensen, T. Hyldig, and B. Nielsen. Influence of body temperature on the development of fatigue during prolonged exercise in the heat. *J.Appl.Physiol.* 86: 1032-1039, 1999.
23. Hales, J. R. S., M. Khogali, A. A. Fawcett, and M. K. Y. Mustafa. Circulatory changes associated with heat stroke: Observations in an experimental animal model. *Clin.Exp.Pharmacol.Physiol.* 14: 761-777, 1987.
24. Kim, Y. D., R. Lake, D. E. Lees, W. H. Schuette, J. M. Bull, V. Weise, and I. J. Kopin. Hemodynamic and plasma catecholamine responses to hyperthermic cancer therapy in humans. *Am.J.Physiol.* 237: H570-H574, 1979.
25. Klausen, K., D. B. Dill, E. E. Jr. Phillips, and D. McGregor. Metabolic reactions to work in the desert. *J.Appl.Physiol.* 22: 292-296, 1967.
26. Latzka, W. A., M. N. Sawka, S. J. Montain, G. S. Skrinar, R. A. Fielding, R. P. Matott, and K. B. Pandolf. Hyperhydration: tolerance and cardiovascular effects during uncompensable exercise-heat stress. *J.Appl.Physiol.* 84: 1858-1864, 1998.
27. Lipsey, M. W. Design Sensitivity: Statistical Power for Experimental Research. Newbury Park, CA, Sage Publications. 1990, 69-145.
28. MacDougal, J. D., W. G. Reddan, C. R. Layton, and J. A. Dempsey. Effects of metabolic hyperthermia on performance during heavy prolonged exercise. *J.Appl.Physiol.* 36: 538-544, 1974.
29. Mahler, D. A., V. F. Froelicher, N. H. Miller, and T. D. York. ACSM's guidelines for exercise testing and prescription. Baltimore, Williams and Wilkins. 1995, 278-279.

30. McLellan, T. M., G. A. Gannon, J. Zamecnik, V. Gil, and G. M. Brown. Low doses of melatonin and diurnal effects on thermoregulation and tolerance to uncompensable heat stress. *J.Appl.Physiol.* 87: 308-316, 1999.
31. Mills, P. J., N. C. Smith, I. Casas, P. Harris, R. C. Harris, and D. J. Marlin. Effects of exercise intensity and environmental stress on indices of oxidative stress and iron homeostasis during exercise in the horse. *Eur.J.Appl.Physiol.* 74: 60-66, 1996.
32. Montain, S. J., M. N. Sawka, B. S. Cadarette, M. D. Quigley, and J. M. McKay. Physiological tolerance to uncompensable heat stress: effects of exercise intensity, protective clothing, and climate. *J.Appl.Physiol.* 77: 216-222, 1994.
33. Nielsen, B., J. R. S. Hales, S. Strange, N. J. Christensen, J. Warberg, and B. Saltin. Human circulatory and thermoregulatory adaptations with heat acclimation and exercise in a hot, dry environment. *J.Physiol.* 460: 467-485, 1993.
34. Nielsen, B., G. Savard, E. A. Richter, M. Hargreaves, and B. Saltin. Muscle blood flow and muscle metabolism during exercise and heat stress. *J.Appl.Physiol.* 69: 1040-1046, 1990.
35. Nielsen, B., S. Strange, N. J. Christensen, J. Warberg, and B. Saltin. Acute and adaptive responses in humans to exercise in a warm, humid environment. *Pflugers Arch.* 434: 49-56, 1997.
36. Nybo, L., T. Jensen, B. Nielsen, and J. Gonzalez-Alonso. Effects of marked hyperthermia with and without dehydration on VO₂ kinetics during intense exercise. *J.Appl.Physiol.* 90: 1057-1064, 2001.
37. Pirnay, F., R. Deroanne, and J. M. Petit. Maximal oxygen consumption in a hot environment. *J.Appl.Physiol.* 218: 642-645, 1970.
38. Rowell, L. B. Human circulation: regulation during physical stress. New York, Oxford University Press. 1986, 363-402.

39. Rowell, L. B., J. R. Blackmon, R. H. Martin, J. A. Mazzaella, and R. A. Bruce. Hepatic clearance of indocyanine green in man under thermal and exercise stresses. *J.Appl.Physiol.* 20: 384-394, 1965.
40. Rowell, L. B., G. L. Brengelmann, J. A. Murray, K. K. Kraning II, and F. Kusumi. Human metabolic responses to hyperthermia during mild to maximal exercise. *J.Appl.Physiol.* 26: 395-402, 1969.
41. Rowell, L. B., H. J. Marx, R. A. Bruce, R. D. Conn, and F. Kusumi. Reductions in cardiac output, central blood volume, and stroke volume with thermal stress in normal men during exercise. *J.Clin.Invest.* 45: 1801-1816, 1966.
42. Sakate, T. The effect of air temperature on physical working capacity. *J.Hum.Ergology (Tokyo)* 7: 127-134, 1978.
43. Saltin, B., A. P. Gagge, U. Bergh, and J. A. J. Stolwijk. Body temperatures and sweating during exhaustive exercise. *J.Appl.Physiol.* 32: 635-643, 1972.
44. Savard, G., B. Nielsen, J. Laszczynska, B. E. Larsen, and B. Saltin. Muscle blood flow is not reduced in humans during moderate exercise and heat stress. *J.Appl.Physiol.* 64: 649-657, 1988.
45. Sawka, M. N., W. A. Latzka, S. J. Montain, B. S. Cadarette, M. A. Kolka, K. K. Kraning II, and R. R. Gonzalez. Physiologic tolerance to uncompensable heat: Intermittent exercise, field vs laboratory. *Med.Sci.Sports Exerc.* 33: 422-430, 2001.
46. Sawka, M. N., A. J. Young, B. S. Cadarette, L. Levine, and K. B. Pandolf. Influence of heat stress and acclimation on maximal aerobic power. *Eur.J.Appl.Physiol.* 53: 294-298, 1985.
47. Sawka, M. N., A. J. Young, W. A. Latzka, P. D. Neufer, M. D. Quigley, and KB. Pandolf. Human tolerance to heat strain during exercise: influence of hydration. *J.Appl.Physiol.* 73: 368-375, 1992.

48. Sen Gupta, J., G. P. Dimri, and M. S. Malhotra. Metabolic responses of Indians during sub-maximal and maximal work in dry and humid heat. *Ergonomics* 20: 33-40, 1977.
49. Shvartz, E. and D. Benor. Heat strain in hot and humid environments. *Aerosp.Med.* 43: 852-855, 1972.
50. Taylor, C. R. and C. P. Lyman. Heat storage in running antelopes: Independence of brain and body temperatures. *Am.J.Physiol.* 222: 114-117, 1972.
51. Taylor, C. R. and V. J. Rowntree. Temperature regulation and heat balance in running cheetahs: A strategy for sprinters? *Am.J.Physiol.* 224: 848-851, 1973.
52. Taylor, H. L., E. Buskirk, and A. Henschel. Maximal oxygen uptake as an objective measure of cardiorespiratory performance. *J.Appl.Physiol.* 8: 73-80, 1955.
53. Tenaglia, S. A., T. M. McLellan, and P. P. Klentrou. Influence of menstrual cycle and oral contraceptives on tolerance to uncompensable heat stress. *Eur.J.Appl.Physiol.* 80: 76-83, 1999.
54. Walters, T. J., K. L. Ryan, L. M. Tate, and P. A. Mason. Exercise in the heat is limited by critical internal temperature. *J.Appl.Physiol.* 89: 799-806, 2000.
55. Williams, C. G., A. G. Bredell, C. H. Wyndham, N. B. Strydom, J. F. Morrison, J. Peter, P. W. Fleming, and J. S. Ward. Circulatory and metabolic reactions to work in the heat. *J.Appl.Physiol.* 17: 625-638, 1962.
56. Young, D. R., R. Mosher, P. Erve, and H. Spector. Body temperature and heat exchange during treadmill running in dogs. *J.Appl.Physiol.* 14: 839-843, 1959.

CHAPTER 4
ELEVATION IN HEART RATE DURING SUBMAXIMAL EXERCISE IN
THE HEAT: RELATION TO RELATIVE METABOLIC INTENSITY¹

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Abstract

Heart rate is elevated during submaximal exercise intensity in the heat compared to a thermoneutral environment, but maximal oxygen uptake ($\dot{V}O_{2\max}$) may not be reduced in the heat. Therefore, there appears to be a dissociation between relative metabolic intensity ($\% \dot{V}O_{2\max}$) and heart rate in the heat. We tested the hypothesis that elevation in heart rate during submaximal exercise is related to increased $\% \dot{V}O_{2\max}$ due to reduced $\dot{V}O_{2\max}$ in the heat. $\dot{V}O_{2\max}$, and $\dot{V}O_2$ and heart rate during submaximal exercise were measured in 22 male and female runners under 4 environmental conditions designed to manipulate heart rate during submaximal exercise and $\dot{V}O_{2\max}$. The conditions involved walking for 20-min at $\sim 33\%$ control $\dot{V}O_{2\max}$ in 25, 35, 40, and 45°C, followed immediately by measurement of $\dot{V}O_{2\max}$. $\dot{V}O_{2\max}$ was reduced 4-17% in the heat. Heart rate at the end of the submaximal exercise increased progressively with increasing environmental temperature (107 ± 10 , 112 ± 10 , 120 ± 10 , and 137 ± 11 bpm, respectively). Heart rate and $\% \dot{V}O_{2\max}$ based on the $\dot{V}O_{2\max}$ measured under the conditions of the study increased in an identical fashion in the heat, and the elevations in heart rate were significantly related to the reductions in $\dot{V}O_{2\max}$ ($r = 0.79$). We conclude that elevation in heart rate during submaximal exercise in the heat is not indicative of dissociation between heart rate and relative metabolic intensity. Rather, the elevation in heart rate during submaximal exercise in the heat is proportional to reduced $\dot{V}O_{2\max}$. Exercise in the heat at the same heart rate as in a thermoneutral environment elicits similar relative metabolic intensity.

INDEX WORDS: Heart rate, Relative metabolic intensity, Maximal oxygen uptake, Heat stress, Treadmill exercise, Submaximal exercise.

Introduction

Heart rate increases linearly as a function of exercise intensity in thermoneutral environment and is closely related to the percentage of maximal oxygen uptake ($\% \dot{V}O_{2\max}$) elicited (1,14). During exercise in the heat, heart rate is elevated at submaximal exercise intensities (6,15,17,18,23,28), but most studies have reported that maximal oxygen uptake ($\dot{V}O_{2\max}$) is unchanged (15,18,21,28) or reduced only slightly (6,10,13,17,20-23,25). These findings suggest there is dissociation of heart rate and $\% \dot{V}O_{2\max}$ in the heat. Elevated heart rate has been explained by increased sympathetic activity (9,16), increased firing rate of the sinoatrial node (8), feedback from skin or core temperatures (5,26), and increased circulatory strain due to lowered stroke volume and higher skin blood flow for thermoregulation (14). However, the dissociation between heart rate during submaximal exercise and $\% \dot{V}O_{2\max}$ in the heat has not been verified by measuring both variables in cool and hot environments.

Two studies (12,13) have reported marked (16-25%) reductions in $\dot{V}O_{2\max}$ in the heat. These reductions were found following mild exercise in the heat that elevated core temperature (12,13). If $\dot{V}O_{2\max}$ is reduced following sustained exercise in the heat, then the oxygen uptake ($\dot{V}O_2$) at submaximal intensities would represent a higher $\% \dot{V}O_{2\max}$ and the relation of heart rate to $\% \dot{V}O_{2\max}$ might not be different from that in cool environment.

Therefore, the aim of this study was to determine whether the elevation in heart rate resulting from sustained submaximal exercise in the heat is related to reduced $\dot{V}O_{2\max}$. We hypothesized that $\dot{V}O_{2\max}$ would be reduced following sustained exercise in the heat, increasing the relative metabolic intensity during submaximal exercise at a constant work rate, and that higher relative metabolic intensity would, in turn, be associated with the elevation in heart rate during sustained submaximal exercise in the heat.

Methods

Subjects: Twenty-two healthy, endurance-trained male ($n=11$, age 23.1 ± 4.7 yr, height 178.2 ± 4.4 cm, mass 70.1 ± 8.8 kg, $\dot{V}O_{2\max}$ 64.7 ± 5.3 ml/kg/min) and female ($n=11$, age 23.8 ± 3.8 yr, height 164.8 ± 5.7 cm, mass 56.0 ± 4.9 kg, $\dot{V}O_{2\max}$ 53.9 ± 7.5 ml/kg/min) runners and triathletes served as subjects. The men and women had run 72.4 ± 42.6 and 59.5 ± 39.5 km/week, respectively, for at least 6 wk and were accustomed to exercising in a hot environment. All subjects were tested at the same time of the day to minimize the effects of circadian rhythm on heart rate, and a minimum of 2 days passed between testing of the same subject. Participation was voluntary and subjects were paid upon completion of the study. The study was approved by the University's Institutional Review Board and a written consent was obtained before testing.

Experimental Design: A repeated-measures experimental design in which subjects served as their own control was used. The dependent variables were % $\dot{V}O_{2\max}$, and heart rate at the end of submaximal exercise. The independent variable was environmental temperature. Other metabolic, cardiorespiratory, and temperature measures were assessed to help interpret the effects of manipulations of the independent variables on the dependent variables.

Treatments: The study was conducted in an environmental chamber at 50% relative humidity (RH) under the following four conditions in which environmental and pretest core temperature were varied: 1) 25°C with a 20-min walking warm-up at ~33% of control $\dot{V}O_{2\max}$ (25W); 2) 35°C with a 20-min walking warm-up at ~33% of control $\dot{V}O_{2\max}$ (35W); 3) 40°C with a 20-min walking warm-up at ~33% of control $\dot{V}O_{2\max}$ (40W); 4) 45°C with a 20-min walking warm-up at ~33% of control $\dot{V}O_{2\max}$ (45W). A control $\dot{V}O_{2\max}$ test was conducted prior to the treatments, which then were conducted in a random order. The conditions were designed to elevate the core temperature to different degrees using active preheating prior to the $\dot{V}O_{2\max}$ test, and to alter the rate of rise in core temperature during the test. The conditions reflect the

effects of increased core temperature and circulatory strain associated with prolonged submaximal exercise in different ambient temperatures. Because there were no significant differences in measures at maximal exercise, reproducibility was assessed using data from the control and 25W tests.

Test Protocol: Subjects reported to the laboratory following a 3-h fast, but well hydrated. They were instructed not to consume alcohol or drugs 48 h prior to testing, not to consume caffeine 12 h prior to testing, and to drink water and other non-caffeinated beverages liberally. On the morning of the test, subjects filled out a 24-h history questionnaire designed to determine adherence to pretest instructions. Then, skinfold thickness measures were taken for estimation of body fat (only done in the control test) and subjects measured their nude body weight. Next, subjects inserted rectal and esophageal thermistors for measurement of core temperature, thermistors for measurement of skin temperature were attached, and a strap containing the electrodes and transmitter for a heart rate monitor was placed around the chest. The subjects ingested water to compensate for the estimated sweat loss that would occur.

The subjects then completed a 20-min walk at ~33% of control $\dot{V}O_{2\max}$ followed by a graded running test to exhaustion. During the exercise, $\dot{V}O_2$ and other metabolic variables of interest; heart rate; and rectal, esophageal, and skin temperatures were monitored. Metabolic, cardiorespiratory, and temperature measures were recorded every 5 min during the 20-min walk and every 2 min during the graded running test.

Three min following the test, a fingerstick blood sample was obtained from the subject and analyzed for lactate. Then subjects dried off and measured their nude body weight to determine the amount of weight loss (dehydration).

Test Procedures: To elicit $\dot{V}O_{2\max}$, subjects ran to exhaustion at a constant speed with the grade of the treadmill increasing 2% every 2 min. A speed was chosen to exhaust subjects in 6-15 min of exercise. During the test, a Sensormedics Vmax 29 metabolic cart was used to measure the metabolic variables over a sampling period of

30 s. Four consecutive 30-s values were averaged for all metabolic measures during submaximal exercise, and 2 consecutive 30-s values were averaged during the $\dot{V}O_{2\max}$ test. The metabolic cart was calibrated using standard gases with concentrations verified to 0.02%.

Attainment of $\dot{V}O_{2\max}$ in the control condition was assured by using a modification of the plateauing criterion of Taylor et al. (25). The criterion for determining a plateau was an increase in $\dot{V}O_2$ (ml/kg/min) between the last two stages of less than 50% of the expected increase, based on the American College of Sports Medicine metabolic equation (11). The criterion varied depending on treadmill speed and ranged from 1.3 ml/kg/min (5.5 mph) to 2.2 ml/kg/min (9 mph). After completion of the graded test, all subjects (regardless of plateau attainment during the graded test) rested for 20 min and then ran until exhaustion at a grade 2% higher than the grade at the end of the graded test. This process was repeated until a plateau was obtained. Using this protocol, all subjects demonstrated plateau in $\dot{V}O_2$ either during the graded exercise test and/or after the first rest period in the control test.

Because we hypothesized that a plateau in $\dot{V}O_2$ might not be evidenced in the heat if performance was limited by hyperthermia, and because it was not possible to do follow-up tests in the heat due to the threat of heat injury, the average of the two highest consecutive 30-s values, or the peak oxygen consumption, was used in the other conditions if heart rate was within 5 bpm of the control condition, or if the increase in $\dot{V}O_2$ (ml/kg/min) met the plateauing criterion tested for in the control condition. If this heart rate/ $\dot{V}O_2$ criterion was not met, the test was repeated on another day. Five tests in males were repeated because of inability to meet this criterion and 2 tests in males and 1 test in females were repeated because of equipment failure. The standard deviations of replicate measures in 25°C in 22 subjects for $\dot{V}O_{2\max}$ (l/min), $\dot{V}O_{2\max}$ (ml/kg/min), and RER at exhaustion were 1.36, 0.085, and 0.02, respectively.

Rectal temperature was measured with an YSI thermister (model 4491E) inserted 12 cm beyond the anal sphincter. Esophageal temperature was measured using an YSI thermister (model 4491E) inserted through the nasal cavity and into the esophagus a distance equal to one-fourth of the standing height. Mean skin temperature was calculated according to the formula of Burton (3) from measurements of skin temperature with YSI thermisters (model 409B), on the forearm, beneath the scapula, and on the thigh. All thermisters were connected to a YSI telethermometer (model 44TD or 4600). The accuracy of all thermisters was verified by a mercury thermometer prior to use. The standard deviations of replicate measures in 25^o C in 22 subjects for rectal temperature, esophageal temperature, and mean skin temperature at exhaustion were 0.25, 0.17, and 0.44^o C, respectively.

Heart rate was measured using a Polar Vantage XL Heart Rate Monitor (model 145900). The standard deviation of replicate measures in 25^o C in 22 subjects for heart rate at exhaustion was 2.6 bpm. Rating of perceived exertion (RPE) was measured by having the subjects holding up the appropriate number of fingers corresponding to Borg 15-point category scale (2). The standard deviation of replicate measures in 25^o C in 22 subjects for RPE at exhaustion was 0.7. Blood lactate was measured with a YSI 2300 STAT plus lactate/glucose analyzer. The standard deviation of replicate measures in 25^o C in 22 subjects for blood lactate was 1.05 mmol/l. Finally, body weight was measured with an electronic scale (A&D Co. Ltd., model FW-150KA1). The standard deviations of replicate measures in 25^o C in 22 subjects for nude body weight and weight loss were 0.66 and 0.18 kg, respectively.

Statistical Analysis: Statistical analyzes were done with SPSS 10 for Windows (SPSS Inc. Chicago, IL). Data are reported as means and SD of the measures. A one-way repeated-measures ANOVA was used to determine the significance of differences among the measures under the different environmental conditions for the metabolic, cardiorespiratory, skin temperature, and RPE measures. A one-way repeated-measures

ANCOVA, with the resting temperature held constant, was used to determine the significance of differences among the measures under the different environmental conditions for the core temperature measures. Planned contrasts (paired samples *t*-test) were used to determine differences between conditions. Simple linear regression and correlation was used to examine the relationship between change in heart rate during submaximal exercise and decrease in $\dot{V}O_{2\max}$ in the heat, and between heart rate at the end of submaximal exercise and % $\dot{V}O_{2\max}$.

A two-tailed α level of 0.05 was used for all significance tests except for $\dot{V}O_{2\max}$. A one-tailed α level of 0.05 was used for $\dot{V}O_{2\max}$ because it was expected to be unchanged or decreased in the heat. The significance level was adjusted using the modified Bonferonni adjustment for the family of contrasts performed.

Results

Data on the metabolic, cardiorespiratory, and perceptual measures are contained in Table 4.1. $\dot{V}O_{2\max}$ was significantly lower in the heat (Table 4.1) compared to the neutral environment by 4% at 35W, 9% at 40W, and 17% at 45W. The reduction was not due to lack of effort in the heat, because indicators of maximal effort suggested that a maximal effort was given under all conditions. Maximal heart rate was within 5 bpm in all conditions, RER was always 1.1 or higher, and RPE was approximately 19 in all conditions. Blood lactate was lower in 45W compared to the other conditions, but that more likely reflected reduced running time and lower grade at exhaustion than lack of effort. Dehydration also was an unlikely contributor to the reductions in $\dot{V}O_{2\max}$ since weight loss was less than 0.7% of body weight.

Table 4.1: Metabolic and performance variables at exhaustion

Measures	25C	25W	35W	40W	45W
$\dot{V}O_{2\max}$ (l/min)	3.76±0.89	3.77±0.89	3.61±0.84*	3.44±0.82*	3.13±0.76*
HR (bpm)	190±10	188±8	188±8	190±10*	192±10*
RER	1.17±0.03	1.16±0.03	1.14±0.04*	1.13±0.04*	1.10±0.05*
Lactate (mmol/l)	8.6±1.5	8.4±2.5	7.5±1.7	7.5±1.9	5.7±1.8*
RPE	19.3±0.8	19.1±0.9	19.0±1.3	19.4±0.9	19.1±0.9

$\dot{V}O_{2\max}$ = maximal oxygen uptake, HR = heart rate, RPE = ratings of perceived exertion, 25C = Control test in 25° C, 25W = 25° C, 35W = 35° C, 40W = 40° C, 45W = 45° C. * = $p < 0.05$ from 25W.

The metabolic, cardiorespiratory, and temperature data after 20-min of submaximal exercise are presented in Table 4.2. At the end of submaximal exercise, $\dot{V}O_2$ was slightly lower in the heat (35W, 40W, 45W) than in the thermoneutral environment (25W), but the differences among the hot conditions in $\dot{V}O_2$ were not significant. Heart rate increased progressively in curvilinear fashion with increasing ambient temperature (Figure 4.1). Oxygen uptake as a percentage of the control-test $\dot{V}O_{2\max}$ (% $\dot{V}O_{2\max\text{-ctrl}}$) was slightly lower in the heat (Figure 4.1), reflecting the reduced submaximal $\dot{V}O_2$ in the heat. However, when $\dot{V}O_2$ was expressed as a percentage of the $\dot{V}O_{2\max}$ measured in each of the conditions (% $\dot{V}O_{2\max\text{-msrd}}$), the relative metabolic intensity during submaximal exercise increased in a curvilinear fashion with increasing environmental temperature (Figure 4.1).

Table 4.2: Metabolic, cardiorespiratory, and temperature variables after 20 min of submaximal exercise

Measures	25W	35W	40W	45W
$\dot{V}O_2$ (l/min)	1.25±0.29	1.21±0.29*	1.22±0.28*	1.20±0.29*
HR (bpm)	107±10	112±10*	120±10*†	137±11*†
% $\dot{V}O_{2\max\text{-msrd}}$	33.4±2.0	33.7±2.4	35.7±2.0*†	38.7±3.5*†
% $\dot{V}O_{2\max\text{-ctrl}}$	33.4±2.0	32.3±2.1*	32.6±1.9*	32.0±2.2*
T _{ESO} (°C)	37.1±0.4	37.0±0.4	37.2±0.4*	37.8±0.2*†
T _{RECTAL} (°C)	37.3±0.3	37.3±0.3	37.3±0.3	37.6±0.3*†
T _{SKIN} (°C)	32.2±0.7	35.1±0.4*	36.1±0.4*†	37.6±0.2*†

$\dot{V}O_{2\max}$ = maximal oxygen uptake, HR = heart rate, % $\dot{V}O_{2\max\text{-msrd}}$ = percent of measured maximal oxygen uptake, % $\dot{V}O_{2\max\text{-ctrl}}$ = percent of control maximal oxygen uptake, T_{ESO} = esophageal temperature, T_{RECTAL} = rectal temperature, T_{SKIN} = skin temperature, 25W = 25°C, 35W = 35°C, 40W = 40°C, 45W = 45°C. * = $p < 0.05$ from 25W, † = $p < 0.05$ from next temperature condition below.

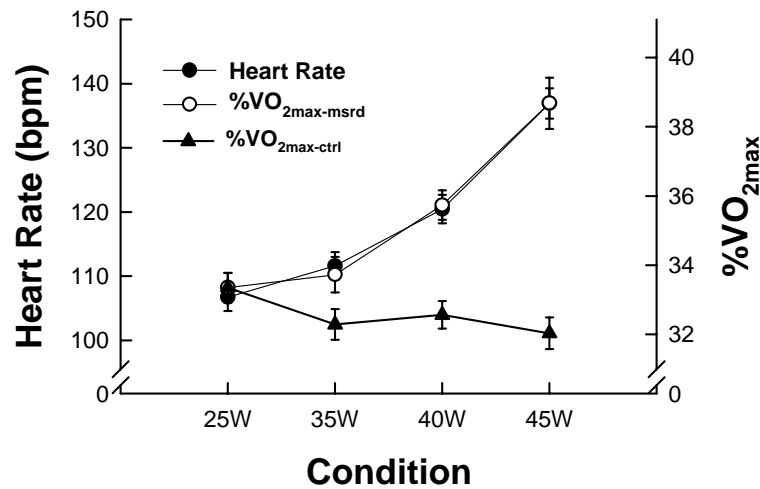


Figure 4.1: Changes in heart rate and % $\dot{V}O_{2\max}$ with increasing environmental temperature. % $\dot{V}O_{2\max\text{-msrd}}$ = percent of measured maximal oxygen uptake, % $\dot{V}O_{2\max\text{-ctrl}}$ = percent of control maximal oxygen uptake, 25W = 25°C, 35W = 35°C, 40W = 40°C, 45W = 45°C.

Skin temperature increased with increasing environmental temperature (Table 4.2). Core temperature, on the other hand, did not change much until 45W, during which it was higher than in other conditions (Table 4.2). That finding indicates that the

subjects were able to dissipate most of the heat produced during the low-intensity submaximal exercise in temperatures up to 40° C.

The change in heart rate at the end of the submaximal exercise in the heat from the heart rate in the thermoneutral condition (25W) correlated significantly with the reduction in $\dot{V}O_{2\max}$ in the heat ($r = 0.79$, Figure 4.2). Similarly, heart rate at the end of submaximal exercise correlated significantly with % $\dot{V}O_{2\max-\text{msrd}}$ ($r = 0.62$, Figure 4.3A). However, heart rate at the end of submaximal exercise was unrelated to % $\dot{V}O_{2\max-\text{ctrl}}$ ($r = 0.06$, Figure 4.3B). Figures 4.3A and 4.3B are identical, except that in the left hand panel, % $\dot{V}O_{2\max-\text{msrd}}$ is the on the X-axis, whereas % $\dot{V}O_{2\max-\text{ctrl}}$ is on the X-axis in the right hand panel. These figures reflect that once reduced exercise capacity in the heat has been taken into account, elevation in heart rate during submaximal exercise is related to relative metabolic intensity, whereas if $\dot{V}O_{2\max}$ is assumed to not be reduced in the heat, heart rate is elevated independent of relative metabolic intensity (no relation).

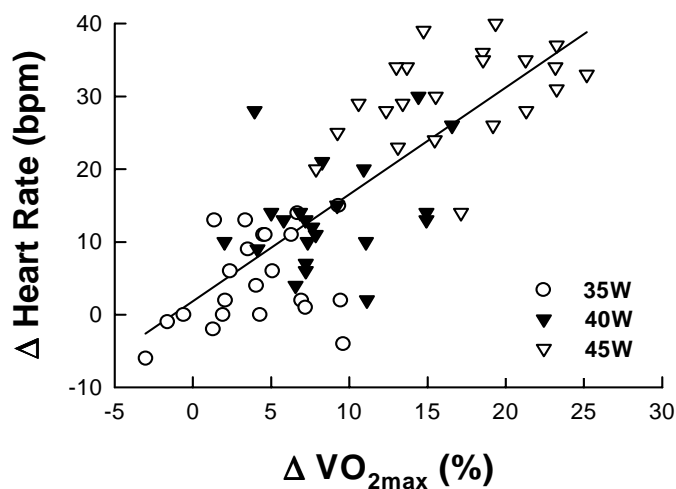


Figure 4.2: Relation between elevations in heart rate during submaximal exercise above the heart rate in 25°C and reductions in $\dot{V}O_{2\max}$ in the heat ($r = 0.79$, SEE 8 bpm). Δ Heart Rate = change in heart rate in the heat from 25°C, $\Delta \% \dot{V}O_{2\max}$ = reduction in maximal oxygen uptake in the heat expressed as a percentage of control maximal oxygen; 35W = 35°C, 40W = 40°C, 45W = 45°C.

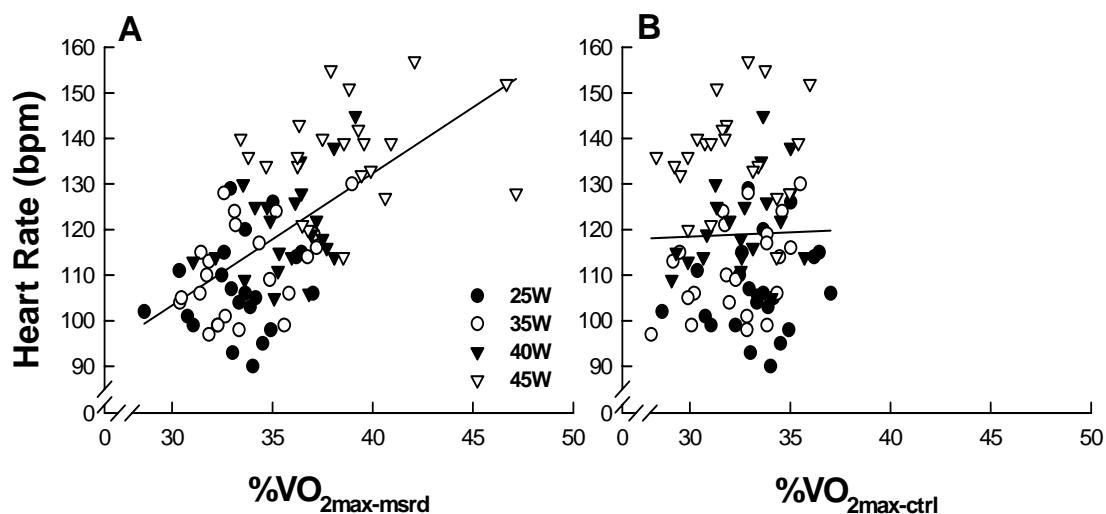


Figure 4.3: Relation of heart rate at the end of 20-min of submaximal exercise to $\% \dot{V}O_{2\max\text{-msrd}}$ (A, $r = 0.62$, SEE 12 bpm) and $\dot{V}O_{2\max\text{-ctrl}}$ (B, $r = 0.03$, SEE 16 bpm). $\% \dot{V}O_{2\max\text{-msrd}}$ = percent of measured maximal oxygen uptake, $\dot{V}O_{2\max\text{-ctrl}}$ = percent of control maximal oxygen uptake, 25W = 25°C, 35W = 35°C, 40W = 40°C, 45W = 45°C.

Discussion

The primary new finding of this study is that increased heart rate at the end of submaximal exercise was related to reduced $\dot{V}O_{2\max}$ and increased relative metabolic intensity following 20-min of low intensity exercise in the heat. As ambient temperature was progressively increased from 25° C to 45° C, the mean heart rate and the mean % $\dot{V}O_{2\max-\text{msrd}}$ following the 20 min of exercise increased in an identical fashion. These data suggest that during sustained exercise in the heat, heart rate accurately reflects the relative metabolic intensity and is not dissociated from $\dot{V}O_2$ as long believed.

Whether $\dot{V}O_{2\max}$ is reduced as a result of heat stress has been debated. Some studies report no change (15,18,21,28), whereas others report very small, often trivial, reductions on the order of 150-350 ml/min or 3-8% (6,10,13,17,20-23,25). Two studies (12,13) have found marked reductions (16-25% or 750-985 ml/min) in $\dot{V}O_{2\max}$ during heat stress, when $\dot{V}O_{2\max}$ was measured following 12-20 min of preliminary low-to-moderate intensity (30-50% of $\dot{V}O_{2\max}$) exercise that elevated the core temperature prior to the $\dot{V}O_{2\max}$ test. The findings of the present study support these latter findings and reconcile the discrepancy between the studies finding small and large reductions in $\dot{V}O_{2\max}$. When the ambient heat stress (35W, 40W) during low-intensity exercise is not severe enough to prevent the body from effectively thermoregulating, small reductions are found in $\dot{V}O_{2\max}$. However, when the heat stress (45W) becomes uncompensable, large reductions are observed in $\dot{V}O_{2\max}$, probably due to attainment of a critical core temperature that causes fatigue (Arngrímsson et al., manuscript in preparation) and/or circulatory strain (14).

Heart rate increases fairly linearly with increased exercise intensity in a thermoneutral environment (1,14). The relation of heart rate to relative metabolic intensity (% $\dot{V}O_{2\max}$) is stronger than to absolute metabolic intensity ($\dot{V}O_2$), probably because the stimuli that increase sympathetic nervous system activity, the primary

factor that increases heart rate during exercise (4), are most closely linked to relative exercise intensity (14,19). During heat stress, heart rate is higher at submaximal exercise intensities than in a thermoneutral environment (6,15,17,18,23,28). Studies reporting no reduction in $\dot{V}O_{2\max}$ in the heat despite increased heart rate during submaximal exercise at the same absolute intensity, suggested that there was dissociation in the relation between heart rate and $\% \dot{V}O_{2\max}$ (15,18,28). The elevated heart rate in the heat independent of altered $\dot{V}O_{2\max}$ has been explained by increased sympathetic activity (9,16), increased rate of sinoatrial node firing (8), feedback from increased core and skin temperature to the heart (5,26), or increased circulatory strain due to elevated skin blood flow for thermoregulation and reduced stroke volume from peripheral displacement of blood (14).

This study is the first to measure $\dot{V}O_{2\max}$, and $\dot{V}O_2$ and heart rate during submaximal exercise, following a period of sustained exercise, under progressively increasing ambient temperatures. We found that $\dot{V}O_{2\max}$ was reduced and the relative metabolic intensity ($\% \dot{V}O_{2\max}$) increased in the heat compared to a thermoneutral environment, and that the changes in the two were strongly related. There was a positive relation between heart rate and $\% \dot{V}O_{2\max}$ in different levels of heat stress if $\dot{V}O_{2\max}$ was measured after 20-min of submaximal exercise, but no relation if $\% \dot{V}O_{2\max}$ was calculated based on the control $\dot{V}O_{2\max}$. Our findings differ from most others primarily because of the conditions under which $\dot{V}O_{2\max}$ was measured. With two exceptions (12,13) that did not report heart rate during submaximal exercise, most other studies reporting the effect of heat on submaximal heart rate have not measured $\dot{V}O_{2\max}$ following sustained exercise in the heat that elevates core temperature (6,15,17,18,23). Instead $\dot{V}O_{2\max}$ was measured following a brief exposure to the heat (15,18). During brief periods of maximal exercise in the heat (no preheating prior to the $\dot{V}O_{2\max}$ test), $\dot{V}O_{2\max}$ may not be reduced because of skin vasoconstriction (14) and insufficient time

to raise the core temperature to critical levels (Arngrímsson et al., manuscript in preparation).

Our findings have practical implications for exercise prescription in the heat. The general advice regarding exercise in the heat has been to stay within the target heart rate zone because heart rate is sensitive to heat stress and provides an index of the overall physiological strain (7). Because heart rate at submaximal intensities is elevated in the heat, exercise intensity must be reduced to maintain the same heart rate as in a thermoneutral environment. Reduction of the exercise intensity lowers the absolute $\dot{V}O_2$ elicited. If $\dot{V}O_{2max}$ is assumed to be unchanged in the heat, then the calculated $\% \dot{V}O_{2max}$ also is lower when exercising at the same heart rate in the heat compared to a thermoneutral environment. The findings of this study suggest, on the other hand, that following sustained exercise in the heat, lowering the exercise intensity to maintain the same heart rate as in thermoneutral environment would elicit similar $\% \dot{V}O_{2max}$ as in a thermoneutral environment, because heart rate is increased in proportion to the reduction in $\dot{V}O_{2max}$.

Because relative metabolic intensity is thought to determine the training stimulus for increasing $\dot{V}O_{2max}$ (24,27), similar percentage improvements in $\dot{V}O_{2max}$ might be expected after training at a given $\% \dot{V}O_{2max}$ in the heat compared to a thermoneutral environment, despite the lower absolute $\dot{V}O_2$ elicited. However, the increase in $\dot{V}O_{2max}$ with training in warm (35° C) compared to cold (20° C) water at the same absolute $\dot{V}O_2$, but different (~25 bpm) heart rates, was the same (29,30), suggesting that the lower absolute $\dot{V}O_2$ elicited at the same $\% \dot{V}O_{2max}$ in the heat provides less of a stimulus for increasing $\dot{V}O_{2max}$ with training. Unfortunately, $\dot{V}O_{2max}$ was only assessed in air in a thermoneutral environment; $\dot{V}O_{2max}$ in the warm water may have been reduced. Additional studies are needed to determine whether the absolute or relative metabolic intensity is a more important stimulus for increasing $\dot{V}O_{2max}$ in a hot climate in which $\dot{V}O_{2max}$ is reduced.

We conclude that elevation in heart rate during submaximal exercise in the heat is not indicative of dissociation between heart rate and relative metabolic intensity. Rather, the elevation in heart rate during submaximal exercise in the heat is proportional to reduced $\dot{V}O_{2\max}$. Exercise in the heat at the same heart rate as in a thermoneutral environment elicits similar relative metabolic intensity.

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References

1. Astrand, P. O. and K. Rodahl. Textbook of work physiology. New York, McGraw-Hill. 1986, 372-378.
2. Borg, G. The perception of physical performance. In Shephard, R. J., ed., *Frontiers of Fitness*. Springfield, IL, Charles C Thomas Publisher. 1971, 280-294.
3. Burton, A. C. Human calorimetry II. The average temperature of the tissues of the body. *J.Nutr.* 9: 61-280, 1935.
4. Christensen, N. J. and O. Brandsborg. The relationship between plasma catecholamine concentration and pulse rate during exercise and standing. *Eur.J.Clin.Invest.* 3: 299-306, 1973.
5. Cooper, K. E. and D. M. Kerslake. Changes in heart rate during exposure of the skin to radiant heat. *Clin.Sci.* 14: 125-135, 1955.
6. Dimri, G. P., M. S. Malhotra, J. Sen Gupta, T. S. Kumar, and B. S. Aora. Alterations in aerobic-anaerobic metabolic proportions of metabolism during work in heat. *Eur.J.Appl.Physiol.* 45: 43-50, 1980.
7. Howley, E. T. and B. D. Franks. Health fitness instructor's handbook. Champaign, IL, Human Kinetics. 1997, 282.
8. Jose, A. D., F. Stitt, and D. Collison. The effects of exercise and changes in body temperature on the intrinsic heart rate in man. *Am.Heart J.* 79: 488-497, 1970.
9. Kim, Y. D., R. Lake, D. E. Lees, W. H. Schuette, J. M. Bull, V. Weise, and I. J. Kopin. Hemodynamic and plasma catecholamine responses to hyperthermic cancer therapy in humans. *Am.J.Physiol.* 237: H570-H574, 1979.
10. Klausen, K., D. B. Dill, E. E. Jr. Phillips, and D. McGregor. Metabolic reactions to work in the desert. *J.Appl.Physiol.* 22: 292-296, 1967.
11. Mahler, D. A., V. F. Froelicher, N. H. Miller, and T. D. York. ACSM's guidelines for exercise testing and prescription. Baltimore, Williams and Wilkins. 1995, 278-279.

12. Nybo, L., T. Jensen, B. Nielsen, and J. Gonzalez-Alonso. Effects of marked hyperthermia with and without dehydration on VO₂ kinetics during intense exercise. *J.Appl.Physiol.* 90: 1057-1064, 2001.
13. Pirnay, F., R. Deroanne, and J. M. Petit. Maximal oxygen consumption in a hot environment. *J.Appl.Physiol.* 218: 642-645, 1970.
14. Rowell, L. B. Human circulation: regulation during physical stress. New York, Oxford University Press. 1986, 213-402.
15. Rowell, L. B., J. R. Blackmon, R. H. Martin, J. A. Mazzarella, and R. A. Bruce. Hepatic clearance of indocyanine green in man under thermal and exercise stresses. *J.Appl.Physiol.* 20: 384-394, 1965.
16. Rowell, L. B., G. L. Brengelmann, and P. R. Freund. Unaltered norepinephrine-heart rate relationship in exercise with exogenous heat. *J.Appl.Physiol.* 62: 646-650, 1987.
17. Rowell, L. B., G. L. Brengelmann, J. A. Murray, K. K. Kraning II, and F. Kusumi. Human metabolic responses to hyperthermia during mild to maximal exercise. *J.Appl.Physiol.* 26: 395-402, 1969.
18. Rowell, L. B., H. J. Marx, R. A. Bruce, R. D. Conn, and F. Kusumi. Reductions in cardiac output, central blood volume, and stroke volume with thermal stress in normal men during exercise. *J.Clin.Invest.* 45: 1801-1816, 1966.
19. Saito, M., M. Tsukanaka, D. Yanagihara, and T. Mano. Muscle sympathetic nerve responses to graded leg cycling. *J.Appl.Physiol.* 75: 663-667, 1993.
20. Sakate, T. The effect of air temperature on physical working capacity. *J.Hum.Ergology (Tokyo)* 7: 127-134, 1978.
21. Saltin, B., A. P. Gagge, U. Bergh, and J. A. J. Stolwijk. Body temperatures and sweating during exhaustive exercise. *J.Appl.Physiol.* 32: 635-643, 1972.

22. Sawka, M. N., A. J. Young, B. S. Cadarette, L. Levine, and K. B. Pandolf. Influence of heat stress and acclimation on maximal aerobic power. *Eur.J.Appl.Physiol.* 53: 294-298, 1985.
23. Sen Gupta, J., G. P. Dimri, and M. S. Malhotra. Metabolic responses of Indians during sub-maximal and maximal work in dry and humid heat. *Ergonomics* 20: 33-40, 1977.
24. Shephard, R. J. Intensity, duration and frequency of exercise as determinants of the response to a training regime. *Int.Z.Angew.Physiol.Einschl.Arbeitsphysiol.* 26: 272-278, 1968.
25. Taylor, H. L., E. Buskirk, and A. Henschel. Maximal oxygen uptake as an objective measure of cardiorespiratory performance. *J.Appl.Physiol.* 8: 73-80, 1955.
26. Thauer, R. Circulatory adjustments to climatic requirements. In Hamilton, W. F. and P. Dow, eds., *Handbook of Physiology. Section : Circulation.* Washington, D.C., American Physiological Society. 1965, 1921-1966.
27. Wenger, H. A. and G. J. Bell. The interactions of intensity, frequency and duration of exercise training in altering cardiorespiratory fitness. *Sports Med.* 3: 346-356, 1986.
28. Williams, C. G., A. G. Bredell, C. H. Wyndham, N. B. Strydom, J. F. Morrison, J. Peter, P. W. Fleming, and J. S. Ward. Circulatory and metabolic reactions to work in the heat. *J.Appl.Physiol.* 17: 625-638, 1962.
29. Young, A. J., M. N. Sawka, L. Levine, P. W. Burgoon, W. A. Latzka, R. R. Gonzalez, and K. B. Pandolf. Metabolic and thermal adaptations from endurance training in hot or cold water. *J.Appl.Physiol.* 78: 793-801, 1995.
30. Young, A. J., M. N. Sawka, M. D. Quigley, B. S. Cadarette, P. D. Neufer, R. C. Dennis, and C. R. Valeri. Role of thermal factors on aerobic capacity improvements with endurance training. *J.Appl.Physiol.* 75: 49-54, 1993.

CHAPTER 5

SUMMARY AND CONCLUSIONS

A hot compared to cool environment compromises physical work capacity and endurance performance. On the other hand, studies have suggested that maximal oxygen uptake ($\dot{V}O_{2\max}$) is unchanged in the heat or reduced only slightly. If $\dot{V}O_{2\max}$ is not reduced in the heat but performance is decreased, it suggests that the two are unrelated in the heat and some factor other than reduced $\dot{V}O_{2\max}$ is the cause of decreased performance. Heart rate also is elevated at submaximal intensities during exercise in the heat. Elevated heart rate at submaximal intensities without reduction in the $\dot{V}O_{2\max}$ suggests that there is dissociation of heart rate and relative metabolic intensity ($\% \dot{V}O_{2\max}$) in the heat.

The reduction in work capacity and performance has commonly been explained by a hypothesized reduction in muscle blood flow due to peripheral displacement of blood to the skin. Such displacement, along with increased sympathetic activity and increased firing of the sinoatrial node in the heat, and feedback from skin or core temperature, would also elevate heart rate at submaximal intensities in the heat. Recently however, it has been demonstrated that muscle blood flow is not reduced in the heat during exhaustive submaximal exercise, and data suggest that high core temperature may limit exercise performance in the heat; exhaustion at similar core temperature during exercise to exhaustion in the heat has been found in numerous studies, which have almost exclusively studied men.

Only two studies have reported large decrease in $\dot{V}O_{2\max}$ in the heat. Both of those studies measured $\dot{V}O_{2\max}$ following a 12-20 min period of light-to-moderate exercise in the heat that elevated core temperature. The findings of these studies

suggest that following prolonged, submaximal exercise in the heat, $\dot{V}O_{2\max}$ may be reduced and the reduction may be due to attainment of critically high core temperature. If $\dot{V}O_{2\max}$ is reduced following submaximal exercise in the heat, the reduction in performance may be related to the reduction in $\dot{V}O_{2\max}$. Reduced $\dot{V}O_{2\max}$ in the heat also would increase the % $\dot{V}O_{2\max}$ during submaximal exercise, which might explain a part of the elevation in heart rate during submaximal exercise in the heat. Therefore, the relation of heart rate to % $\dot{V}O_{2\max}$ in the heat might not be different from that in thermoneutral environment.

The main purpose of this study was to determine whether $\dot{V}O_{2\max}$ during exercise in the heat is limited by attainment of a critical core temperature in endurance-trained men and women. A second purpose was to determine whether the elevation in heart rate resulting from sustained submaximal exercise in the heat is related to reduced $\dot{V}O_{2\max}$.

$\dot{V}O_{2\max}$ and esophageal temperature at exhaustion (T_{ESOmax}) were measured in 11 men and 11 women under environmental conditions ranging from 25°C to 45°C following periods of active, passive, or no preheating. Furthermore, measures of oxygen uptake and heart rate were obtained following a 20-min period of active (walking) preheating. The conditions were designed to manipulate core temperature at the beginning of, and the rate of rise during, the $\dot{V}O_{2\max}$ test, thereby manipulating the time to fatigue.

$\dot{V}O_{2\max}$ was moderately reduced (3-9%) in the heat under conditions in which critical core temperature was not attained, but larger reductions (17-19%) were observed in conditions which the critical core temperature was reached. However, there were considerable individual differences in the environmental conditions in which critical core temperature was first observed. The reductions in performance time were strongly related to reductions in $\dot{V}O_{2\max}$ in men and women ($r = 0.82-0.84$). Responses of men and women in T_{ESOmax} , $\dot{V}O_{2\max}$, and work capacity in the heat were almost

identical. $\% \dot{V}O_{2\max}$ and heart during submaximal exercise in the heat increased in an identical curvilinear fashion with increasing environmental temperature. Elevation in heart rate at the end of submaximal exercise in the heat correlated significantly with the reduction in $\dot{V}O_{2\max}$ in the heat ($r = 0.79$), as did heart at the end of submaximal exercise and $\% \dot{V}O_{2\max}$ ($r = 0.62$).

Based on the results from this study, it is concluded that:

1) Large ($> 10\%$) reductions in $\dot{V}O_{2\max}$ and work capacity in men and women in severe, uncompensable heat stress are associated with attainment of a critical core temperature. Without such attainment, moderate decreases due to circulatory strain are observed.

2) Reduced physical work capacity is strongly related to reduced $\dot{V}O_{2\max}$ in the heat. That relation appears to depend, in part, on the effect of attainment of a critical core temperature on both measures because it causes concomitant decrease in both variables.

3) Men and women do not differ in their T_{ESOMax} responses, or in the relation of the core temperature to the reductions in $\dot{V}O_{2\max}$ and work capacity in the heat. Therefore, men and women appear to be subject to the same limitations in $\dot{V}O_{2\max}$ and performance during exercise in the heat.

4) The elevation in heart rate during sustained submaximal exercise in the heat is not indicative of dissociation between heart rate and $\% \dot{V}O_{2\max}$. Rather the elevation in heart rate during submaximal exercise in the heat is proportional to reduced $\dot{V}O_{2\max}$. Therefore, exercise in the heat at the same heart rate as in a thermoneutral environment elicits similar $\% \dot{V}O_{2\max}$.

CHAPTER 6

LITERATURE CITED

1. Adams, W. C., R. H. Fox, A. J. Fry, and I. C. MacDonald. Thermoregulation during marathon running in cool, moderate, and hot environments. *J.Appl.Physiol.* 38: 1030-1037, 1975.
2. Andersen, P. and B. Saltin. Maximal perfusion of skeletal muscle in man. *J.Physiol.* 366: 233-249, 1985.
3. Aoyagi, Y., T. M. McLellan, and R. J. Shephard. Effects of endurance training and heat acclimation on psychological strain in exercising men wearing protective clothing. *Ergonomics* 41: 328-357, 1998.
4. Armstrong, R. B., M. D. Delp, E. F. Goljan, and M. H. Laughlin. Distribution of blood flow in muscles of miniature swine during exercise. *J.Appl.Physiol.* 62: 1285-1289, 1987.
5. Armstrong, R. B., M. D. Delp, E. F. Goljan, and M. H. Laughlin. Progressive elevations in muscle blood flow during prolonged exercise in swine. *J.Appl.Physiol.* 63: 285-291, 1987.
6. Asmussen, E. The cardiac output in rest and work in humid heat. *Am.J.Physiol.* 131: 54-59, 1940.
7. Asmussen, E., F. Bonde-Petersen, and K. Jorgensen. Mechano-elastic properties of human muscles at different temperatures. *Acta Physiol.Scand.* 96: 83-93, 1976.
8. Asmussen, E. and I. Hemmingsen. Determination of maximum working capacity at different ages in work with the legs or with the arms. *Scand.J.Clin.Lab.Invest.* 10: 67-71, 1958.

9. Astrand, P.-O. and B. Saltin. Maximal oxygen uptake and heart rate in various types of muscular activity. *J.Appl.Physiol.* 16: 977-981, 1961.
10. Astrand, P. O. and K. Rodahl. Textbook of work physiology. New York, McGraw-Hill. 1986, 229-378.
11. Ball, D., C. Burrows, and A. J. Sargeant. Human power output during repeated sprint cycle exercise: the influence of thermal stress. *Eur.J.Appl.Physiol.* 79: 360-366, 1999.
12. Barger, A. C., W. F. Greenwood, J. R. DiPalma, J. I. Stokes, and L. H. Smith. Venous pressure and cutaneous reactive hyperemia in exhausting exercise and certain other circulatory stresses. *J.Appl.Physiol.* 2: 81-96, 1949.
13. Bender, P. R. and B. J. Martin. Maximal ventilation after exhaustive exercise. *Med.Sci.Sports Exerc.* 17: 164-167, 1985.
14. Bergh, U. and B. Ekblom. Influence of muscle temperature on maximal muscle strength and power output in human skeletal muscles. *Acta Physiol.Scand.* 107: 33-37, 1979.
15. Bergh, U. and B. Ekblom. Physical performance and peak aerobic power at different body temperatures. *J.Appl.Physiol.* 46: 885-889, 1979.
16. Binkhorst, R. A., L. Hoofd, and A. C. A. Vissers. Temperature and force-velocity relationship of human muscles. *J.Appl.Physiol.* 42: 471-475, 1977.
17. Binkhorst, R. A. and P. Van Leeuwen. A rapid method for determination of aerobic capacity. *Int.Z.Angew.Physiol.Einschl.Arbeitsphysiol.* 19: 459-467, 1963.
18. Bobbert, A. C. Physiological comparison of three types of ergometer. *J.Appl.Physiol.* 15: 1007-1014, 1960.
19. Booth, J., F. Marino, and J. J. Ward. Improved running performance in hot humid conditions following whole body precooling. *Med.Sci.Sports Exerc.* 29: 943-949, 1997.

20. Brengelmann, G. L., J. M. Johnson, L. Hermansen, and L. B. Rowell. Altered control of skin blood flow during exercise at high internal temperature. *J.Appl.Physiol.* 43: 790-794, 1977.
21. Brooks, G. A., K. J. Hittelman, J. A. Faulkner, and R. E. Beyer. Temperature, skeletal muscle mitochondrial functions, and oxygen debt. *Am.J.Physiol.* 220: 1053-1059, 1971.
22. Brouha, L., P. E. Smith, Jr., R. De Lanne, and M. E. Maxfield. Physiological reactions of men and women during muscular activity and recovery in various environments. *J.Appl.Physiol.* 16: 133-140, 1960.
23. Brown, N. J., L. A. Stephenson, G. Lister, and E. R. Nadel. Relative anaerobiosis during heavy exercise in the heat. *Fed.Proc.* 41: 1677, 1982.
24. Bruck, K., E. Bahner, B. Krannig, and G. Neumann. Exercise performance and adaptive modifications in the thermoregulatory system. *Int.J.Biometeorol.[Suppl.]* 7: 42-53, 1980.
25. Bruck, K. and H. Olschewski. Body temperature related factors diminishing the drive to exercise. *Can.J.Physiol.Pharmacol.* 65: 1274-1280, 1987.
26. Buick, F. J., N. Gledhill, A. B. Froese, L. Spriet, and E. C. Meyers. Effect of induced erythrocythemia on aerobic work capacity. *J.Appl.Physiol.* 48: 636-642, 1980.
27. Burger, F. J. and F. A. Fuhrman. Evidence of injury to tissues after hyperthermia. *Am.J.Physiol.* 206: 1062-1064, 1964.
28. Buskirk, E. R., J. Kollias, R. F. Akers, E. K. Prokop, and E. P. Reategui. Maximal performance at altitude and on return from altitude in conditioned runners. *J.Appl.Physiol.* 23: 259-266, 1967.
29. Bynum, G. D., K. B. Pandolf, W. H. Schuette, R. F. Goldman, D. E. Lees, J. Whang-Peng, E. R. Atkinson, and J. M. Bull. Induced hyperthermia in sedated

- humans and the concept of critical thermal maximum. *Am.J.Physiol.* 235: R228-R236, 1978.
30. Caputa, M., G. Feistkorn, and C. Jessen. Effects of brain and trunk temperatures on exercise performance in goats. *Pflugers Arch.* 406: 184-189, 1986.
 31. Caputa, M., W. Kadiela, and J. Narebski. Cerebral temperature regulation in resting and running guinea-pigs (*cavia porcellus*). *J.Therm.Biol.* 8: 265-272, 1983.
 32. Carlisle, F. Effects of preliminary passive warming on swimming performance. *Res.Q.* 27: 143-152, 1956.
 33. Cerretelli, P., P. E. Di Prampero, and G. Sassi. The heart rate - VO₂ relationship in different types of dynamic exercise. *Arch.Fisiol.* 65: 358-365, 1967.
 34. Chao, T. C., R. Sinniah, and J. E. Pakiam. Acute heat stroke deaths. *Pathology* 13: 145-156, 1981.
 35. Chapman, C. B., A. Henschel, J. Minckler, A. Forsgren, and A. Keys. The effects of exercise on renal plasma flow in normal male subjects. *J.Clin.Invest.* 27: 639-644, 1948.
 36. Chase, G. A., C. Grave, and L. B. Rowell. Independence of changes in functional and performance capacities attending prolonged bed rest. *Aerosp.Med.* 37: 1232-1238, 1966.
 37. Cheung, S. S. and T. M. McLellan. Heat acclimation, aerobic fitness, and hydration effects on tolerance during uncompensable heat stress. *J.Appl.Physiol.* 84: 1731-1739, 1998.
 38. Cheung, S. S. and T. M. McLellan. Influence of hydration status and fluid replacement on heat tolerance while wearing NBC protective clothing. *Eur.J.Appl.Physiol.* 77: 139-148, 1998.

39. Clarke, D. H. and J. Royce. Rate of muscle tension development and release under extreme temperatures. *Int.Z.Angew.Physiol.Einschl.Arbeitsphysiol.* 19: 330-336, 1962.
40. Clarke, R. S. J., R. F. Hellon, and A. R. Lind. The duration of sustained contractions of the human forearm at different muscle temperatures. *J.Physiol.* 143: 454-473, 1958.
41. Clausen, J. P. Effects of physical training on cardiovascular adjustments to exercise in man. *Physiol.Rev.* 57: 779-815, 1977.
42. Cooper, K. E. and D. M. Kerslake. Changes in heart rate during exposure of the skin to radiant heat. *Clin.Sci.* 14: 125-135, 1955.
43. Costill, D. L. Effects of water temperature on aerobic working capacity. *Res.Q.* 39: 67-73, 1966.
44. Coyle, E. F., M. K. Hemmert, and A. R. Coggan. Effects of detraining on cardiovascular responses to exercise: role of blood volume. *J.Appl.Physiol.* 60: 95-99, 1986.
45. Craig, F. N. and H. L. Froehlich. Endurance of preheated men in exhausting work. *J.Appl.Physiol.* 24: 636-639, 1968.
46. Craig, F. N., H. W. Garren, H. Frankel, and W. V. Blevins. Heat load and voluntary tolerance time. *J.Appl.Physiol.* 6: 634-644, 1954.
47. Cumming, G. R. and W. Friesen. Bicycle ergometer measurement of maximal oxygen uptake in children. *Can.J.Physiol.Pharmacol.* 45: 937-946, 1967.
48. Cunningham, D. A., P. B. Goode, and J. B. Critz. Cardiorespiratory response to exercise on a rowing and bicycle ergometer. *Med.Sci.Sports* 7: 37-43, 1975.
49. Cunningham, D. A., B. Macfarlane Van Waterschoot, D. H. Paterson, M. Lefcoe, and S. P. Sangal. Reliability and reproducibility of maximal oxygen uptake measurement in children. *Med.Sci.Sports* 9: 104-108, 1977.

50. Davies, C. T. M. and A. J. Sargeant. Physiological responses to one- and two-legged exercise breathing air and 45% oxygen. *J.Appl.Physiol.* 36: 142-148, 1974.
51. Davies, C. T. M. and J. Sargeant. Effects of training on the physiological responses to one- and two-leg work. *J.Appl.Physiol.* 38: 377-381, 1975.
52. Davies, C. T. M. and K. Young. Effect of temperature on the contractile properties and muscle power of triceps surae in humans. *J.Appl.Physiol.* 55: 191-195, 1983.
53. Davies, M., B. Ekblom, U. Bergh, and I. Kanstrup-Jensen. The effects of hypothermia on submaximal and maximal work performance. *Acta Physiol.Scand.* 95: 201-202, 1975.
54. Dempsey, J. A., L. Adams, D. M. Ainsworth, R. F. Fregosi, C. G. Gallagher, A. Guz, B. D. Johnson, and S. K. Powers. Airway, lung, and respiratory function during exercise. In Rowell, L. B. and J. T. Shepherd, eds., *Handbook of physiology*. Section 12: Exercise: Regulation and integration of multiple systems. New York, Oxford University Press. 1996, 448-514.
55. Dempsey, J. A., P. E. Hanson, C. Pegelow, and A. Claremont. Limitations to exercise capacity and endurance: pulmonary system. *Can.J.Appl.Sport Sci.* 7: 4-13, 1982.
56. Dempsey, J. A., P. G. Hanson, and K. S. Henderson. Exercise-induced arterial hypoxaemia in healthy human subjects at sea level. *J.Physiol.* 355: 161-175, 1984.
57. Dempsey, J. A. and J. Rankin. Physiologic adaptations of gas transport system to muscular work in health and disease. *Am.J.Phys.Med.* 46: 582-647, 1967.
58. Dill, D. B., H. T. Edwards, P. S. Bauer, and E. J. Levenson. Physical performance in relation to external temperature. *Arbeitsphysiologie* 3: 508-518, 1931.

59. Dimri, G. P., M. S. Malhotra, J. Sen Gupta, T. S. Kumar, and B. S. Aora. Alterations in aerobic-anaerobic metabolic proportions of metabolism during work in heat. *Eur.J.Appl.Physiol.* 45: 43-50, 1980.
60. Dotan, R. and O. Bar-Or. Climatic heat stress and performance in the Wingate anaerobic test. *Eur.J.Appl.Physiol.* 44: 237-243, 1980.
61. Edwards, R. H. T., R. C. Harris, E. Hultman, L. Kaijser, D. Koh, and L.-O. Nordesjo. Effect of temperature on muscle energy metabolism and endurance during successive isometric contractions, sustained to fatigue, of the quadriceps muscle in man. *J.Physiol.* 220: 335-352, 1972.
62. Ekblom, B., A. N. Goldbarg, and B. Gullbring. Response to exercise after blood loss and reinfusion. *J.Appl.Physiol.* 33: 175-180, 1972.
63. Ekblom, B., R. Hout, E. M. Stein, and A. Thorstensson. Effects of changes in arterial oxygen content on circulation and physical performance. *J.Appl.Physiol.* 39: 71-75, 1975.
64. Ekblom, B., G. Wilson, and P.-O. Astrand. Central circulation during exercise after venesection and reinfusion of red blood cells. *J.Appl.Physiol.* 40: 379-383, 1976.
65. Falk, B., S. Radom-Isaac, J. R. Hoffman, Y. Wang, Y. Yarom, A. Magazanik, and Y. Weinstein. The effect of heat exposure on performance of and recovery from high-intensity, intermittent exercise. *Int.J.Sports Med.* 19: 1-6, 1998.
66. Falls, H. B. and L. D. Humphrey. A comparison of methods for eliciting maximum oxygen uptake from college women during treadmill walking. *Med.Sci.Sports* 5: 239-241, 1973.
67. Faulkner, J. A., J. Kollias, C. B. Favour, E. R. Buskirk, and B. Balke. Maximum aerobic capacity and running performance at altitude. *J.Appl.Physiol.* 24: 685-691, 1968.

68. Faulkner, J. A., D. E. Roberts, R. L. Elk, and J. Conway. Cardiovascular responses to submaximum and maximum effort cycling and running. *J.Appl.Physiol.* 30: 457-461, 1971.
69. Febbraio, M. A. Does muscle function and metabolism affect exercise performance in the heat? *Exerc.Sport Sci.Rev.* 28: 171-176, 2000.
70. Febbraio, M. A., M. F. Carey, R. J. Snow, C. G. Stathis, and M. Hargreaves. Influence of elevated muscle temperature on metabolism during intense, dynamic exercise. *Am.J.Physiol.* 271: R1251-R1255, 1995.
71. Febbraio, M. A., P. Murton, S. E. Selig, S. A. Clark, D. L. Lambert, D. J. Angus, and M. F. Carey. Effect of CHO ingestion on exercise metabolism and performance in different ambient temperatures. *Med.Sci.Sports Exerc.* 28: 1380-1387, 1996.
72. Ferguson, R. J., G. G. Murcotte, and R. R. Montpetit. A maximal oxygen uptake test during ice skating. *Med.Sci.Sports* 1: 207-211, 1969.
73. Fink, W. J., D. L. Costill, and W. J. VanHandle. Leg muscle metabolism during exercise in the heat and cold. *Eur.J.Appl.Physiol.* 34: 183-190, 1975.
74. Froelicher, V. F. Jr., H. Brammell, G. Davis, I. Noguera, A. Stewart, and M. C. Lancaster. A comparison of three maximal treadmill exercise protocol. *J.Appl.Physiol.* 36: 720-725, 1974.
75. Fruth, J. M. and C. V. Gisolfi. Work-heat tolerance in endurance-trained rats. *J.Appl.Physiol.* 54: 249-253, 1983.
76. Fuller, A., R. N. Carter, and D. Mitchell. Brain and abdominal temperatures at fatigue in rats exercising in the heat. *J.Appl.Physiol.* 84: 877-883, 1998.
77. Galloway, S. D. R. and R. J. Maughan. Effects of ambient temperature on the capacity to perform prolonged cycle exercise in man. *Med.Sci.Sports Exerc.* 29: 1240-1249, 1997.

78. Glassford, R. G., G. H. Y. Baycroft, A. W. Sedgwick, and R. B. J. Macnab. Comparison of maximal oxygen uptake values determined by predicted and actual methods. *J.Appl.Physiol.* 20: 509-513, 1965.
79. Gledhill, N. The influence of altered blood volume and oxygen transport capacity on aerobic performance. *Exerc.Sport Sci.Rev.* 13: 75-93, 1985.
80. Gledhill, N., D. Cox, and R. Jamnik. Endurance athletes' stroke volume does not plateau: major advantage is diastolic function. *Med.Sci.Sports Exerc.* 26: 1116-1121, 1994.
81. Gleser, M. A. Effects of hypoxia and physical training on hemodynamic adjustments to one-legged exercise. *J.Appl.Physiol.* 34: 655-659, 1973.
82. Gleser, M. A., D. H. Horstman, and R. P. Mello. The effect on Vo₂ max of adding arm work to maximal leg work. *Med.Sci.Sports* 6: 104-107, 1974.
83. Gollnick, P. D., R. B. Armstrong, C. W. Saubert IV, K. Piehl, and B. Saltin. Enzyme activity and fiber composition in skeletal muscle of untrained and trained men. *J.Appl.Physiol.* 33: 312-319, 1972.
84. Gonzalez-Alonso, J., J. A. L. Calbet, and B. Nielsen. Muscle blood flow is reduced with dehydration during prolonged exercise in humans. *J.Physiol.* 513: 895-905, 1998.
85. Gonzalez-Alonso, J., J. A. L. Calbet, and B. Nielsen. Metabolic and thermodynamic responses to dehydration-induced reductions in muscle blood flow in exercising humans. *J.Physiol.* 520: 577-589, 1999.
86. Gonzalez-Alonso, J., R. Mora-Rodriguez, P. R. Below, and E. F. Coyle. Dehydration markedly impairs cardiovascular function in hyperthermic endurance athletes during exercise. *J.Appl.Physiol.* 82: 1229-1236, 1997.
87. Gonzalez-Alonso, J., C. Teller, S. L. Andersen, F. B. Jensen, T. Hyldig, and B. Nielsen. Influence of body temperature on the development of fatigue during prolonged exercise in the heat. *J.Appl.Physiol.* 86: 1032-1039, 1999.

88. Grimby, G. Renal clearances at rest and during physical exercise after injection of bacterial pyrogen. *J.Appl.Physiol.* 20: 137-141, 1965.
89. Grimby, G. Renal Clearances during prolonged supine exercise at different loads. *J.Appl.Physiol.* 20: 1294-1298, 1965.
90. Grose, J. E. Depression of muscle fatigue curves by heat and cold. *Res.Q.* 29: 19-31, 1958.
91. Hagberg, J. M., F. J. Nagle, and J. L. Carlson. Transient O₂ uptake response at the onset of exercise. *J.Appl.Physiol.* 44: 90-92, 1978.
92. Hales, J. R. S., R. W. Hubbard, and S. L. Gaffin. Limitations of heat tolerance. In Fregly, M. J. and C. M. Blatteis, eds., *Handbook of Physiology. Section 4: Environmental physiology.* New York, Oxford University Press. 1996, 285-355.
93. Hales, J. R. S., M. Khogali, A. A. Fawcett, and M. K. Y. Mustafa. Circulatory changes associated with heat stroke: Observations in an experimental animal model. *Clin.Exp.Pharmacol.Physiol.* 14: 761-777, 1987.
94. Hanson, P. G. and S. W. Zimmerman. Exertional heatstroke in novice runners. *JAMA* 242: 154-157, 1979.
95. Happ, W. P., W. W. Tuttle, and M. Wilson. The physiological effects of cold abdominal packs. *Res.Q.* 20: 153-164, 1949.
96. Hargreaves, M. and M. Febbraio. Limits to exercise performance in the heat. *Int.J.Sports Med.* 19: S115-S116, 1998.
97. Henry, J. P. and O. H. Gauer. The influence of temperature upon venous pressure in the foot. *J.Clin.Invest.* 29: 855-861, 1950.
98. Hermansen, L., B. Ekblom, and B. Saltin. Cardiac output during submaximal and maximal treadmill and bicycle exercise. *J.Appl.Physiol.* 29: 82-86, 1970.
99. Hermansen, L. and B. Saltin. Oxygen uptake during maximal treadmill and bicycle exercise. *J.Appl.Physiol.* 26: 31-37, 1969.

100. Hessemer, V., D. Langusch, K. Bruck, R. H. Bodeker, and T. Breidenbach. Effect of slightly lowered body temperatures on endurance performance in humans. *J.Appl.Physiol.* 57: 1731-1737, 1984.
101. Ho, C. W., J. L. Beard, P. A. Farrell, C. T. Minson, and W. L. Kenney. Age, fitness, and regional blood flow during exercise in the heat. *J.Appl.Physiol.* 82: 1126-1135, 1997.
102. Hoffman, J. R., B. Falk, S. Radom, Y. Wang, Y. Yarom, and Y. Weinstein. The effect of heat exposure on performance and recovery from anaerobic exercise. *Med.Sci.Sports Exerc.* 26: S126, 1994.
103. Hogan, M. C., J. Roca, P. D. Wagner, and J. B. West. Limitation of maximal O₂ uptake and performance by acute hypoxia in dog muscle in situ. *J.Appl.Physiol.* 65: 815-821, 1988.
104. Hohimer, A. R., J. R. Hales, L. B. Rowell, and O. A. Smith. Regional distribution of blood flow during mild dynamic leg exercise in the baboon. *J.Appl.Physiol.* 55: 1173-1177, 1983.
105. Holmer, I. and U. Bergh. Metabolic and thermal response to swimming in water at varying temperatures. *J.Appl.Physiol.* 37: 702-705, 1974.
106. Horvath, S. M. and E. D. Jr. Michael. Responses of young women to gradually increasing and constant load maximal exercise. *Med.Sci.Sports* 2: 128-131, 1970.
107. Howley, E. T., D. R. Jr. Bassett, and H. G. Welch. Criteria for maximal oxygen uptake: review and commentary. *Med.Sci.Sports Exerc.* 27: 1292-1301, 1995.
108. Howley, E. T. and B. D. Franks. Health fitness instructor's handbook. Champaign, IL, Human Kinetics. 1997, 282.
109. Hubbard, R. W. Effects of exercise in the heat on predisposition to heatstroke. *Med.Sci.Sports Exerc.* 11: 66-71, 1979.

110. Hubbard, R. W., W. D. Bowers, W. T. Matthew, F. C. Curtis, R. E. L. Criss, G. M. Sheldon, and J. W. Ratteree. Rat model of acute heat stroke mortality. *J.Appl.Physiol.:Respirat.Environ.Exercise Physiol.* 42: 809-816, 1977.
111. Issekutz, B., N. C. Birkhead, and K. Rodahl. The use of respiratory quotients in assessment of aerobic work capacity. *J.Appl.Physiol.* 17: 47-50, 1962.
112. Jessen, C. Hyperthermia and its effect on exercise performance. In Hales, J. R. S. and D. A. B. Richards, eds., *Heat Stress: Physical Exertion and Environment*. Amsterdam, Elsevier Science Publishers B.V. (Biomedical Division). 1987, 241-249.
113. Johnson, R. L. Jr. Pulmonary diffusion as a limiting factor in exercise stress. *Circ.Res.Suppl.* XX and XXI: I-154-I-160, 1967.
114. Johnson, R. L. Jr., G. J. F. Heigenhauser, C. C. W. Hsia, N. L. Jones, and P. D. Wagner. Determinants of gas exchange and acid-base balance during exercise. In Rowell, L. B. and J. T. Shepherd, eds., *Handbook of Physiology. Section 12: Exercise: Regulation and integration of multiple systems*. New York, Oxford University Press. 1996, 515-584.
115. Jose, A. D., F. Stitt, and D. Collison. The effects of exercise and changes in body temperature on the intrinsic heart rate in man. *Am.Heart J.* 79: 488-497, 1970.
116. Kamon, E. and K. B. Pandolf. Maximal aerobic power during laddermill climbing, uphill running, and cycling. *J.Appl.Physiol.* 32: 467-473, 1972.
117. Kanstrup, I.-L. and B. Ekblom. Acute hypervolemia, cardiac performance, and aerobic power during exercise. *J.Appl.Physiol.* 52: 1186-1191, 1982.
118. Kanstrup, I.-L. and B. Ekblom. Blood volume and hemoglobin concentration as determinants of maximal aerobic power. *Med.Sci.Sports Exerc.* 16: 256-262, 1984.

119. Kasch, F. W., W. H. Phillips, W. D. Ross, J. E. L. Carter, and J. L. Boyer. A comparison of maximal oxygen uptake by treadmill and step-test procedures. *J.Appl.Physiol.* 21: 1387-1388, 1966.
120. Katch, V. L., S. S. Sady, and P. Freedson. Biological variability in maximum aerobic power. *Med.Sci.Sports Exerc.* 14: 21-25, 1982.
121. Kellogg, D. L. Jr., J. M. Johnson, and W. A. Kosiba. Control of internal temperature threshold for active cutaneous vasodilation by exercise. *J.Appl.Physiol.* 71: 2476-2482, 1991.
122. Kenney, R. A. The effect of exercise in hot humid environments on the renal function of the West African. *J.Physiol.* 118: 25P-26P, 1952.
123. Kim, Y. D., R. Lake, D. E. Lees, W. H. Schuette, J. M. Bull, V. Weise, and I. J. Kopin. Hemodynamic and plasma catecholamine responses to hyperthermic cancer therapy in humans. *Am.J.Physiol.* 237: H570-H574, 1979.
124. Klausen, K., D. B. Dill, E. E. Jr. Phillips, and D. McGregor. Metabolic reactions to work in the desert. *J.Appl.Physiol.* 22: 292-296, 1967.
125. Klausen, K., N. H. Secher, J. P. Clausen, O. Hartling, and J. Trap-Jensen. Central and regional circulatory adaptations to one-leg training. *J.Appl.Physiol.* 52: 976-983, 1982.
126. Kozlowski, S., Brzezinska.Z., B. Kruk, H. Haciuba-Uscilko, J. E. Greenleaf, and K. Nazar. Exercise hyperthermia as a factor limiting physical performance: temperature effect of muscle metabolism. *J.Appl.Physiol.* 59: 766-773, 1985.
127. Kruk, B., H. Pekkarinen, K. Manninen, and O. Hanninen. Comparison in men of physiological responses to exercise of increasing intensity at low and moderate ambient temperature. *Eur.J.Appl.Physiol.* 62: 353-357, 1990.
128. Latzka, W. A., M. N. Sawka, S. J. Montain, G. S. Skrinar, R. A. Fielding, R. P. Matott, and K. B. Pandolf. Hyperhydration: tolerance and cardiovascular effects

- during uncompensable exercise-heat stress. *J.Appl.Physiol.* 84: 1858-1864, 1998.
129. Lee, D. T. and E. M. Haymes. Exercise duration and thermoregulatory responses after whole body precooling. *J.Appl.Physiol.* 79: 1971-1976, 1995.
 130. MacDougal, J. D., W. G. Reddan, C. R. Layton, and J. A. Dempsey. Effects of metabolic hyperthermia on performance during heavy prolonged exercise. *J.Appl.Physiol.* 36: 538-544, 1974.
 131. Magel, J. R. and J. A. Faulkner. Maximum oxygen uptakes of college swimmers. *J.Appl.Physiol.* 22: 929-933, 1967.
 132. Maksud, M. G. and K. D. Coutts. Comparison of continuous and discontinuous graded treadmill test for maximal oxygen uptake. *Med.Sci.Sports* 3: 63-65, 1971.
 133. Margaria, R., P. Cerretelli, S. Marchi, and L. Rossi. Maximum exercise in oxygen. *Int.Z.Angew.Physiol.Einschl.Arbeitsphysiol.* 18: 465-467, 1961.
 134. Marx, H. J., L. B. Rowell, R. D. Conn, R. A. Bruce, and F. Kusumi. Maintenance of aortic pressure and total peripheral resistance during exercise in the heat. *J.Appl.Physiol.* 22: 519-525, 1967.
 135. Maxwell, N. S., T. C. Aitchison, and M. A. Nimmo. The effect of climatic heat stress on intermittent supramaximal running performance in humans. *Exp.Physiol.* 81: 833-845, 1996.
 136. McArdle, W. D., F. I. Katch, and G. S. Pechar. Comparison of continuous and discontinuous treadmill and bicycle tests for max VO₂. *Med.Sci.Sports Exerc.* 5: 156-160, 1973.
 137. McArdle, W. D., F. I. Katch, G. S. Pechar, L. Jacobson, and S. Ruck. Reliability and interrelationships between maximal oxygen intake, physical work capacity and step test scores in college women. *Med.Sci.Sports* 4: 182-186, 1972.

138. McArdle, W. D. and J. R. Magel. Physical work capacity and maximal oxygen uptake in treadmill and bicycle exercise. *Med.Sci.Sports* 2: 118-123, 1970.
139. McArdle, W. D., J. R. Magel, G. R. Lesmes, and G. S. Pechar. Metabolic and cardiovascular adjustment to work in air and water at 18, 25 and 33 degrees C. *J.Appl.Physiol.* 40: 85-90, 1976.
140. McCann, D. J. and W. C. Adams. Wet bulb globe temperature index and performance in competitive distance runners. *Med.Sci.Sports Exerc.* 29: 955-961, 1997.
141. McLellan, T. M. and S. S. Cheung. Impact of fluid replacement on heat storage while wearing protective clothing. *Ergonomics* 43: 2020-2030, 2000.
142. McLellan, T. M., G. A. Gannon, J. Zamecnik, V. Gil, and G. M. Brown. Low doses of melatonin and diurnal effects on thermoregulation and tolerance to uncompensable heat stress. *J.Appl.Physiol.* 87: 308-316, 1999.
143. McLellan, T. M., J. I. Pope, J. B. Cain, and S. S. Cheung. Effects of metabolic rate and ambient vapour pressure on heat strain in protective clothing. *Eur.J.Appl.Physiol.* 74: 518-527, 1996.
144. Mehta, A. C. and R. N. Baker. Persistent neurological deficits in heat stroke. *Neurology* 20: 336-340, 1970.
145. Milic-Emili, G., J. M. Petit, and R. Deroanne. Mechanical work of breathing during exercise in trained and untrained subjects. *J.Appl.Physiol.* 17: 43-46, 1962.
146. Mills, P. J., N. C. Smith, I. Casas, P. Harris, R. C. Harris, and D. J. Marlin. Effects of exercise intensity and environmental stress on indices of oxidative stress and iron homeostasis during exercise in the horse. *Eur.J.Appl.Physiol.* 74: 60-66, 1996.
147. Mitchell, J. H. and Blomquist. Maximal oxygen uptake. *N.Engl.J.Med.* 284: 1018-1022, 1971.

148. Mitchell, J. H., B. J. Sproule, and C. B. Chapman. The physiological meaning of the maximal oxygen intake test. *J.Clin.Invest.* 37: 538-547, 1958.
149. Miyamura, M. and Y. Honda. Oxygen intake and cardiac output during maximal treadmill and bicycle exercise. *J.Appl.Physiol.* 32: 185-188, 1972.
150. Montain, S. J., M. N. Sawka, B. S. Cadarette, M. D. Quigley, and J. M. McKay. Physiological tolerance to uncompensable heat stress: effects of exercise intensity, protective clothing, and climate. *J.Appl.Physiol.* 77: 216-222, 1994.
151. Moody, D. L., J. Kollias, and E. R. Buskirk. Evaluation of aerobic capacity in lean and obese women with four test procedures. *J.Sports Med.Phys.Fitness* 9: 1-9, 1969.
152. Mosley, P. L. Exercise, heat, and thermotolerance: molecular mechanisms. In Gisolfi, C. V., D. R. Lamb, and E. R. Nadel, eds., *Perspectives in Exercise Science and Sports Medicine: Exercise, Heat, and Thermoregulation*. Dubuque, Brown and Benchmark. 1993, 305-328.
153. Myers, J., D. Walsh, M. Sullivan, and V. Froelicher. Effect of sampling on variability and plateau in oxygen uptake. *J.Appl.Physiol.* 68: 404-410, 1990.
154. Myler, G. R., A. G. Hahn, and D. M. Tumilty. The effect of preliminary skin cooling on performance of rowers in hot conditions. *Excel* 6: 17-21, 1989.
155. Nadel, E. R. Circulatory and thermal regulations during exercise. *Fed.Proc.* 39: 1491-1497, 1980.
156. Nadel, E. R., E. Cafarelli, M. F. Roberts, and C. B. Wenger. Circulatory regulation during exercise in different ambient temperatures. *J.Appl.Physiol.* 46: 430-437, 1979.
157. Nadel, E. R., S. M. Fortney, and C. B. Wenger. Effect of hydration state on circulatory and thermal regulations. *J.Appl.Physiol.* 49: 715-721, 1980.
158. Newton, J. L. The assessment of maximal oxygen intake. *The Journal of Sports Medicine and Physical Fitness* 3: 164-169, 1963.

159. Nielsen, B. The implications of endogenous versus exogenous heat loads in stress tolerance. In Hales, J. R. S. and D. A. B. Richards, eds., *Heat Stress: Physical Exertion and Environment*. Amsterdam, Elsevier Science Publishers B.V. (Biomedical Division). 1987, 337-353.
160. Nielsen, B. Heat stress causes fatigue. In Marconnet, P., P. V. Komi, B. Saltin, and O. M. Sejersted, eds., *Muscle fatigue mechanisms in exercise and training*. Karger, Basel. 1992, 207-217.
161. Nielsen, B., J. R. S. Hales, S. Strange, N. J. Christensen, J. Warberg, and B. Saltin. Human circulatory and thermoregulatory adaptations with heat acclimation and exercise in a hot, dry environment. *J.Physiol.* 460: 467-485, 1993.
162. Nielsen, B., R. Kubica, A. Bonnesen, I. B. Rasmussen, J. Stoklosa, and B. Wilk. Physical work capacity after dehydration and hyperthermia. *Scand.J.Sports Sci.* 3: 2-10, 1981.
163. Nielsen, B., S. L. Nielsen, and F. B. Petersen. Thermoregulation during positive and negative work at different environmental temperatures. *Acta Physiol.Scand.* 85: 249-257, 1972.
164. Nielsen, B., L. B. Rowell, and F. Bonde-Petersen. Cardiovascular responses to heat stress and blood volume displacements during exercise in man. *Eur.J.Appl.Physiol.* 52: 370-374, 1984.
165. Nielsen, B., G. Savard, E. A. Richter, M. Hargreaves, and B. Saltin. Muscle blood flow and muscle metabolism during exercise and heat stress. *J.Appl.Physiol.* 69: 1040-1046, 1990.
166. Nielsen, B., S. Strange, N. J. Christensen, J. Warberg, and B. Saltin. Acute and adaptive responses in humans to exercise in a warm, humid environment. *Pflugers Arch.* 434: 49-56, 1997.

167. Nybo, L., T. Jensen, B. Nielsen, and J. Gonzalez-Alonso. Effects of marked hyperthermia with and without dehydration on VO₂ kinetics during intense exercise. *J.Appl.Physiol.* 90: 1057-1064, 2001.
168. Olschewski, H. and K. Bruck. Thermoregulatory, cardiovascular, and muscular factors related to exercise after precooling. *J.Appl.Physiol.* 64: 803-811, 1988.
169. Ouellet, Y., S. Poh, and M. Becklake. Circulatory factors limiting maximal aerobic exercise capacity. *J.Appl.Physiol.* 52: 874-880, 1969.
170. Parkin, J. M., M. F. Carey, S. Zhao, and M. A. Febbraio. Effect of ambient temperature on human skeletal muscle metabolism during fatiguing submaximal exercise. *J.Appl.Physiol.* 86: 902-908, 1999.
171. Patton, J. F. and J. A. Vogel. Effects of acute cold exposure on submaximal endurance performance. *Med.Sci.Sports Exerc.* 16: 494-497, 1984.
172. Petersen, E. B. and H. Vejby-Christensen. Effect of body temperature on steady state ventilation and metabolism in exercise. *Acta Physiol.Scand.* 89: 342-351, 1973.
173. Petrofsky, J. S. and A. R. Lind. Insulative power of body fat on deep muscle temperatures and isometric endurance. *J.Appl.Physiol.* 39: 639-642, 1975.
174. Pirnay, F., R. Deroanne, and J. M. Petit. Maximal oxygen consumption in a hot environment. *J.Appl.Physiol.* 218: 642-645, 1970.
175. Pirnay, F., R. Deroanne, and J. M. Petit. Influence of water temperature on thermal, circulatory and respiratory responses to muscular work. *Eur.J.Appl.Physiol.* 37: 129-136, 1977.
176. Pollock, M. L., R. L. Bohannon, K. H. Cooper, J. J. Ayres, A. Ward, S. R. White, and A. C. Linnerud. A comparative analysis of four protocols for maximal treadmill stress testing. *Am.Heart J.* 92: 39-46, 1976.

177. Powers, S. K., S. Dodd, J. Woodyard, R. E. Beadle, and G. Church. Alterations in haemoglobin saturation during incremental arm and leg exercise. *Br.J.Sports Med.* 18: 212-216, 1984.
178. Powers, S. K. and J. Williams. Exercise-induced hypoxaemia in highly trained athletes. *Sports Med.* 4: 46-53, 1987.
179. Quirion, A., L. Laurencelle, L. Paulin, A. Therminarias, G. R. Brisson, A. Audet, S. Dulac, and P. Vogelaere. Metabolic and hormonal responses during exercise at 20, 0, and -20 C. *Int.J.Biometeorol.* 33: 227-232, 1989.
180. Radigan, L. R. and S. Robinson. Effects of environmental heat stress and exercise on renal blood flow and filtration rate. *J.Appl.Physiol.* 2: 185-191, 1949.
181. Reybouck, T., G. F. Heigenhauser, and J. A. Faulkner. Limitations to maximum oxygen uptake in arm, leg, and arm-leg ergometry. *J.Appl.Physiol.* 38: 774-779, 1975.
182. Robbins, A. C. The effects of hot and cold shower baths upon adolescents participating in physical education classes. *Res.Q.* 13: 373-380, 1942.
183. Robertson, R. J., R. Gilcher, K. R. Metz, G. S. Skrinar, T. G. Allison, H. T. Bahnson, R. A. Abbott, R. Becker, and J. Falkel. Effect of induced erythrocythemia on hypoxia tolerance during physical exercise. *J.Appl.Physiol.* 53: 490-492, 1982.
184. Robinson, B. F., S. E. Epstein, R. L. Kahler, and E. Braunwald. Circulatory effects of acute expansion of blood volume: Studies during maximal exercise and rest. *Circ.Res.* 19: 26-32, 1966.
185. Robinson, S. Experimental studies of physical fitness in relation to age. *Arbeitsphysiologie* 10: 251-323, 1938.

186. Rodahl, K., S. M. Horvath, N. C. Birkhead, and B. Issekutz. Effect of dietary protein on physical work capacity during severe cold stress. *J.Appl.Physiol.* 17: 763-767, 1962.
187. Rosen, M. The effects of a cold abdominal spray upon a repeat performance in the 440 yard run. *Res.Q.* 23: 226-230, 1952.
188. Rowell, L. B. Human cardiovascular adjustments to exercise and thermal stress. *Physiol.Rev.* 54: 75-159, 1974.
189. Rowell, L. B. Cardiovascular adjustments to thermal stress. In Shepherd, J. T. and F. M. Abboud, eds., *Handbook of Physiology. The Cardiovascular System. Peripheral Circulation and Organ Blood Flow.* Bethesda MD, American Physiological Society. 1983, 967-1023.
190. Rowell, L. B. *Human circulation: regulation during physical stress.* New York, Oxford University Press. 1986, 213-402.
191. Rowell, L. B. *Human cardiovascular control.* New York, Oxford University Press, Inc. 1993, 206.
192. Rowell, L. B., J. R. Blackmon, and R. A. Bruce. Indocyanine green clearance and estimated hepatic blood flow during mild to maximal exercise in upright man. *J.Clin.Invest.* 43: 1677-1690, 1964.
193. Rowell, L. B., J. R. Blackmon, R. H. Martin, J. A. Mazzaella, and R. A. Bruce. Hepatic clearance of indocyanine green in man under thermal and exercise stresses. *J.Appl.Physiol.* 20: 384-394, 1965.
194. Rowell, L. B., G. L. Brengelmann, J. R. Blackmon, R. D. Twiss, and F. Kusumi. Splanchnic blood flow and metabolism in heat stressed man. *J.Appl.Physiol.* 24: 475-484, 1968.
195. Rowell, L. B., G. L. Brengelmann, J.-M. R. Detry, and C. Wyss. Venomotor responses to rapid changes in skin temperature in exercising man. *J.Appl.Physiol.* 30: 64-71, 1971.

196. Rowell, L. B., G. L. Brengelmann, and P. R. Freund. Unaltered norepinephrine-heart rate relationship in exercise with exogenous heat. *J.Appl.Physiol.* 62: 646-650, 1987.
197. Rowell, L. B., G. L. Brengelmann, J. A. Murray, K. K. Kraning II, and F. Kusumi. Human metabolic responses to hyperthermia during mild to maximal exercise. *J.Appl.Physiol.* 26: 395-402, 1969.
198. Rowell, L. B. and J. M. Johnson. Role of the splanchnic circulation in reflex control of the cardiovascular system. In Shepherd, A. T. and D. N. Granger, eds., *Physiology of the Intestinal Circulation*. New York, Raven Press. 1984, 153-163.
199. Rowell, L. B., K. K. Kraning II, J. W. Kennedy, and T. O. Evans. Central circulatory responses to work in dry heat before and after acclimatization. *J.Appl.Physiol.* 22: 509-518, 1967.
200. Rowell, L. B., H. J. Marx, R. A. Bruce, R. D. Conn, and F. Kusumi. Reductions in cardiac output, central blood volume, and stroke volume with thermal stress in normal men during exercise. *J.Clin.Invest.* 45: 1801-1816, 1966.
201. Rowell, L. B., J. A. Murray, G. L. Brengelmann, and K. K. Kraning II. Human cardiovascular adjustments to rapid changes in skin temperature during exercise. *Circ.Res.* 24: 711-724, 1969.
202. Rowell, L. B., B. Saltin, and N. J. Christensen. Is peak quadriceps flow in humans even higher during exercise with hypoxemia? *Am.J.Physiol.* 251: H1038-H1044, 1986.
203. Rowell, L. B., H. L. Taylor, Y. Wang, and W. B. Carlson. Saturation of arterial blood with oxygen during maximal exercise. *J.Appl.Physiol.* 19: 284-286, 1964.
204. Sakate, T. The effect of air temperature on physical working capacity. *J.Hum.Ergology (Tokyo)* 7: 127-134, 1978.

205. Saltin, B. Cold, work, and altitude: Central circulatory aspects. In Helfferich, C., ed., *Proceedings Symposia on Arctic Biology and Medicine VI. The Physiology of Work in Cold and Altitude*. 1966, 313-360.
206. Saltin, B. and P.-O. Astrand. Maximal oxygen uptake in athletes. *J.Appl.Physiol.* 23: 353-358, 1967.
207. Saltin, B., G. Blomqvist, J. H. Mitchell, R. L. Jr. Johnson, K. Wildenthal, and C. B. Chapman. Response to exercise after bed rest and after training. *Circulation* 38 Suppl. 7: VII-1-VII-78, 1968.
208. Saltin, B., A. P. Gagge, U. Bergh, and J. A. J. Stolwijk. Body temperatures and sweating during exhaustive exercise. *J.Appl.Physiol.* 32: 635-643, 1972.
209. Saltin, B. and P. Gollnick. Skeletal muscle adaptability: Significance for metabolism and performance. In Peachey, L. D., R. H. Adrian, and S. R. Geiger, eds., *Handbook of Physiology. Section 10: Skeletal muscle*. Bethesda, MD, American Physiological Society. 1983, 555-631.
210. Saltin, B., K. Nazar, D. L. Costill, E. Stein, E. Jansson, B. Essen, and P. D. Gollnick. The nature of the training response; peripheral and central adaptations to one-legged exercise. *Acta Physiol.Scand.* 96: 289-305, 1976.
211. Sarelius, I. H. and J. D. Sinclair. Effects of small changes of blood volume on oxygen delivery and tissue oxygenation. *Am.J.Physiol.* 240: H177-H184, 1981.
212. Sargeant, A. J. Effect of muscle temperature on leg extension force and short-term power output in humans. *Eur.J.Appl.Physiol.* 56: 693-698, 1987.
213. Savard, G., B. Nielsen, J. Laszczynska, B. E. Larsen, and B. Saltin. Muscle blood flow is not reduced in humans during moderate exercise and heat stress. *J.Appl.Physiol.* 64: 649-657, 1988.
214. Sawka, M. N., W. A. Latzka, S. J. Montain, B. S. Cadarette, M. A. Kolka, K. K. Kraning II, and R. R. Gonzalez. Physiologic tolerance to uncompensable heat:

- Intermittent exercise, field vs laboratory. *Med.Sci.Sports Exerc.* 33: 422-430, 2001.
215. Sawka, M. N., A. J. Young, B. S. Cadarette, L. Levine, and K. B. Pandolf. Influence of heat stress and acclimation on maximal aerobic power. *Eur.J.Appl.Physiol.* 53: 294-298, 1985.
 216. Sawka, M. N., A. J. Young, W. A. Latzka, P. D. Neufer, M. D. Quigley, and KB. Pandolf. Human tolerance to heat strain during exercise: influence of hydration. *J.Appl.Physiol.* 73: 368-375, 1992.
 217. Schmidt, V. and K. Bruck. Effect of precooling maneuver on body temperature and exercise performance. *J.Appl.Physiol.:Respirat.Environ.Exercise Physiol.* 50: 772-778, 1981.
 218. Secher, N. H., J. P. Clausen, K. Klausen, I. Noer, and J. Trap-Jensen. Central and regional circulatory effects of adding arm exercise to leg exercise. *Acta Physiol.Scand.* 100: 288-297, 1977.
 219. Sen Gupta, J., G. P. Dimri, and M. S. Malhotra. Metabolic responses of Indians during sub-maximal and maximal work in dry and humid heat. *Ergonomics* 20: 33-40, 1977.
 220. Shephard, R. J. Intensity, duration and frequency of exercise as determinants of the response to a training regime. *Int.Z.Angew.Physiol.Einschl.Arbeitsphysiol.* 26: 272-278, 1968.
 221. Shephard, R. J. The maximum oxygen uptake: an international reference standard of cardiorespiratory fitness. *Bull.W.H.O.* 38: 757-764, 1968.
 222. Shephard, R. J., C. Allen, and O. Bar-Or. The working capacity of Toronto schoolchildren: part I. *Can.Med.Assoc.J.* 100: 560-566, 1969.
 223. Shephard, R. J., E. Bouhlel, H. Vandewalle, and H. Monod. Muscle mass as a factor limiting physical work. *J.Appl.Physiol.* 64: 1472-1479, 1988.

224. Shibolet, S., M. C. Lancaster, and Y. Danon. Heat stroke: A review. *Aviat.Space Environ.Med.* 47: 280-301, 1976.
225. Shvartz, E. and D. Benor. Heat strain in hot and humid environments. *Aerosp.Med.* 43: 852-855, 1972.
226. Sidney, K. H. and R. J. Shephard. Maximum and submaximum exercise tests in men and women in the seventh, eighth, and ninth decades of life. *J.Appl.Physiol.* 43(2): 280-287, 1977.
227. Sills, F. D. and V. E. O'Riley. Comparative effects of rest, exercise, and cold spray upon performance in spot running. *Res.Q.* 27: 217-219, 1956.
228. Simmons, R. and R. J. Shephard. Measurement of cardiac output in maximum exercise. Application of an acetylene rebreathing method to arm and leg exercise. *Int.Z.Angew.Physiol.Einschl.Arbeitsphysiol.* 29: 159-172, 1971.
229. Skidmore, R., J. A. Gutierrez, V. Guerriero JR, and K. C. Kregel. HSP70 induction during exercise and heat stress in rats: role of internal temperature. *Am.J.Physiol.* 268: R92-R97, 1995.
230. Skinner, J. S., O. Bar-Or, V. Bergsteinova, C. W. Bell, D. Royer, and E. R. Buskirk. Comparison of continuous and intermittent tests for determining maximal oxygen intake in children. *Acta Paediatr.Scand.[Suppl.]* 217: 24-28, 1971.
231. Skranc, O., V. Havel, and K. Bartak. A comparison of work capacity measured by graded step-test and on a bicycle ergometer. *Ergonomics* 13: 675-683, 1970.
232. Sloniger, M. A., K. J. Cureton, D. I. Carrasco, B. M. Prior, D. A. Rowe, and R. W. Thompson. Effect of the slow-component rise in oxygen uptake on $\dot{V}O_{2max}$. *Med.Sci.Sports Exerc.* 28: 72-78, 1996.
233. Smith, J. H., S. Robinson, and M. Percy. Renal Responses to exercise heat and dehydration. *J.Appl.Physiol.* 4: 659-665, 1952.

234. Smolander, J., P. Kolari, O. Korhonen, and R. Ilmarinen. Aerobic and anaerobic responses to incremental exercise in a thermoneutral and a hot dry environment. *Acta Physiol.Scand.* 128: 15-21, 1986.
235. Smolander, J. and V. Louhevaara. Effect on heat stress on muscle blood flow during dynamic handgrip exercise. *Eur.J.Appl.Physiol.* 65: 215-220, 1992.
236. Squires, R. W. and E. R. Buskirk. Aerobic capacity during acute exposure to simulated altitude, 914 to 2,286 meters. *Med.Sci.Sports Exerc.* 14: 36-40, 1982.
237. Stamford, B. A. Maximal oxygen uptake during treadmill walking and running at various speeds. *J.Appl.Physiol.* 39: 386-389, 1975.
238. Stanley, D. C., W. J. Kraemer, R. L. Jr. Howard, L. E. Armstrong, and C. M. Maresh. The effects of hot water immersion on muscle strength. *J.Strength Cond.Res.* 8: 134-138, 1994.
239. Stenberg, J., P.-O. Astrand, B. Ekblom, B. Royce, and B. Saltin. Hemodynamic response to work with different muscle groups, sitting and supine. *J.Appl.Physiol.* 22: 61-70, 1967.
240. Strandell, T. and J. T. Shepherd. The effect in humans of increased sympathetic activity on the blood flow to active muscle. *Acta Med.Scand.[Suppl.]* 472: 146-167, 1967.
241. Taylor, C. Some properties of maximal and submaximal exercise with reference to physiological variation and the measurement of exercise tolerance. *Am.J.Physiol.* 142: 200-212, 1944.
242. Taylor, C. R. and C. P. Lyman. Heat storage in running antelopes: Independence of brain and body temperatures. *Am.J.Physiol.* 222: 114-117, 1972.
243. Taylor, C. R. and V. J. Rowntree. Temperature regulation and heat balance in running cheetahs: A strategy for sprinters? *Am.J.Physiol.* 224: 848-851, 1973.

244. Taylor, H. L., E. Buskirk, and A. Henschel. Maximal oxygen uptake as an objective measure of cardiorespiratory performance. *J.Appl.Physiol.* 8: 73-80, 1955.
245. Taylor, H. L., Y. Wang, L. Rowell, and G. Blomqvist. The standardization and interpretation of submaximal and maximal tests of working capacity. *Pediatrics* 32: 703-722, 1963.
246. Taylor, W. F., J. M. Johnson, D. O'Leary, and M. K. Park. Effect of high local temperature on reflex cutaneous vasodilation. *J.Appl.Physiol.* 57: 191-196, 1984.
247. Tenaglia, S. A., T. M. McLellan, and P. P. Klenrou. Influence of menstrual cycle and oral contraceptives on tolerance to uncompensable heat stress. *Eur.J.Appl.Physiol.* 80: 76-83, 1999.
248. Thauer, R. Circulatory adjustments to climatic requirements. In Hamilton, W. F. and P. Dow, eds., *Handbook of Physiology. Section : Circulation.* Washington, D.C., American Physiological Society. 1965, 1921-1966.
249. Thompson, J. M., J. A. Stone, A. D. Ginsburg, and P. Hamilton. O₂ transport during exercise following blood reinfusion. *J.Appl.Physiol.* 53: 1213-1219, 1982.
250. Thompson, J. M., J. A. Stone, A. D. Ginsburg, and P. Hamilton. The effects of blood reinfusion during prolonged heavy exercise. *Can.J.Appl.Sport Sci.* 8: 72-78, 1983.
251. Upfold, J. B., M. S. R. Smith, and M. J. Edwards. Maternal hyperthermia and its effects on embryonic and fetal brain development. In Hales, J. R. S. and D. A. B. Richards, eds., *Heat Stress: Physical Exertion and Environment.* Amsterdam, Elsevier Science Publishers B.V. (Biomedical Division). 1987, 303-311.

252. Van Der Linde, A., A. J. Kielblock, D. A. Rex, and S. E. Terblanche. Diagnostic and prognostic criteria for heat stroke with special reference to plasma enzyme and isoenzyme release patterns. *Int.J.Biochem.* 24: 477-485, 1992.
253. Wade, O. L. and J. M. Bishop. Cardiac output and regional blood flow. Oxford, Blackwell. 1962, 99-107.
254. Walters, T. J., K. L. Ryan, L. M. Tate, and P. A. Mason. Exercise in the heat is limited by critical internal temperature. *J.Appl.Physiol.* 89: 799-806, 2000.
255. Welch, H. G. and P. K. Pedersen. Measurement of metabolic rate in hyperoxia. *J.Appl.Physiol.:Respirat.Environ.Exercise Physiol.* 51: 725-731, 1981.
256. Wenger, C. B., M. F. Roberts, J. A. J. Stolwijk, and E. R. Nadel. Forearm blood flow during body temperature transients produced by leg exercise. *J.Appl.Physiol.* 38: 58-63, 1975.
257. Wenger, H. A. and G. J. Bell. The interactions of intensity, frequency and duration of exercise training in altering cardiorespiratory fitness. *Sports Med.* 3: 346-356, 1986.
258. White, H. L. and D. Rolf. Effects of exercise and some other influences on the renal circulation in man. *Am.J.Physiol.* 152: 505-516, 1948.
259. Williams, C. G., A. G. Bredell, C. H. Wyndham, N. B. Strydom, J. F. Morrison, J. Peter, P. W. Fleming, and J. S. Ward. Circulatory and metabolic reactions to work in the heat. *J.Appl.Physiol.* 17: 625-638, 1962.
260. Wyndam, C. H., N. B. Strydom, W. P. Leary, and C. G. Williams. Studies of the maximum capacity of men for physical effort part I. A comparison of methods of assessing the maximum oxygen intake. *Int.Z.Angew.Physiol.Einschl.Arbeitsphysiol.* 22: 285-295, 1966.
261. Wyndam, C. H., N. B. Strydom, A. J. Van Rensburg, and G. G. Rogers. Effects on maximal oxygen intake of acute changes in altitude in a deep mine. *J.Appl.Physiol.* 29: 552-555, 1970.

262. Wyndham, C. H., N. B. Strydom, J. S. Maritz, J. F. Morrison, J. Peter, and Z. U. Potgieter. Maximum oxygen intake and maximum heart rate during strenuous work. *J.Appl.Physiol.* 14: 927-936, 1959.
263. Young, D. R., R. Mosher, P. Erve, and H. Spector. Body temperature and heat exchange during treadmill running in dogs. *J.Appl.Physiol.* 14: 839-843, 1959.

APPENDIX A
SCALES OF PERCEPTUAL MEASURES

Perceived Exertion Scale

6	
7	VERY, VERY LIGHT
8	
9	VERY LIGHT
10	
11	FAIRLY LIGHT
12	
13	SOMEWHAT HARD
14	
15	HARD
16	
17	VERY HARD
18	
19	VERY, VERY HARD
20	

Pain Intensity Scale

- | | |
|----|---|
| 0 | NO PAIN AT ALL |
| ½ | VERY FAINT PAIN (just noticeable) |
| 1 | WEAK PAIN |
| 2 | MILD PAIN |
| 3 | MODERATE PAIN |
| 4 | SOMEWHAT STRONG PAIN |
| 5 | STRONG PAIN |
| 6 | |
| 7 | VERY STRONG PAIN |
| 8 | |
| 9 | |
| 10 | EXTREMELY INTENSE PAIN
(almost unbearable) |
| • | UNBEARABLE PAIN |

Scale of Thermal Sensation

- | | |
|---|--------------------|
| 1 | NEUTRAL |
| 2 | WARM |
| 3 | HOT |
| 4 | VERY HOT |
| 5 | AS HOT AS POSSIBLE |

Scale of Comfort Sensation

- | | |
|---|------------------------------|
| 1 | COMFORTABLE |
| 2 | SLIGHTLY UNCOMFORTABLE |
| 3 | UNCOMFORTABLE |
| 4 | VERY UNCOMFORTABLE |
| 5 | AS UNCOMFORTABLE AS POSSIBLE |

Motivation to Continue Scale

HOW STRONG IS YOUR MOTIVATION TO CONTINUE?

- | | |
|---|-------------|
| 1 | VERY WEAK |
| 2 | WEAK |
| 3 | AVERAGE |
| 4 | STRONG |
| 5 | VERY STRONG |

APPENDIX B

INSTRUCTIONS FOR THE SCALES OF PERCEPTUAL MEASURES

Perceived Exertion Scale Instructions

The scale you see before you contains numbers from 6 to 20. We want you to try to estimate how physically stressful you feel the exercise to be; that is, we want you to rate the degree of perceived exertion you feel. By perceived exertion we mean the total amount of exertion and physical fatigue, combining all sensations and feelings of physical stress, effort, and fatigue. Don't concern yourself with any one factor such as leg pain or shortness of breath, but try to concentrate on your total, inner feeling of exertion. Try to estimate as honestly and objectively as possible. Don't underestimate the degree of exertion you feel, but don't overestimate it either. Just try to estimate it as accurately as possible.

Looking at the scale before you, the number 6 corresponds with no exertion at all; it is basically sitting at rest just like you are now. Note that the verbal expressions begin with "very, very light", and most people would consider this level just noticeable. The verbal title, "very, very hard" (the number 19) refers to work that is extremely heavy. For most people this will be equal to the heaviest or most stressful physical exercise they have ever done, basically as hard as you have ever pushed yourself during a race or a run. The numerical scale will allow you to report your perceived exertion. Feel free to use any number on the scale.

During an actual data collection session, you will have the mouthpiece in your mouth, which will prevent you from speaking. When you are prompted for your perceived exertion, we will present you with the scale so that you can indicate your number by holding up that number of fingers. The numbers 6-10 correspond to 1-5

fingers (1 finger means 6, 5 fingers mean 10). The numbers 11-15 and 16-20 also correspond to 1-5 fingers (1 finger means 11 or 16 and 5 fingers mean 15 or 20).

During the test, a test administrator will read back to you're your selected number to clarify your choice. For example if you hold up 3 fingers when asked for RPE (your perceived exertion), the administrator may say 13. If that is correct, give thumbs up, if not shake your hand and the administrator will try either the number 8 or 18. This hand signaling will be practiced at rest prior to the test. Once the initial number during the test is established, the administrator will hardly ever associate the number of fingers you hold up with the wrong perception of effort.

Do you have any questions?

Pain Intensity Scale Instructions

You are about to undergo an exercise test. The scale before you contains the numbers 0-10. You will use this scale to assess perceptions of pain in your legs during the exercise test. In this context, Pain is defined as the intensity of hurt that you feel. Do not underestimate or overestimate the degree of hurt you feel, just try to estimate it as honestly and objectively as possible.

The numbers on the scale represent a range of pain intensity from “very faint pain” (number ½) to “extremely intense pain - almost unbearable” (number 10). When you feel no pain in your legs, you should respond with the number 0. If your legs feel extremely strong pain that is almost unbearable, you should respond with the number 10. If the pain is greater than 10, respond with the number that represents the pain intensity you feel in relation to 10. For example, if the pain is twice as intense as 10 give the number 20.

Repeatedly during the test you will be asked to rate the feelings of pain in your legs. When rating these pain sensations, be sure to attend only to the specific sensations in your legs and not report other pains you may be feeling (e.g. chest pain).

It is very important that your ratings of pain intensity reflect only the degree of hurt you are feeling in your legs during exercise. Do not use your ratings as an expression of fatigue (i.e., inability of the muscle to produce force).

During an actual data collection session, you will have the mouthpiece in your mouth, which will prevent you from speaking. When you are prompted for your leg pain, we will present you with the scale so that you can indicate your number by holding up that number of fingers. The numbers 1-5 correspond to 1-5 fingers (1 finger means 1, 5 fingers mean 5). The numbers 5-10 also correspond to 1-5 fingers (1 finger means 6 and 5 fingers mean 10). During the test, a test administrator will read back to you're your selected number to clarify your choice. For example if you hold up 3 fingers when asked for leg pain, the administrator may say 3. If that is correct, give thumbs up, if not shake your hand and the administrator will try the number 8. Zero should be signaled by making a zero with your index finger and thumb and ½ by making a U with your index finger and thumb. For ratings higher than 10, raise your fist, and the administrator will ask you after the test what number the fist meant. This hand signaling will be practiced at rest prior to the test. Once the initial number during the test is established, the administrator will hardly ever associate the number of fingers you hold up with the wrong perception of effort.

In summary, you will be asked to: 1) provide pain intensity ratings in your legs only; 2) give ratings as accurately as possible; and 3) not under- or overestimate the pain, but simply rate your pain honestly. You should use the verbal expressions to help rate your perceptions.

Do you have any questions?

Scale of Thermal Sensation Instructions

You are about to undergo an exercise test. The scale before you contains the numbers 1-5. You will use this scale to assess your whole body thermal sensation during and prior to the exercise test. By whole body thermal sensation we mean how

cold or hot you are feeling over your entire body. Do not underestimate or overestimate your degree of thermal sensation, just try to estimate it as honestly and objectively as possible.

The numbers represent a range of thermal sensation from “neutral” (number 1) to “as hot as possible” (number 5). If you are neither hot nor cold during or prior to the exercise, respond with the number 1. If you are as hot as possible during or prior to the exercise, respond with the number 5.

Repeatedly prior to and during the test you will be asked to rate your thermal sensation. It is very important that your ratings of thermal sensation reflect only the degree of thermal sensation you are feeling prior to and during the exercise test.

During an actual data collection session, you will have the mouthpiece in your mouth, which will prevent you from speaking. When you are prompted for your thermal sensation, we will present you with the scale so that you can indicate your number by holding up that number of fingers. The numbers 1-5 correspond to 1-5 fingers (1 finger means 1, 5 fingers mean 5). During the test, a test administrator will read back to you're your selected number to clarify your choice. This hand signaling will be practiced at rest prior to the test.

Do you have any questions?

Scale of Comfort Sensation Instructions

You are about to undergo an exercise test. The scale before you contains the numbers 1-5. You will use this scale to assess your level of comfort during and prior to the exercise test. By comfort we mean how comfortable or uncomfortable you feel over your whole body both internally and externally. Do not underestimate or overestimate your degree of comfort, just try to estimate it as honestly and objectively as possible.

The numbers represent a range of thermal sensation from “comfortable” (number 1) to “as uncomfortable as possible” (number 5). If you are comfortable

during or prior to the exercise, respond with the number 1. If you are as uncomfortable as possible during or prior to the exercise, respond with the number 5.

Repeatedly prior to and during the test you will be asked to rate your comfort sensation. It is very important that your ratings of comfort sensation reflect only the degree of comfort sensation you are feeling prior to and during the exercise test. You should rate your overall comfort level and not confuse it with your sensation of heat and leg pain since it is possible that you could be hot and comfortable or without leg pain and uncomfortable.

During an actual data collection session, you will have the mouthpiece in your mouth, which will prevent you from speaking. When you are prompted for your comfort level, we will present you with the scale so that you can indicate your number by holding up that number of fingers. The numbers 1-5 correspond to 1-5 fingers (1 finger means 1, 5 fingers mean 5). During the test, a test administrator will read back to you're your selected number to clarify your choice. This hand signaling will be practiced at rest prior to the test.

Do you have any questions?

Motivation to Continue Scale Instructions

You are about to undergo an exercise test. The scale before you contains the numbers 1-5. You will use this scale to assess the intensity of your motivation to continue exercising during the exercise test. Do not underestimate or overestimate your degree of motivation to continue exercising, just try to estimate it as honestly and objectively as possible.

The numbers represent the intensity of your motivation to continue exercising ranging from "very weak" (number 1) to "very strong" (number 5). If you are very weakly motivated to continue the exercise, respond with the number 1. If you are very strongly motivated to continue the exercise, respond with the number 5. However, do

not stop exercising merely because your motivation to continue has decreased; continue to exercise until you physically cannot go any longer.

Repeatedly during the test you will be asked to rate your motivation to continue exercising. It is very important that your ratings of motivation to continue reflect only the degree of motivation you are feeling to continue to exercise during the exercise test. Do not use your ratings as an expression of fatigue (i.e., physical inability to continue exercising) since you may be motivated to continue exercising despite being tired or unmotivated to continue exercising despite feeling no fatigue.

During an actual data collection session, you will have the mouthpiece in your mouth, which will prevent you from speaking. When you are prompted for your motivation to continue, we will present you with the scale so that you can indicate your number by holding up that number of fingers. The numbers 1-5 correspond to 1-5 fingers (1 finger means 1, 5 fingers mean 5). During the test, a test administrator will read back to you're your selected number to clarify your choice. This hand signaling will be practiced at rest prior to the test.

Do you have any questions?