THE EFFECT OF ACTIVE WARM-UP AND STRETCHING ON ANAEROBIC PERFORMANCE

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Bachelor of Science, University of Victoria, 1985

A THESIS SUBMITTED IN PARTIAL FULFILLMENT
OF THE REQUIREMENTS FOR THE DEGREE OF
MASTER OF ARTS

in the School of Physical Education

We accept this thesis as conforming
to the required standard

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ABSTRACT

The purpose of this study was to determine the effect of active warm-up, to a 1 °C increase in core temperature, and/or stretching, on selected physiological and performance variables during and following a 45-second, all-out cycle ergometer test. Ten male subjects performed the criterion cycle ergometer test (ACT) on four occasions, following active warm-up (AWU), stretching (S), active warm-up and stretching (AWUS), or no warm-up (C) protocols. Oxygen consumption was measured during active warm-up and the ACT, and heart rates and blood lactates were taken, 15 seconds and 3 minutes, post-ACT.

There were no significant differences in $\dot{V}0_2$ between AWU and C conditions over 15, 30, or 45 seconds of the ACT. Post-ACT heart rates were significantly higher following the AWU versus C protocol but differences in blood lactates were not significant.

Following S, mean $\dot{V}0_2$ was not different between the S and C groups over 15 or 30 seconds of the ACT but was significantly higher for the C condition over 45 seconds. Heart rates and blood lactates were not significantly different.

Mean $\dot{V}0_2$ over 15, 30, and 45 seconds of the ACT and blood lactates were not different between AWUS and C conditions. Heart rates 15 seconds post-ACT were higher following the AWUS protocol. Despite differences in selected physiological variables, there were no significant differences in peak power or total work, which suggests that active warm-up and/or stretching does not influence performance of a criterion task which challenges anaerobic capacity.

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Acknowledgements

I would like to thank my supervisor, Dr. Howard A. Wenger, for his guidance, patience, and encouragement through this program. I sincerely appreciate his wonderful sense of humour, and his ability to "flog" my thesis and make me feel good about myself at the same time. I learned more about life and myself than I had planned - thanks to Howie and Jan for their support through the process!

Thank you to Dr. David Docherty and Dr. Frank Robinson for participating as members of my advisory committee, and a special thank you to Dr. Richard Backus for his interest, time, and energy in helping me develop a muscle temperature thermocouple protocol.

A heartfelt thank you to all my brave and courageous subjects: Ben, Rob, Simon, Bruce, Alex, Carson, David, Bob, Victor, and Jim. We made it! Thanks for sticking with the program.

A very special thank you to Wendy, Lisa, Kathy, P.J., my fellow graduate students, and my undergrad friends for helping me with preparation and testing in the Sport and Fitness Centre, and for encouraging and supporting me through the darkest times. Mark from the Computer Lab - I couldn't have done it without your help!

Finally, to my friend Gladys, thanks for all the Dim Sum lunches and for just being you!

DEDICATION

This thesis is dedicated to the memory of my father, who shared his love of knowledge with us and taught us to believe we could be all that we wanted to be, and to my mother, whose unconditional love and support will give me courage always.

INTRODUCTION

With the onset of dynamic exercise, the thermal energy balance of resting muscle is upset and heat is produced in proportion to the intensity of exercise (Nadel, 1983). As muscle temperature increases, the temperature gradient between working muscle and surrounding tissue changes, enhancing heat exchange by conduction to the surrounding tissues (Werner & Buse, 1988) and to the blood (Mitchell, 1977). Through convection, the heat produced by the exercising muscle is carried away through venous return to the heart and body core, elevating body core temperature (Fortney & Vroman, 1985).

A number of metabolic and physiological changes occur when body and muscle temperature are increased (warm-up) that may enhance athletic performance. Astrand and Rodahl (1977) reported that increased muscle temperature is associated with decreased intramuscular resistance, resulting in more energy for external work. An increase in blood temperature is associated with increased dissociation of oxygen from hemoglobin and myoglobin, resulting in greater aerobic metabolism (Barcroft & King, 1909). Warm-up (WU) is also thought to decrease the time for initial metabolic adjustment to heavy work, resulting in a lower oxygen deficit (Gutin et al., 1976). Ruch et al. (1966) have reported that WU can increase the velocity of nerve conduction and Cureton (1947) has reported that WU opens more capillaries in the muscles, enhancing exchange between muscles and blood.

Warm-up is used by athletes on the premise that performance will be enhanced. A rationale for the use of warm up prior to performance is its effect on aerobic metabolism. Several researchers (Asmussen & Boje, 1945; Martin et al., 1975; Ingjer & Stromme,

1979) have suggested that an acceleration of aerobic processes occurs in the warm up condition. An increase in the contribution of energy via aerobic pathways at the start of maximal tasks could theoretically benefit performance by helping to reserve some of the anaerobic sources of ATP until needed later in the task (Andzel & Gutin, 1976). It would appear from the results of these and several other studies that WU may be physiologically warranted as an ergogenic aid to performance of maximal or supramaximal effort (relative to $\dot{V}0_{2max}$) (Craig & Froehlich, 1968; Barnard et al., 1973; Martin et al., 1975; Gutin et al., 1976; Chwalbinska-Moneta & Hanninen, 1989). However, research is equivocal as to improvements in anaerobic performance as a result of unquantified WU.

In order to determine the effect of WU on anaerobic performance, many variables have been considered: the criterion test (CT) of performance (Thompson, 1958; Dickinson et al., 1979); the type of WU used, ie. whether it is identical to the criterion test, specific to selected muscles groups or general (de Vries, 1958); the intensity and duration of the WU (Inbar & Bar-Or, 1975); the length of the rest interval between the CT and WU (Genovely & Stamford, 1982); the fitness level of the participants in the study (Knowlton et al., 1978); whether or not the WU was active, encorporating muscular activity to increase muscle and/or body temperature (AWU), or passive, using hot water baths or radio diathermy to increase temperatures (PWU) (Dolan et al., 1985); and the actual amount of body warming, as measured by muscle temperature (Tm) or body core temperature (Tc) (Franks, 1983; Sargeant, 1983; Sargeant, 1987). All of these factors, alone or in combination, have the potential to affect the degree of WU, the physiological responses to both the WU and the CT, and the magnitude of the CT itself.

In the majority of studies which reported on the impact of WU on work

performance, one or more of the above variables have either not been reported or controlled, making the reproduction of the test protocol impossible (Pacheco, 1957; Clarke et al., 1958; Thompson, 1958; Howard et al., 1966; Pyke, 1967; Barnard et al., 1973; Binkhorst et al., 1977).

The use of AWU as a preliminary activity has been recommended (Michael et al., 1957; de Vries, 1958; Thompson, 1958; De Bruyn-Prevost & Lefebvre, 1980). Increasing the temperature of the specific muscles involved in the criterion test is advantageous for shortening the metabolic and circulatory adjustments during performance (Asmussen & Boje, 1945; Busuttil & Ruhling, 1977; Shellock, 1983) and an optimal intensity and duration of WU has been shown to be necessary to produce the physiological changes required for optimal anaerobic performance (Robinson & Heron, 1924; Lotter, 1959; Karlsson et al., 1975; Stamford et al., 1978; Gutin et al., 1981).

To quantify and standardize the effect of WU, Tm and/or Tc must be measured and/or controlled. AWU has been shown to produce increases in Tm ranging from two to four °C (Martin et al., 1975) and in Tc ranging from 0.9 °C (Asmussen & Boje, 1945) to 1.3 °C (Ingjer & Stromme, 1979), depending on the duration and intensity of WU. Two studies have manipulated or controlled Tm or Tc during AWU and observed the changes in the physiological response to, and/or performance of, a specific anaerobic criterion test. Bergh and Ekblom (1979) manipulated Tm and Tc by passively cooling the legs in cold water to produce Tc and Tm of 35-37 and 30-35 °C respectively, and actively warming up by intermittent cycle ergometer exercise to produce Tc and Tm of 36-38 and 36-39 °C respectively. Performances (maximal muscle strength, vertical jump height, sprint performance, and peak power output) were positively related to Tm; as Tm increased,

performance also increased. However, the intensity and duration of the WU protocol was not reported.

Ingjer and Stromme (1979) examined the effect of AWU (cycling at 50-60% $\dot{V}O_{2max}$) and PWU (immersion in 40 °C water) on a treadmill run, at three percent grade, for four minutes at a speed determined to produce 100% $\dot{V}O_{2max}$. Each WU was performed until a core temperature of 38.35 °C was reached. This normally required about 20 to 25 minutes and the mean increase in Tc was 1.3 °C. During the criterion task, significantly higher oxygen uptake, lower lactate, and higher blood pH values were found when the work was preceded by AWU as compared with PWU or no WU. Although performance was not measured, it was concluded that the physiological effects of a thorough AWU may be of substantial benefit to athletic performance.

Although the studies which manipulated or controlled the increase in Tm or Tc support the hypothesis that AWU could be beneficial to anaerobic performance, no studies have examined the effect of warm up on the physiological response to and actual performance of an activity which taxes the anaerobic capacity (45 seconds).

Stretching is performed prior to vigorous activity on the premise that it also enhances performance. Stretching the agonist muscle will cause the agonist muscle to relax, increasing the potential range of motion of that muscle and improving its ability to produce force. While its benefits in the potential reduction and prevention of injury are clinically documented (Nicholas, 1970; Shellock, 1983), its effects on anaerobic performance are not. Harris (1969), Cotten and Waters (1970), and Cornelius et al.

(1988), have reported the effect of stretching on flexibility, but few researchers have documented the effect of stretching on dynamic performance.

Pacheco (1957) found vertical jump heights greater following a stretching program for hip and leg muscles, while Pyke (1967) found no significant differences between experimental and control groups on cricket ball throw, jump reach, 60-yard dash and a 10-second bicycle ergometer test of leg speed following a general flexibility program. Pacheco (1957) used static stretches, held for 15 seconds, for a total of 3 minutes prior to criterion testing, while the general flexibility program used by Pyke (1967) was 12 repetitions each of backward double arm circling, standing trunk turns, and standing toe touches, ie. no held stretches, and the differences in protocol may account for the differences in results obtained.

Only two studies were found in which warm-up and a stretching program were used to determine their combined effect on the physiological response to and performance of an anaerobic criterion test of longer than few seconds duration. deVries (1958) incorporated a strength and stretch protocol prior to a variety of criterion swim styles and found this program was beneficial to breast-stroke and dolphin swim specialists but detrimental to free stylers, and Howard et al. (1966) found a 10-15 minute program of jogging, calisthenics and stretching had no effect on heart rate during a number of runs at various distances. Due to the nature of the protocol used by deVries (1958) and the lack of description of the warm-up used by Howard et al. (1966), the influence of the stretch portion of the warm-up cannot be determined.

Statement of the problem

The purposes of this study were to examine the effect of active warm-up and/or preliminary stretching on: metabolic and circulatory responses to an anaerobic capacity test on a cycle ergometer, and the peak power and total work performed on an anaerobic capacity test on the cycle ergometer.

Research Questions

1. Is there a change in the physiological response to, and the performance of, an anaerobic criterion test following an active warm-up which elicits a 1.0 °C increase in Tc?

2. Is there a change in the physiological response to, and the performance of, an anaerobic criterion test following a stretching program?

3. Is there a change in the physiological response to, and the performance of, an anaerobic criterion test following both an active warm-up which elicits a 1.0 °C increase in Tc and a stretching program?

Operational Definitions

Active warm-up (AWU): A preliminary cycling activity which was designed to increase core temperature (Tc) by 1 °C. The intensity of the AWU was approximately 60-65% of pre-determined $V0_{2max}$.

Stretching program (S): Preliminary exercises to relax the muscle groups involved in the anaerobic criterion test (ACT) including the hip extensors, knee extensors, ankle plantar flexors, hip flexors, knee flexors, and ankle dorsi flexors.

Anaerobic criterion test (ACT): A 45-second, all-out sprint on a cycle ergometer at a load corresponding to 95 grams per kilogram body weight of the subject.

Peak power (PP): The highest average power output (watts) over a five second period, recorded during the ACT.

Total work (TW): The total work (kilojoules) completed during the 45 seconds of the ACT.

RESEARCH METHODS

Subjects

Ten males read and signed Informed Consent pre-screening forms (Appendix A), and volunteered to participate in the study. The sample was composed of athletes competing in rugby (n=3) and rowing (n=1), competitive triathletes (n=2), and physically active individuals in the School of Physical Education (n=4) at the University of Victoria (See Table 1).

The subjects reported to the University of Victoria Sport and Fitness Centre (Fitness Centre) five times during the course of the study. During the first testing session, maximal oxygen consumption ($\dot{V}O_{2max}$) was assessed on a cycle ergometer (Monark, model 868), and anthropometric measures of height, weight, and four skinfolds were taken. For the four following sessions, each subject underwent a different pre-ACT protocol: active warm-up (AWU), stretching (S), active warm-up and stretching (AWU & S), or no warm-up (C), with the order randomly assigned .

Testing sessions were separated by at least 48 hours, with a maximum of two sessions per week. Subjects reported to the Fitness Centre at the same time each testing day, to minimize the influence of the circadian rhythm on core temperature and other physiological variables (Winget et al., 1985). Subjects were asked to refrain from vigorous physical activity for 24 hours prior, and eating for three hours prior to reporting for testing.

Table 1. Physical characteristics of the subjects

Variable	n	x	SD	range
Age (years)	10	28.3	4.9	21-36
Height (cm)	10	181.2	6.7	167.2-187.9
Weight (kg)	10	78.5	8.2	63.5-88.5
%Body Fat	10	15.3	3.3	12.2-21.7
$\dot{V}0_{2max}$ ($mL \cdot kg^{-1} \cdot min^{-1}$)	10	52.1	5.0	46.1-62.9

EXPERIMENTAL PROCEDURES

$\dot{V}O_{2max}$ protocol

$\dot{V}O_{2max}$ was determined on a Monark friction-braked cycle ergometer using a continuous, incremental protocol (MacDougall et al., 1982). Expired air was collected by a two-way Rudolph valve and analyzed every 30 seconds using a Beckman metabolic measurement cart . The cart was calibrated before each test using primary standard gases and heart rate was recorded telemetrically every minute using a Sportester (model PE 3000) heart rate monitor.

$\dot{V}O_{2max}$ was achieved when two or more of the following criteria had been met: a plateau or decrease in oxygen consumption despite an increase in power output; a respiratory quotient which exceeded 1.1; the achievement of predicted maximum heart rate; or volitional fatigue. $\dot{V}O_{2max}$ was expressed as $L \cdot min$ and $mL \cdot kg^{-1} \cdot min^{-1}$. The power output corresponding to 60% of achieved relative $\dot{V}0_{2max}$ was the load used during the active warm-up.

Anthropometry

Height, weight, and the sum of four skinfolds: biceps, triceps, subscapular, and suprailiac, were taken to characterize the subjects and percent body fat was estimated by the method of Durnin & Wormersley (1974).

Pre-warmup Preparation

In a separate room, each subject recorded nude body weight (for calculating the resistance setting for the cycle ergometer), and inserted a sterile rectal probe (YSI Series 400, general purpose) to a depth of approximately eight centimeters. The subject then dressed in shorts and a T-shirt and reported back to the Fitness Centre where a sterile YSI tissue implantable thermocouple microprobe (Type IT-18) (microprobe), was inserted into the muscle of the right thigh to measure muscle temperature (Tm).

The microprobe was inserted at an angle of approximately 90 degrees into the lateral thigh (one to two centimeters from the mid-line) approximately 15-20 centimeters above the patella (Saltin et al., 1968). The probe was injected inside an 18-gauge, thin-walled, disposable monoject needle to a depth below the skin surface of approximately 3.5 centimeters. The needle was then withdrawn, leaving the probe in place in the leg. The muscle temperature probe was then taped in place with 3M Blenderm waterproof surgical tape. The rectal temperature and muscle temperature probes were connected to a YSI Electronic Telethermometer (model 46 Tuc) and a Bailey Instruments Telethermometer (model Bat. 8) respectively, and initial readings recorded.

The temperature probes were calibrated before use each testing day. After use, both rectal and muscle temperature probes were washed with disinfectant soap, rinsed with ethanol, then sterilized in a 10% germaphene solution for not less than 20 minutes.

Active Warm-Up Protocol

The ACT and AWU were performed on a Monark friction-braked cycle ergometer (model 868). Subjects commenced cycling at approximately 60% of pre-determined $\dot{V}O_{2max}$. Oxygen consumption and heart rates were monitored to confirm and maintain the specified intensity of warm-up which continued until Tc had increased 1.0 °C. Heart rate, Tc, and Tm were recorded every minute during the AWU. Upon reaching target Tc, a blood lactate sample was taken, and subjects immediately commenced the ACT protocol.

Stretching Protocol

Following preparation, subjects commenced a light warm-up on the cycle ergometer, at 60-70 revolutions per minute and a load not greater than one kilogram, for 2 minutes. Subjects then stretched the muscle groups which were used to perform the ACT: for pedal depression - hip extensors to a slight degree, knee extensors, and ankle flexors; for lifting the pedal - hip flexors, knee flexors, and ankle extensors (Weineck, 1986). For a full description of stretches used, see Appendix B. Each stretch was held statically for 20 seconds at the point where a light tension was felt in the muscle and adjusted when necessary as the muscle lengthened (Alter, 1990). One side of the body was alternated with the other for each stretch; each stretch was performed twice per side for a total of 40 seconds.

Immediately following cessation of the stretching protocol, the subject was seated on the cycle ergometer, the metabolic cart appartus was attached and the probes were plugged into their respective telethermometers; the subject then commenced the ACT protocol.

Anaerobic Criterion Test Protocol

Subjects pedalled for two minutes prior to the test, at a load not greater than 1.0 kilogram resistance, prior to a 45-second, all-out sprint on the cycle ergometer (Bar-Or, 1987). Immediately prior to the sprint, subjects were instructed to "pick up the pace" and commenced pedalling as fast as possible in order to overcome the initial resistance of the flywheel as the resistance was set. Resistance was set within one to two seconds, during which the words "ready, set, go" were given to cue the subjects. At the word "go", timing commenced and the subjects sprinted with all-out effort for 45 seconds. Resistance for the ACT was set at 0.095 kilograms per kilogram of body weight, which has been shown to elicit a higher peak power output than produced by the original 0.075 setting described by Bar-Or et al. (1977)(Lavoie et al., 1984) . Subjects were given verbal encouragement throughout the test and time checks were given at regular intervals during the sprint test. The last ten seconds were counted out loud to motivate the subjects.

The number of flywheel revolutions during the 45-second test was recorded. Oxygen consumption was measured every 15 seconds during the criterion test. Heart rate was recorded immediately prior to the test and at 15 seconds after the test. Finger-prick blood lactate samples were taken immediately before the test, immediately after the test, and three minutes post-test.

Upon completing the ACT, subjects remained seated on the cycle ergometer and continued unloaded pedalling until heart rates had returned to approximately 120 beats per minute. Once off the ergometer, subjects were asked to lie on a mat with feet up on a chair, and were given fruit juice.

Blood Sampling Protocol

A blood sample was collected by a technician immediately after the warm-up and/or stretching protocol and analyzed for lactate content to ensure that anaerobic lactic energy sources had not been utilized during warm-up. Blood samples for lactate analysis were also taken immediately before, immediately after, and three minutes post ACT. All blood samples were analyzed by a YSI Lactate Analyzer (model 23L). Samples were obtained by finger prick using a sterilized Auto-lancet after the finger was disinfected with an alcohol swab.

STATISTICAL ANALYSIS

Students' t-tests were used to determine if there were any statistically significant differences between conditions. The level of significance was set at $p < 0.05$.

RESULTS

Is there a change in the physiological response to, and the performance of, an anaerobic criterion test following an active warm up which elicits a 1.0 °C increase in Tc?

The mean time to increase Tc by 1 °C through AWU was 34.6 minutes and over this same time frame Tm rose by 3.2 °C (Fig. 1a). Core temperature and muscle temperature prior to the ACT following AWU were significantly higher than the C condition (Fig. 2 and 3) and remained elevated to the start of the ACT.

The mean $\dot{V}0_2$ showed no significant differences between AWU and C conditions over 15, 30, or 45 seconds of the anaerobic criterion test (Fig. 4). Heart rates measured 15 seconds post-ACT were significantly higher after the AWU protocol versus the C condition (Fig. 5), and differences in blood lactate, peak power and total work were not significant (Table 2, Table 3).

Is there a change in the physiological response to, and the performance of, an anaerobic criterion test following a stretching program?

The average time to complete the stretching program was 16 minutes. The S protocol did not produce a change in Tc and despite an increase in Tm of 0.6 °C, there were no significant differences in Tc or Tm between the S and C conditions (Fig. 2 & 3). Mean $\dot{V}0_2$ was not different between the two conditions over 15 or 30 seconds of the ACT but

was significantly higher for the C condition than the S condition over 45 seconds (Fig. 4). Heart rates, blood lactate, peak power and total work between the S and C condition were not significantly different (Table 2, Table 3).

Is there a change in the physiological response to, and the performance of, an anaerobic criterion test following both an active warm up which elicits a 1.0 °C increase in Tc and a stretching program?

After the AWU portion of the AWUS protocol, there was a 1.5 °C (46%) decrease in Tm and .4 °C (40%) decrease in Tc during the stretching protocol (Fig. 1b). Core temperature was not significantly different than the C condition prior to the ACT, but Tm was higher ($p<0.05$). Mean $\dot{V}0_2$ at 15,30, and 45 seconds of the ACT were not different between the two conditions (Fig. 4). Heart rates 15 seconds post-ACT were higher following the AWUS protocol than the C condition ($p<0.001$) (Fig. 5). There were no statistically significant differences in blood lactate, peak power, and work performed (Table 2, Table 3).

Figure 1a. Core temperature (Tc) -- -- --
and muscle temperature (Tm) --+-+--
during Active Warm-up (AWU),
prior to the
Anaerobic Criterion Test (ACT).

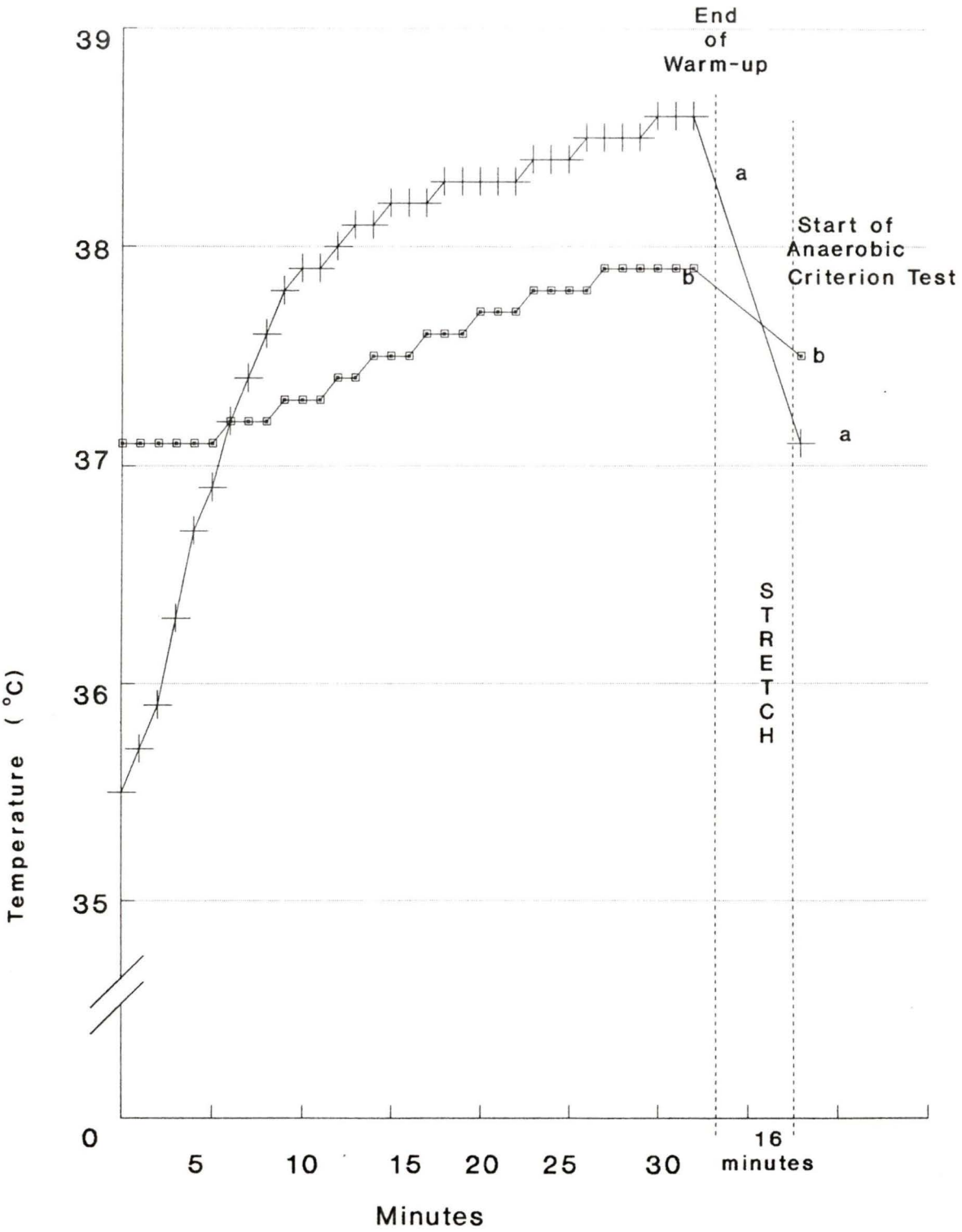

End
of
Warm-up
a
Start of
Anaerobic
Criterion Test
b
b
a
39
38
37
36
35
0
STRETCH
16
minutes
5
10
15
20
25
30
Minutes
Temperature (°C)

Figure 1b. Core temperature (Tc) -- -- --
and muscle temperature(Tm) --+--+--
during Active Warm-up and Stretch
(AWUS), prior to the Anaerobic
Criterion Test (ACT).

Paired letters show groups which are
significantly different ($p < 0.05$).

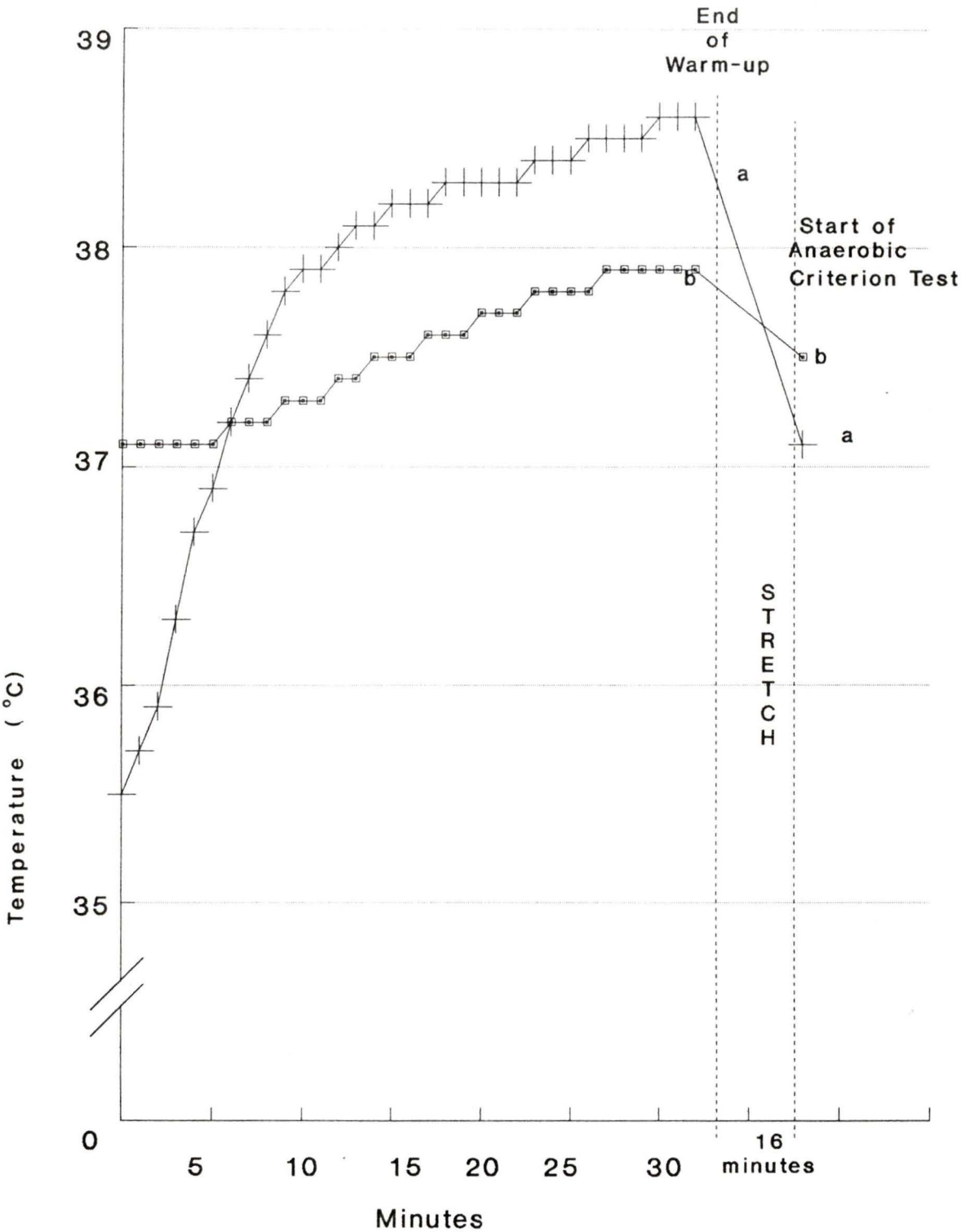

End
of
Warm-up
a
Start of
Anaerobic
Criterion Test
b
b
a
STRETCH
39
38
37
36
35
0
Temperature (°C)
5
10
15
20
25
30
16
minutes
Minutes

Figure 2. Core temperature (Tc) following the different conditions at the start of the Anaerobic Criterion Test (ACT).

Paired letters show groups which are significantly different (a $p < 0.001$; b $p < 0.005$; c < 0.05).

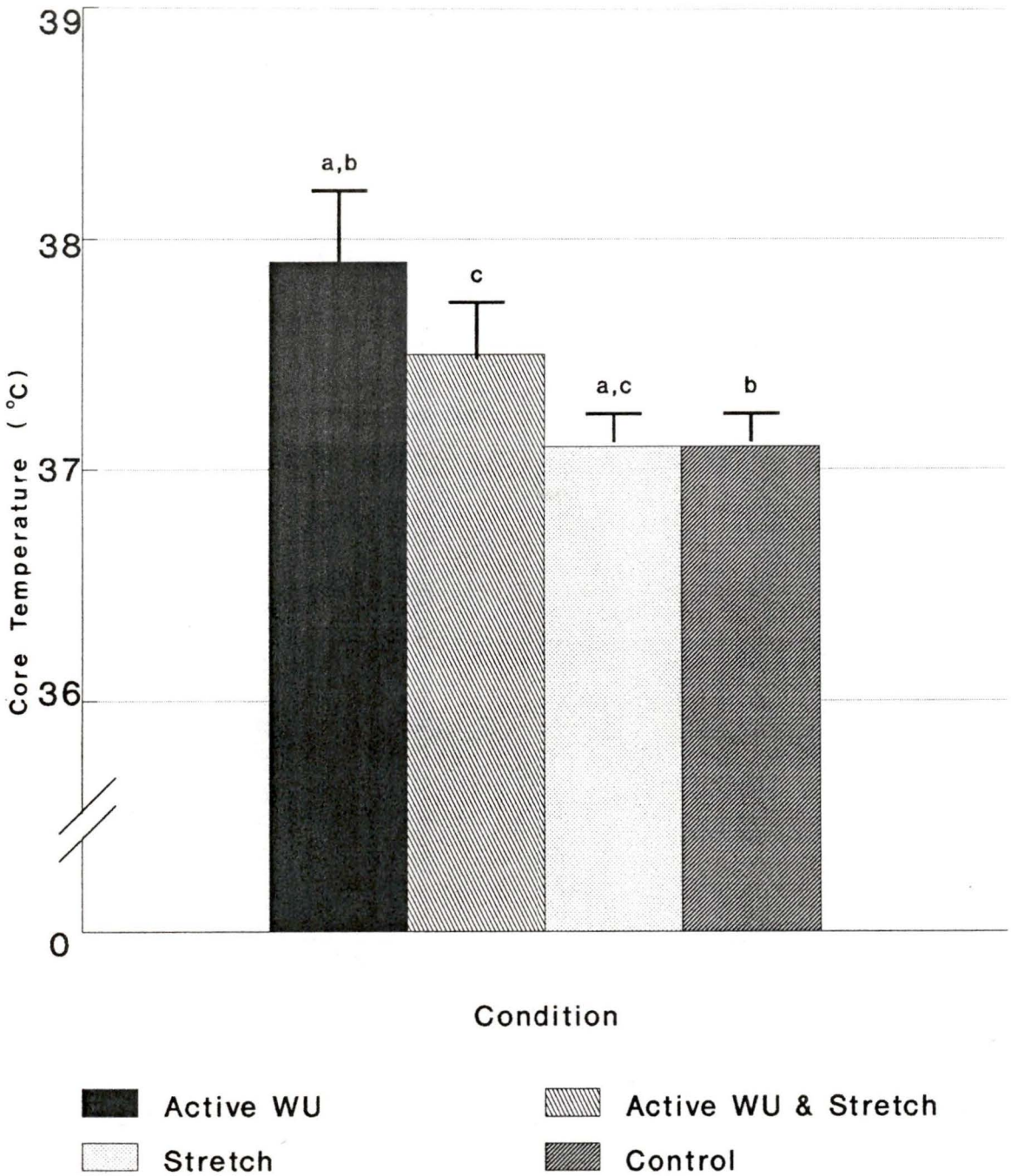

39
38
37
36
0
Core Temperature (°C)
a,b
c
a,c
b
Condition
Active WU
Active WU & Stretch
Stretch
Control

Figure 3. Muscle temperature (Tm) following the different conditions at the start of the Anaerobic Criterion Test (ACT).

Paired letters show groups which are significantly different (a,b,c $p < 0.05$).

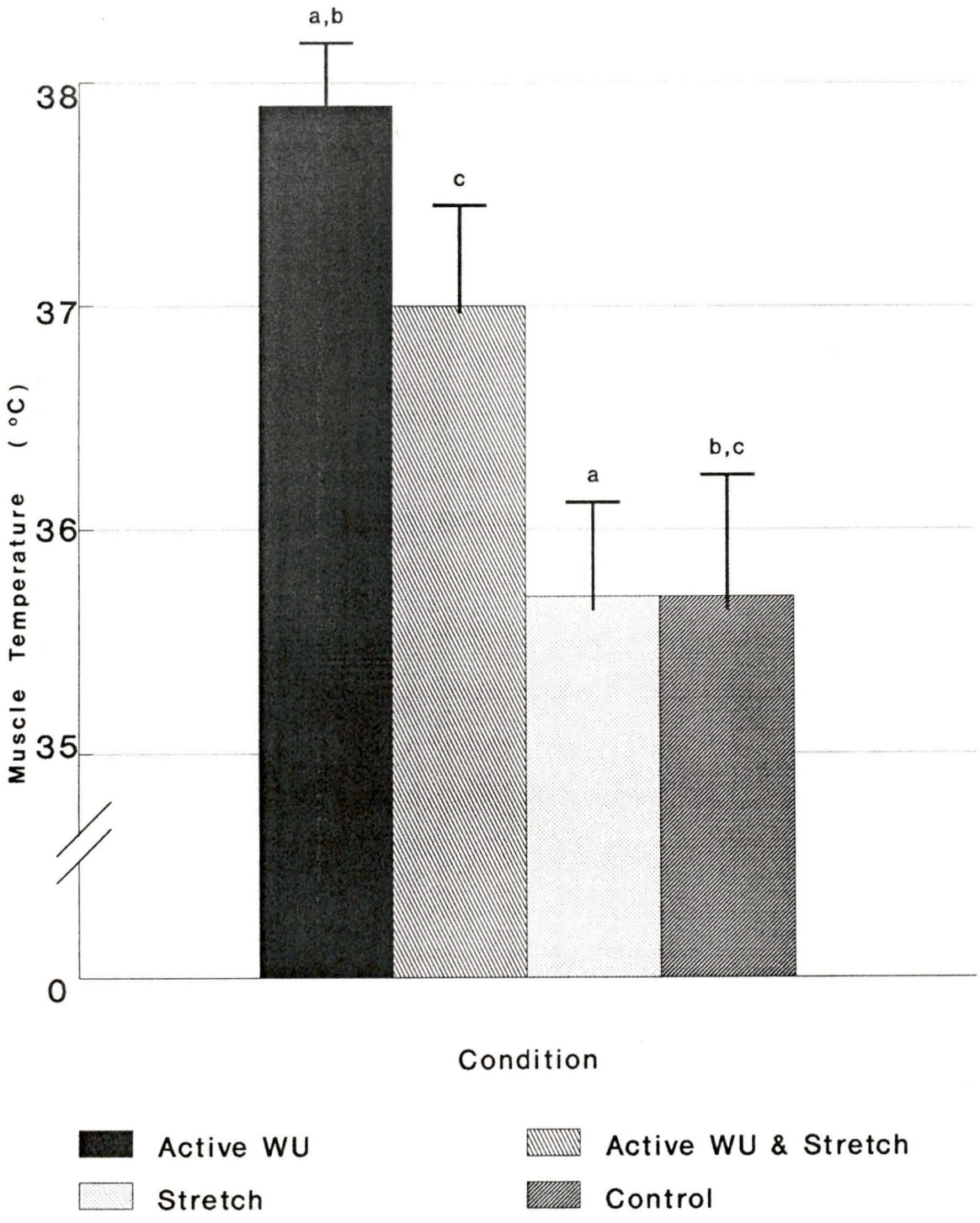

a,b
c
a
b,c
38
37
36
35
0
Muscle Temperature (°C)
Condition
Active WU
Active WU & Stretch
Stretch
Control

Table 2. Post-exercise Blood Lactate, Peak Power (PP) and Total Work (TW) during anaerobic criterion test (ACT) for each condition (x ± SD).

Condition	Lactate($mM \cdot L^{-1}$)	PP(W)	TW(kJ)
AWU	10.3 ± 1.7	891 ± 126	27.1 ± 3.2
AWUS	10.3 ± 2.0	915 ± 161	27.6 ± 3.6
S	10.8 ± 1.8	897 ± 140	28.0 ± 3.6
C	10.6 ± 1.9	862 ± 171	27.6 ± 3.3

Table 3. Work performed over 15, 30, and 45 seconds of the anaerobic criterion test (ACT) (x + SD)

Condition	Work-15 seconds	Work-30 seconds	Work-45 seconds
AWU	11.8 ± 1.7	20.4 ± 2.7	27.3 ± 1.1
AWUS	12.0 ± 1.7	20.7 ± 2.6	27.6 ± 3.6
S	12.0 ± 1.5	21.0 ± 2.3	28.0 ± 3.6
C	11.6 ± 1.6	20.4 ± 2.4	27.5 ± 3.3

Figure 4. Mean oxygen consumption over 15, 30, and 45 seconds of the Anaerobic Criterion Test (ACT).

Paired letters show groups which are significantly different (a,b $p < 0.05$).

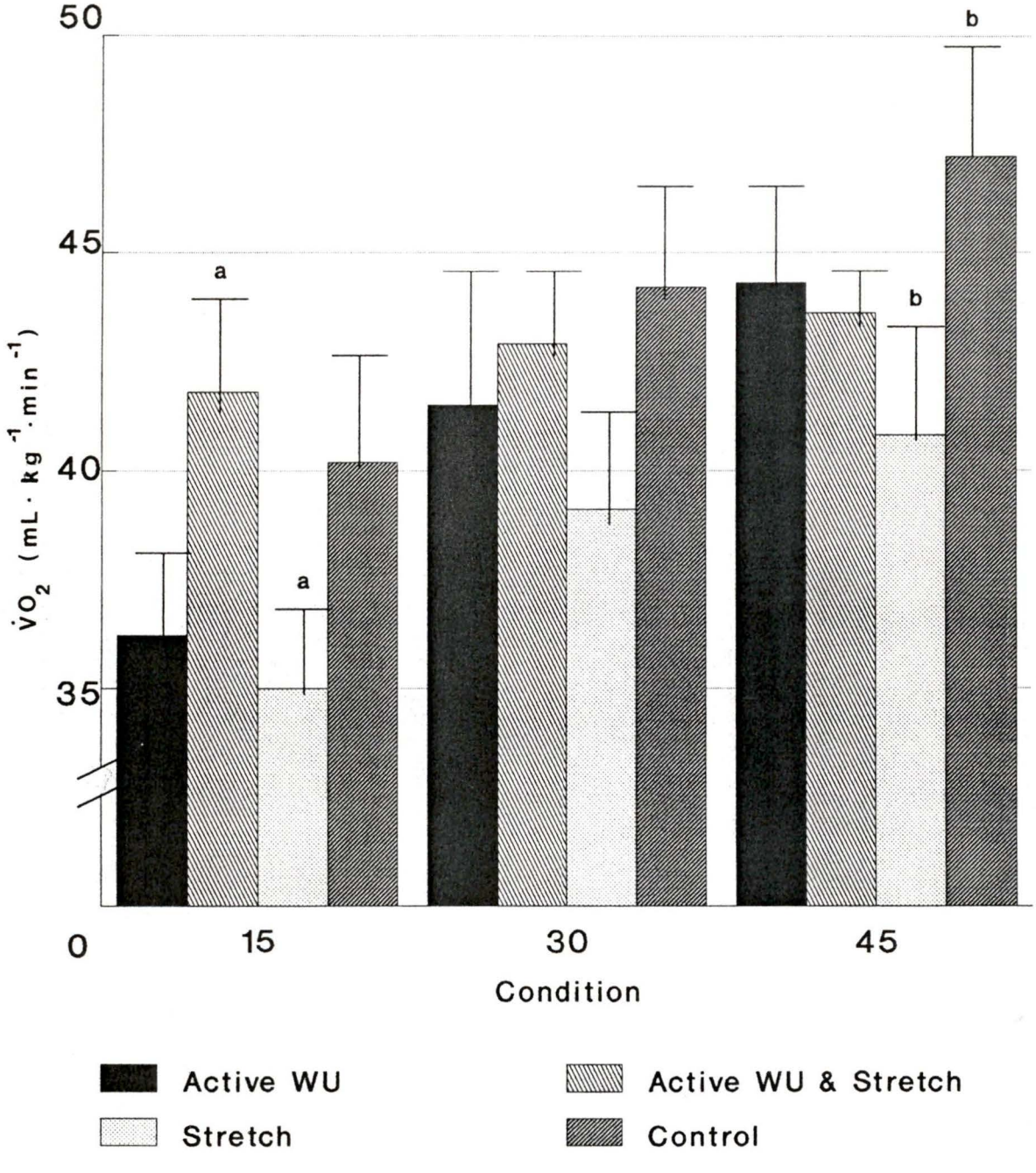
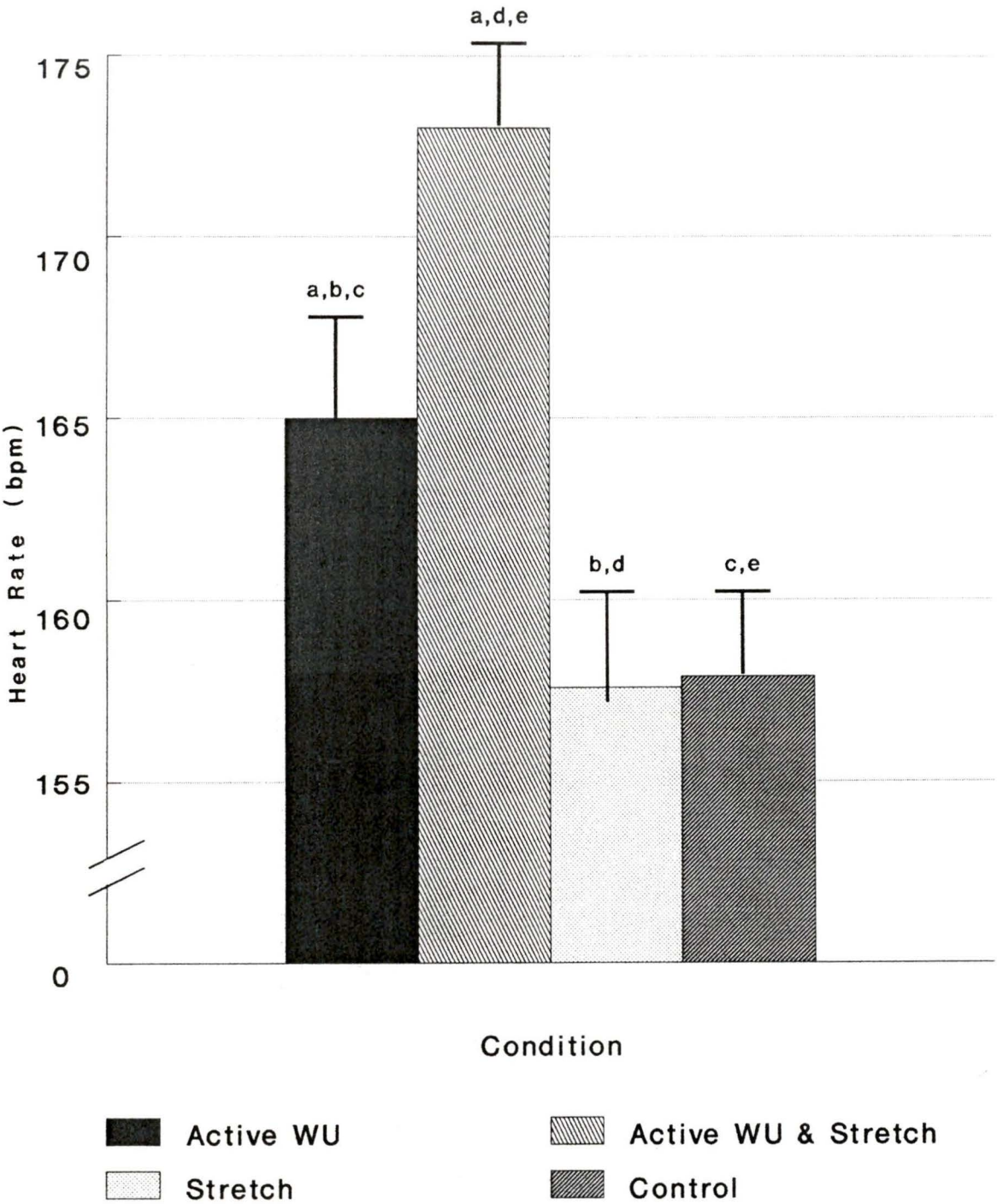

50
45
40
35
0
15
30
45
a
a
b
b
$\dot{V}O_2$ (mL · kg^{-1} · min^{-1})
Condition
Active WU
Active WU & Stretch
Stretch
Control

Figure 5. Heart rates post-Anaerobic Criterion Test (ACT) for the different conditions.

Paired letters show groups which are significantly different (a,c $p < 0.05$; b $p < 0.01$; d,e $p < 0.001$).


a,d,e
175
170
a,b,c
165
b,d
c,e
160
155
Heart Rate (bpm)
0
Condition
Active WU
Active WU & Stretch
Stretch
Control

DISCUSSION

The purpose of this study was to examine the effect of active warm-up and/or stretching on selected physiological responses to, and performance of, a 45-second anaerobic criterion cycle ergometer test. The present study has shown that active warm-up, stretching, and a combination of the two had no significant effect on performance of this test, even though some of the physiological indices measured were affected by the warm-up procedure.

Active Warm-up

With AWU, core temperature increased 1 °C in 34.6 minutes. Asmussen and Boje (1945) found similar results, with Tc increasing 0.8 °C in 30 minutes following preliminary work at a rate of 110 watts. Despite the elevation of Tc by 1°C and Tm by 3.2 °C during the AWU condition, no significant differences were found in mean oxygen consumption, blood lactate, peak power or total work between AWU and control conditions.

At the onset of strenuous exercise, the metabolic demand for 0_2 is greater than the cardiovascular system can provide. This 0_2 deficit results in the utilization of anaerobic energy reserves to meet the energy demands of the task, and if the task requires total utilization of these reserves, ie. event greater than 60 seconds (Astrand & Rodahl, 1970), the amount of reserves depleted to meet the initial 0_2 deficit could reduce the amount of work available for the rest of the task. If an individual could elevate cardiac output and

muscle blood flow immediately before the task, this deficit could be reduced, and subsequent performance in tasks requiring anaerobic capacity improved (Di Prampero et al., 1970).

A difference in oxygen consumption values may have been expected in this study, as several reasons have been suggested as to explain why a higher percentage of total energy expenditure might come for aerobic processes when the criterion task is preceded by active warm-up. Increased blood flow might have been expected due to the vasodilatory effect of warm-up on precapillary resistance vessels and the local effect of increased metabolism of the working muscles, resulting in greater delivery of oxygen to the working muscle (Martin et al., 1975; Ingjer & Stromme, 1979). Also, activity in the sympathetic adrenergic vasoconstrictor fibers would cause a redistribution of blood volume, reducing blood flow to the splanchnic regions, kidneys, and skin, resulting in an estimated 2.2 litres per minute of blood redistributed to working muscles during maximum vasoconstriction of these regions (Rowell, 1974). Increased mobilization of hormones such as epinephrine probably also contributes to increased blood flow (Ingjer & Stromme, 1979), and increased intramusuclar temperature has been shown to enhance enzyme activity, shifting the oxygen dissociation curve to the right (Barcroft & King, 1909). All of these factors would contribute to a greater a-$\bar{v}$ 0_2 difference, leading to an increase in $\dot{V}_{02}$.

The $\dot{V}0_2$ during the 15-second segments of the ACT were not different from each other in any one condition or from the comparable time frame between the AWU and C condition. Also, the $\dot{V}0_2$ over 15 seconds, 30 seconds, and 45 seconds were not different

between the AWU and C conditions. This finding is in agreement with De Bruyn-Prevost and Lefebvre (1980), who found $\dot{V}0_2$ values increased very little during the criterion task following a continuous warm-up at 75% $\dot{V}0_{2max}$. It was suggested that despite an increase in heart rate from the warm-up to the criterion task, $\dot{V}0_2$ was not able to increase further above warm-up levels because of a decrease in stroke volume at high heart rate or a lower 0_2 extraction in the muscle due to the high speed of movement. As the oxygen consumption increased very little during the criterion task itself, the 0_2 debt was not reduced, and therefore the anaerobic energy contribution to the work was not decreased by the AWU.

The intensity of the 75% $\dot{V}0_{2max}$ warm-up used by De Bruyn-Prevost and Lefebvre (1980) was detrimental to performance, producing the shortest performance time of all the groups studied. The 5 minute warm-up at 30% or 75% of $\dot{V}0_{2max}$ probably would not have elicited a significant increase in either core or muscle temperature, while the present study elicited a 1°C increase in Tc. The mean performance time of their criterion test ranged from 35 to 47 seconds, similar to the 45-second duration of the ACT in the present study, but the nature of the tasks was different. In the present study, the time was set at 45 seconds and an all-out effort was required for the duration. In the study of De Bruyn-Prevost and Lefebvre (1980), the maximal task was to volitional fatigue at a predetermined pedalling rate. In both cases, warm-up did not influence any measured performance variables.

That $\dot{V}0_2$ was not significantly different between AWU and control conditions is in

agreement with Busuttil and Ruhling (1977), Knowlton et al. (1978), and Chwalbinska-Moneta and Hanninen (1989). Discrepancies concerning cardiorespiratory and metabolic responses to physical work may be related to the methodological differences among the studies in the warm-up and criterion task used, ranging from the type of task, intensity and duration of effort, and the length of the rest periods used. The majority of studies in which $\dot{V}0_2$ was significantly different between experimental and control groups involved criterion tasks of greater than 60 seconds duration (Inbar & Bar-Or, 1975; Martin et al., 1975; Watt & Hodgson, 1975; Andzel, 1978; Ingjer & Stromme, 1979; Gutin et al., 1976; Gutin et al., 1981). Warm-up duration in these studies ranged from three (Andzel, 1978) to twenty minutes (Inbar & Bar-Or, 1975), with 10 to 15 minutes duration most common (Inbar & Bar-Or, 1975; Martin et al., 1975; Watt & Hodgson, 1975; Gutin et al., 1976). Intensity of criterion task was defined by various methods including a percentage of maximum heart rate (Andzel, 1978), all-out effort (Inbar & Bar-Or, 1975), or the effort predicted to produce exhaustion within a certain period of time (Martin et al., 1975; Watt & Hodgson, 1975). Length of rest intervals ranged from no rest (Martin et al., 1975) up to five minutes (Inbar et al., 1975; Ingjer & Stromme, 1979). Although these studies found significantly higher oxygen consumption during the criterion tasks following warm-up, no two studies were similar enough in protocols for comparisons to be made.

Faster cardiorespiratory adaptation to exercise subsequent to warm-up might be expected to be accompanied by a decrease in exercise-induced blood lactate accumulation. The 2-3 $mM \cdot L^{-1}$ increase in blood lactate concentration resulting from the AWU at the

beginning of the ACT protocol was significantly greater than any of the other conditions, suggesting that some anaerobic energy sources were being utilized during the warm-up itself. However, these values are well below the arbitrarily defined level of the anaerobic threshold, where blood lactate values greater than 4 mM·L are taken as significantly different from resting levels (MacDougall et al., 1982). Lactate values found post-ACT ranged between 10.3 and 10.8 mM·L, slightly higher than those found by Ingjer and Stromme (1979) for cross-country skiers or long-distance runners after the criterion task, and slightly lower than on-ice training values for Canadian National speed skaters (Smith & Roberts, 1990).

Martin et al. (1975) and Ingjer and Stromme (1979) found blood lactate lower after criterion tasks following warm-up while Asmussen and Boje (1945), Knowlton et al. (1978), and De Bruyn-Prevost and Lefebvre (1980) found no significant differences in lactate between warm-up and control conditions which supports the lack of differences in lactate accumulation between AWU and C conditions in the present study. This suggests that metabolic and circulatory changes as a result of warm-up were either not sufficient to enhance this type of performance or would not affect this type of performance regardless of their magnitude.

In the present study, blood lactate was taken at three minutes post-ACT after all the experimental conditions. It is possible that blood lactate may have peaked at different times following the different warm-up conditions and may be one reason why no significant differences were found between conditions in the present study, and in other studies (Knowlton et al., 1978; De Bruyn-Prevost & Lefebvre, 1979).

Post-ACT heart rates were significantly higher following the AWU condition than the

C condition. This is in agreement with almost all other studies except Howard et al. (1966) who found no differences in maximum heart rate between exercises performed with and without warm-up, probably because of the loosely-defined warm-up protocol. There is considerable debate as to what an elevated heart rate means in terms of performance. Inbar and Bar-Or (1975) have suggested that with a higher heart rate, a higher maximal cardiac output is at least partly responsible for higher $\dot{V}0_{2max}$ values found. Ingjer and Stromme (1979) stated that increased body temperature following AWU may in itself be responsible for most of the higher heart rates observed and suggested that increased heart rate should not be regarded as indicative of better preparedness of the circulatory system. Craig and Froehlich (1968) suggested that elevation of heart rate in response to body temperature narrows the margin by which the heart can accelerate in response to the demands of exercise. This margin was shown to be highly correlated with walking time.

In the present study, a significantly higher heart rate after the ACT following AWU was not indicative of greater preparedness of cardiorespiratory systems or cellular metabolic adaptations as a result of warm-up, as no changes in oxygen consumption, blood lactate accumulation, or performance were evident. This is in agreement with Howard et al. (1966), Grodjinousky et al. (1970), and Stewart et al. (1973). However, due to the length of the AWU, thermoregulatory demands may have interfered with the metabolic and circulatory needs of the body, resulting in a competition between exercise and thermoregulatory processes for the blood (Rowell, 1977). Exercise potential can be reduced due to the tendency of heat stress to lower cardiac filling pressure and stroke volume (Ludbrook, 1987). Stroke volume may have decreased due to vasodilation of

subcutaneous blood vessels, impairing the ability of the cardiovascular system to transport oxygen and produce energy (Ludbrook, 1987). Thus, increase in heart rate may have been necessary to maintain cardiac output during the ACT.

The effect of warm-up on performance would depend on a number of variables associated with the warm-up itself, including the type of warm-up used, the intensity and duration of the warm-up, the fitness level of subjects, and the rest interval between warm-up and the criterion task. The results of the majority of studies examining the effect of warm-up on anaerobic performance are not comparable, as the methodology has varied considerably between studies. No studies are directly comparable to the present study, as the protocols followed were not identical to any of the studies researched. However, a discussion of the general trends in the research can be related to the results of the present study.

Contrary to the results of Michael et al. (1957), Pacheco (1957), de Vries (1958), and De Bruyn-Prevost and Lefebvre (1980), no significant improvements or changes in performance were found in the ACT following AWU. Except for the study of De Bruyn-Prevost and Lefebvre (1980), the other studies used performance or criterion tasks which were skill-oriented, such as softball throw, or swimming different strokes. Perhaps AWU would have a beneficial effect on tasks requiring more of a skill component than a more physiologically based task.

Peak power values were similar (La Voie et al., 1984) or higher than other values reported (Smith, 1987; Hill & Smith, 1989) due to higher resistances used in the present study. Total work values were higher than those found for the 30-second Wingate Anaerobic Test because of the extra 15 seconds of the ACT (Hill & Smith, 1989). Only a

brief active rest interval at a low resistance was given between the AWU and the ACT in the present study. If any portion of anaerobic energy stores had been employed during the warm-up, there was little time to replenish them before the ACT began, a possible detriment to the development of peak power and a definite influence on total work.

Stretching

The present study showed that a 16-minute stretching program did not change the physiological response to, or the performance of a 45-second anaerobic criterion test. This finding is in agreement with de Vries (1963) who showed that when the effects of temperature were separated from increased flexibility to eliminate temperature and circulatory factors, no improvement in efficiency in the 100-yard dash could be demonstrated, suggesting that improvement in efficiency was temperature-related. Contrary to this, Pacheco (1957) found that stretching prior to the task, 15 seconds per stretch, improved performance in a vertical jump test.

Mean $\dot{V}0_2$ over the 45-second ACT was significantly higher for the C condition than the S condition. No other studies were found in which $\dot{V}0_2$ was measured in a criterion task following a stretching program. The fact that heart rates and lactate values were not significantly different between the two groups does not explain the difference in $\dot{V}0_2$. A possible explanation is that stretching increased the efficiency of the stretched muscles, resulting in the recruitment of fewer muscle fibers to perform the same task, and lowering the oxygen demand.

Active Warm-up and Stretching

Although temperature differences existed in the muscle, no significant differences were found in oxygen consumption, blood lactate values, peak power, and work performed between AWUS and C. Heart rate following the ACT after AWUS was higher than following the C condition. Several explanations may exist for this. If AWU can elevate cardiac output and increase muscle blood flow, it is possible that the effects were not lost over the 16 minute stretching program. The stretching program and accompanying movements may have helped to keep the heart rate elevated during this portion of the protcol. By narrowing the margin by which heart rate could increase, as suggested by Craig and Froehlich (1968), heart rates may have had to reach higher values to maintain the same $\dot{V}0_2$. Also, as mentioned for the AWU, thermoregulatory processes may have been competing with circulatory and metabolic demands for blood. If so, in order to maintain cardiac output with a decreased stroke volume, heart rate would have to be elevated.

The findings of this present study are in agreement with Howard et al. (1966) who found no significant differences in performance in a running test between those who warmed up with a jogging, calisthenics, and stretching program for 10-15 minutes, and those who did not. deVries (1958) found that a calisthenics and stretching program did improve performance times in some 100-yard swimming events, but in both these studies, the exact role of stretching in the warm-up protocol is impossible to assess. No other studies were found in which a standardized warm-up protocol and standardized stretching program were combined.

In summary, the present study showed that active warm-up and/or stretching prior to performance had no affect on performance of a 45-second all-out cycle ergometer task, even though some physiological changes were elicited by the warm-up protocols.

CONCLUSIONS

1. Although some physiological changes were observed, there was no difference in performance of an anaerobic criterion task following an active warm-up which elicited a 1.0 °C increase in core temperature.

2. There were no differences in selected physiological variables or performance of an anaerobic criterion task following a stretching program.

3. There were no differences in selected physiological variables or performance of an anaerobic criterion task following an active warm-up, to a 1°C increase in core temperature, and stretching program.

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APPENDIX A

Informed Consent

RESEARCH OUTLINE

Purpose:

To investigate the effect of active warm up, passive warm up and stretching on anaerobic performance and selected physiological variables.

Procedures:

Subjects will be asked to come to the Fitness Centre on seven different occasions. During the first and last session, maximal oxygen consumption will be assessed on a bicycle ergometer. Anthropometric measures of height, weight, and skinfolds will also be taken at this time.

For each subsequent session, subjects will be asked to perform a 45-second all-out sprint on a bicycle ergometer following one of five warm up conditions. All-out effort will be required for the 45-second duration. Subjects will be asked to report to the Fitness Centre at approximately the same time each testing day. Subjects will be asked to maintain their training schedule, but will be asked to refrain from vigorous physical activity for 24 hours prior, and from eating or smoking for three hours prior, to arriving for testing.

Warm up conditions include active warm up (AWU) to a 1 °C increase in core temperature (Tc) to a maximum of 40 °C, passive warm up (PWU) to a similar increase in Tc, very light AWU and stretching, very light AWU only, and AWU to 1 °C increase in Tc and stretching. All AWU will be performed on a bicycle ergometer at approximately 60% of pre-determined $\dot{V}O_2max$.

Subjects will be asked to insert a sterile rectal temperature thermocouple prior to each of these five testing sessions, and a sterile muscle temperature thermocouple will be placed in the lateral portion of the right thigh for the duration of the testing session.

Finger prick blood samples will be taken at four times during the testing session: immediately following warm up, immediately before and after the criterion, 45-second all-out test, and three minutes post-test.

RISKS

Implantation of muscle temperature thermocouples and the drawing of finger prick blood samples will be performed by a skilled technician. These procedures are considered safe with little chance of complication, although there is a small chance of infection with any laceration of the skin. Slight bruising may also occur at the implantation and finger prick sites.

INFORMED CONSENT

I confirm that I have been given a satisfactory and complete explanation of the procedures in which I am to participate. I further confirm that I have been advised that I may ask for further explanation and/or demonstration of such procedures at any time. I also acknowledge that I have been advised that I may terminate participation in any or all of the procedures at any time as a matter of my own pesonal discretion or volition. I hereby waive and disclaim any entitlement against the University of Victoria, the personnel involved in this research study, the research investigators, or any other person in repsect of liability that my arise from my participation as a research subject in this study.

Name (please print): _____

Signature: _____

Date: _____

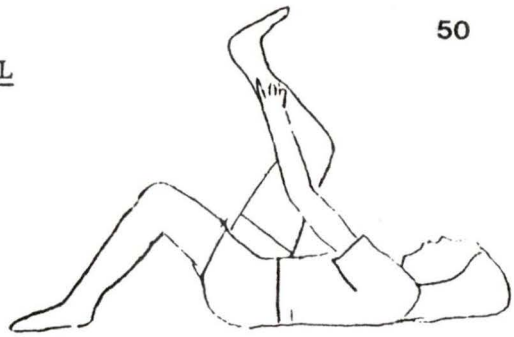
APPENDIX B

Stretching Program

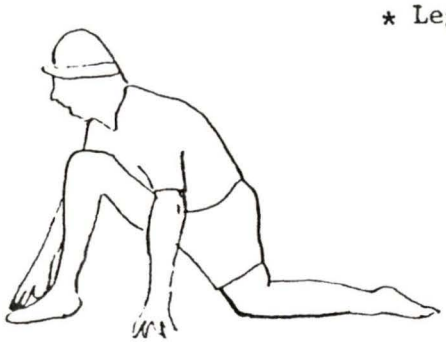
STRETCHING PROTOCOL

* Andersen, 1980

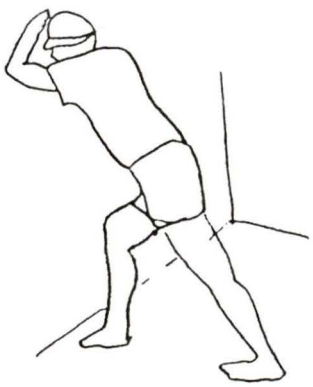
Ankle plantor flexor



* Leg flexor



* Hip flexor



*Ankle dorsiflexor

* Leg extensor

* Hip extensor
Ankle dorsiflexor

Hip extensor

APPENDIX C

REVIEW OF LITERATURE

DETERMINANTS OF SKELETAL MUSCLE & CORE TEMPERATURE

The temperature of any tissue is determined by its rate of energy metabolism and its blood flow (Werner & Buse, 1988). Resting skeletal muscle has a metabolic rate of about 2 millilitres of oxygen per minute per kilogram of body weight, and a blood flow rate of approximately 25 millilitres per minute per kilogram of body weight (Nadel, 1983). Skeletal muscle temperature is the net result of a regional balance of heat production and heat loss (Mitchell, 1977). Temperature in resting muscle shows considerable variation, and in the muscles of the extremities, resting muscle temperature generally ranges from 34 to 35.5 °C (Buchtal et al., 1944).

With the onset of dynamic exercise, there is an upset in the thermal energy balance of resting muscle. Heat is produced as a result of exercise, the amount produced being proportional to the intensity of exercise (Nadel, 1983). As muscle temperature increases, the temperature gradient between working muscle and surrounding tissue changes, enhancing heat exchange by conduction to surrounding tissues (Werner & Buse, 1988). The temperature gradient between arterial blood (at body core temperature) and working muscle is also reversed, resulting in a transfer of heat from muscle to blood (Mitchell, 1977).

Convective heat transfer to blood vessels is determined by the geometry and the hydromechanical properties of the vessels (Werner & Buse, 1988). In larger vessels, there is little heat transfer with muscle tissue while in muscle capillaries, there is an almost immediate thermal balance achieved with the environment (Werner & Buse, 1988). Heat transfer relevant to thermoregulatory processes therefore occurs in terminal arteries,

arterioles, and venules, and heat is transferred from the muscle to the blood during the period it resides in muscle capillaries (Fortney & Vromam, 1985).

During exercise, heat in working muscles is produced at a rate 15-20 times that of the entire resting metabolic rate (Nadel, 1977), depending on the metabolic rate of these muscles (Werner & Buse, 1988). The rate of muscle blood flow can increase to approximately 30 times that of resting conditions and is the primary factor for dissipating the heat produced (Nadel, 1983). The rate of heat production and heat loss eventually meet a balance in steady-state exercise, and a new elevated Tm is achieved for the remainder of the exercise (Mitchell, 1977). In summary, blood enters the exercising muscle at core temperature, reaches thermal equilibrium with the muscle in its capillaries, and leaves the muscle at muscle temperature. Through convection, the heat produced by the exercising muscle is carried away by the blood through venous return to the heart and body core, elevating body core temperature (Fortney & Vroman, 1985).

The thermal receptors in the hypothamlamus receive a representative sample of this warm blood, and together with other information received by other sources, ie. thermal receptors in the skin, an integrated heat loss/dissipation response is triggered (Nadel, 1977; Fortney & Vroman, 1985). Heat is transferred to the skin via the blood (Rowell, 1977) and is lost to the environment via radiation and convection, at a rate proportional to the difference between skin temperature and ambient temperatures (Fortney & Vroman, 1985; Werner & Buse, 1988), and evaporation through sweating (Stolwijk et al., 1977).

The temperature of exercising muscle rapidly achieves and exceeds temperature maintained in the body core (Aikas et al., 1962). Of the three experimentally used body core temperature measurement sites, tympanic and esophageal temperatures have been

shown to respond more quickly than rectal temperature measurement, exhibiting stable, elevated levels within 15 minutes of the onset of steady state exercise (Asmussen & Boje, 1945, Brengelmann, 1977;). Body core temperatures recorded through measurement of rectal temperature respond more slowly, plateauing after approximately 30 minutes of steady state exercise (Asmussen & Boje, 1945). Core temperature has been shown to be set according to the relative workload of the individual (relative to maximal oxygen consumption) and not to the absolute workload performed (Saltin & Hermansen, 1966). Of the two, muscle temperature appears to be of greater benefit to subsequent performance than does core temperature (Asmussen & Boje, 1945; Martin et al., 1975; Dolan & Sargeant, 1983).

PHYSIOLOGICAL BENEFITS OF WARM-UP

There are many physiologically beneficial reasons proposed in the literature for warming up the body before vigorous exercise - they are summarized by Franks (1983) as follows: 1.) decreased intramuscular resistance resulting in more energy for external work (Astrand & Rodahl, 1977); 2.) increase in blood temperature causing increased dissociation of oxygen from hemoglobin and myoglobin, resulting in greater aerobic metabolism (Barcroft & King, 1909); 3.) decreased time for initial metabolic adjustment to heavy work, resulting in a decreased oxygen deficit (Gutin et al., 1976); 4.) increased nerve conduction velocity (Ruch et al., 1966); 5.) greater number of open capillaries in muscle allowing greater oxygen exchange (Cureton, 1947). Muscle viscosity is also

affected by temperature, with below normal body temperatures producing an increase in viscosity and a decrease in physical performance (Zuntz et al., 1906).

Another important consideration for warming up is its effect on the heart and its function. Barnard et al. (1973) found that warm-up eliminated or reduced abornormal ECG responses to sudden exercise. Adaptation to coronary blood flow to rapid increases in cardiac workload is apparently not instantaneous. Warm-up in this case would prepare the heart for an increase in workload and help decrease or prevent the periods of ischemia in the heart which may occur under sudden physical stress. Research has shown that warm-up may also reduce the chance of an asthma episode in subjects susceptible to exercise-induced asthma performing maximally for six minutes (Reiff et al., 1989).

Therefore, changes in metabolic processes occur which may be of benefit to anaerobic performance, in which the initial work loads are maximal or supramaximal with respect to $V0_2$ (Gutin et al., 1976; Ingjer & Stromme, 1979). Research investigating the effects of subnormal temperature on performance supports the premise that warming up the body may be of benefit, with below normal muscle temperatures impairing or reducing anaerobic performance.

Bergh & Ekblom (1979) found that performance in short-term exercises such as jumping and sprinting is reduced at low muscle temperatures and enhanced when muscle temperature is above normal. It was also discovered that during rest and submaximal exercise, lowered esophageal (Te) and skin temperatures (Tsk) produced oxygen uptake above normal levels (attributed to shivering) and lower heart rates for a given oxygen uptake (Bergh, 1980). Peak aerobic power and peak heart rate during exhaustive exercise of 3-8 minutes duration was positively related to Te and Tsk, and maximal anaerobic

power was positively related to muscle temperature (Tm) - the effect of temperature was 4-6% increase in power per °C. Bergh (1980) also found that at exercise durations less than two minutes, lower Tm produced a faster rate of lactate accumulation in working muscle, probably attributable to a slower rate of lactate removal; with exercise durations of 3-8 minutes, this was combined with reduced aerobic power. Maximum muscle strength was positively related to Tm, showing an effect of 2-6% per °C.

It has been proposed that cooling of the muscle prior to exercise is likely to affect chemical reactions which occur in the muscle during exercise (Asmussen et al., 1976). Gilmour et al. (1986) found decreases in peak power and mean power after performing a Wingate test following cold immersion of the legs. Cooling the muscle may have slowed the chemical reactions occurring in the muscle, delaying muscle fibril cross-bridge formation and slowing the rate of tension development in the muscle (Bergh & Ekblom, 1979). Blomstrand et al. (1986) found a greater increase in glycolysis in muscle at subnormal muscle temperatures, possibly as a result of decreased work efficiency and or a reduced flow of blood in the cold muscle. These results show that subnormal temperatures can have a negative effect on performance.

PHYSIOLOGY OF ANAEROBIC PERFORMANCE

In any maximal or supramaximal task, energy required to do the task is provided through a combination of aerobic and anaerobic metabolic processes with the contribution of anaerobic processes increasing as the intensity of the activity increases (Katch &

Weltman, 1979). At the onset of such exercise, the metabolic demand for oxygen can be greater than the cardiovascular system can provide and anaerobic energy reserves are utilized to meet the demands of the task (Gutin et al., 1976). For short-term, high-intensity exercises, oxygen deficit for the duration of the exercise is common, with the anaerobic sources providing the majority of the ATP (Gollnick et al., 1986). If the task requires total utilization of these reserves ie. any event greater than 60 seconds duration (Astrand & Rodahl, 1970), the amount of reserves depleted could reduce the amount available for the rest of the task (Gutin et al., 1976).

With a rapid acceleration of anaerobic glycolysis, there is an equally rapid accumulation of lactic acid (Hultman & Sjoholm, 1983). Muscle glycogen stores provide fuel for this process and as these stores are used up, lactic acid accumulates in the muscle and the blood (Gollnick et al., 1986). As lactic acid levels in muscle increase, muscular contraction is hindered by a downward shift in muscle pH (Donaldson & Hermansen,1978).

The formation and turnover of actin-myosin cross-bridges is mediated by the release of calcium ions from the sarcoplasmic reticulum and their coupling to the troponin molecule; both of these biochemical processes are liable to interference by a higher hydrogen ion concentration (Hultman & Sjoholm, 1986). The activity of the enzyme responsible for breaking actin-myosin cross-bridges, myofibrillar ATPase, is also decreased with low pH conditions (Gollnick et al., 1986) and as its muscle glycogen stores are depleted, the muscle exhausts its fuel supply, with muscular activity being terminated or its intensity greatly reduced (Gollnick, 1987).

It has been suggested that the level of lactate (the dissociating salt of lactic acid) in the blood could be an indicator of the predominant energy processes involved in exercise. The concentration of lactate in the blood is dependent on the release of lactate from the contracting muscle and on the rate of lactate removal from the blood (Katz & Sahlin, 1988). Data supports the hypothesis that lactate production during submaximal and maximal exercise is to a large extent dependent on the availability of oxygen in the contracting muscle (Katz & Sahlin, 1988). As the intensity of exercise increases, an increase in muscle and blood lactate concentration is evident, becoming exponential at a certain intensity (Gollnick et al., 1986). An increase in the contribution of energy from aerobic processes during maximal tasks at the same power output would theoretically benefit performance by helping to reserve some of the anaerobic sources of ATP until needed later in the task (Andzel & Gutin, 1976). However, Dill et al. (1932) suggest that lactic acid accumulation alone is inconclusive evidence of oxygen deficiency.

Most of the literature has suggested that warming up before vigorous activity has an activating effect on aerobic metabolism. A number of these studies have examined the metabolic and circulatory effects of warm-up on the body and on performance (Asmussen & Boje, 1945; Inbar & Bar-Or, 1975; Martin et al., 1975; Gutin et al., 1976; Andzel, 1978; Ingjer & Stromme, 1979; Gutin et al., 1981; Blomstrand et al., 1986; Chwalbinska & Hanninen, 1989). Three physiological indices of the effect of warm-up on metabolic processes and circulation and their subsequent effect on anaerobic performance prevail in these studies: changes in oxygen consumption, heart rate, and blood lactate values during the criterion task.

Warm-up and Oxygen Consumption

In 1909, Barcroft and King discovered that higher temperatures affected the dissociation curve of hemoglobin. Hemoglobin at an oyxgen tension of 30 mm Hg gave up almost two times as much oxygen at 41 °C as at 36 °C, with oxygen dissociating from hemoglobin about two times faster. In a series of experiments on body temperature and the capacity for work, Asmussen and Boje (1945) showed that oxygen uptake was greater in the 'warm' versus 'cold' condition, but the oxygen necessary for actually performing the work was actually less in the warm condition, suggesting an acceleration of aerobic processes in the warm condition and a reduction in the internal resistances. Only two studies were found in which warm-up did not affect metabolic or cirulatory responses to the criterion task.

In a study of the metabolic responses of untrained individuals to warm-up, Knowlton et al. (1978) concluded that untrained individuals lack the cardiovascular and cellular adaptations necessary to show metabolic benefits from warm-up. However, preliminary activity of two minutes at 20% and at 40% of a subject's physical work capacity hardly appears adequate to raise muscle or body core temperature to a significant degree and the intensity of the criterion task was below $\dot{V}0_{2max}$. Chwalbinska and Hanninen (1989) found that a 10-minute warm-up at 40% $\dot{V}0_{2max}$ did not affect the circulatory and ventilatory responses to an incremental exercise to exhaustion and oxygen uptake at submaximal or maximal loads. It is likely that the lower levels of the incremental exercise probably served as a warm-up in themselves, thus negating the influence of lower

intensity preliminary activity. A significant increase was seen however in the absolute work load corresponding to the anaerobic threshold (at blood lactate level four mmol per litre), as well as in the absolute and relative loads, $\dot{V}0_2$ and heart rates corresponding to individual anaerobic thresholds.

Several reasons have been suggested to explain why a higher percentage of total energy expenditure might come from aerobic processes when the criterion task is preceded by active warm-up. Increasing blood flow could result in a greater delivery of oxygen to the working muscle - this may occur as a result of the vasodilatory effect of the warm-up on the precapillary resistance vessels, and the local effect of increased metabolism on the capillaries of the working muscles (Martin et al., 1975; Ingjer & Stromme, 1979). Activity of the sympathetic adrenergic vasoconstrictor fibers during warm-up can also cause a redistribution of blood, with flow being reduced to splanchnic regions, kidneys, and skin, and shifted to the working muscles (Fortney & Vroman, 1985). The mobilization of hormones such as epinephrine, which dilates the resistance vessels in skeletal muscle, probably also contributes to an increased blood flow (Fortney & Vroman, 1985).

Increased intramuscular temperatures as a result of warm-up would theoretically increase the enzymatic activity and increase the rate at which chemical reactions proceeded in the muscle (Bennett, 1984). A rightward shift in the oxygen dissociation curve and/or a greater a-$\bar{v}$ 0_2 difference under these conditions have also been proposed to explain the increases in the rate of oxygen consumption in certain criterion tasks after warm-up (Inbar & Bar-Or, 1975; Martin et al., 1975; Ingjer & Stromme, 1979). Increases in heart rate

usually accompany increases in the rate of oxygen consumption (Inbar & Bar-Or, 1975; Ingjer & Stromme, 1979). Gutin et al. (1976) suggested that higher $\dot{V}0_2$ seen following prior exercise was a function of higher heart rate, not higher stroke volume or $a\text{-}\bar{v}\ 0_2$ difference. In a study on the effect of altering heart rate on oxygen uptake at exercise onset, Casaburi et al. (1989) demonstrated that ventilation-independent changes in oxygen uptake could be induced at the onset and cessation of exercise in patients with programmable pacemakers under conditions of constrained and accelerated heart rates. Alterations in oxygen uptake found were predictable from differences in blood flow resulting from differences in the time course of the heart rate.

Warm-up and Heart Rate

Heart rate has a potential to effect performance because of its role in the delivery of blood and oxygen to the working muscles of the body. During exercise, cardiac output increases, brought about by increases in stroke volume and heart rate. Stroke volume increases as exercise intensity progresses from rest to moderate work, but does not necessarily increase beyond a moderate workload. Martin et al. (1975) found stroke volume unaffected or impaired as heart rate approached and exceeded 165 beats per minute while other authors suggest stroke volume does not increase beyond a heart rate of 150-180 beats per minute (De Bruyn-Prevost & Lefebvre, 1980). Once stroke volume reaches its limit, further increases in cardiac output are achieved only through increases in heart rate (Fox & Mathews, 1981).

All but one of the studies examining the effect of warming up on heart rate have

found that heart rate during the criterion task is elevated above the level of the controls after warm-up, regardless of the type of warm-up (Inbar & Bar-Or, 1975; Martin et al., 1975; Watt & Hodgson, 1975; Gutin et al., 1976; De Bruyn-Prevost, 1980). Only Howard et al. (1966) found no effect of warm-up on heart rate during the performance of fifteen, repetitive 100-yard sprints. However, the warm-up activities prior to the task were loosely defined, non- structured, and left up to the discretion of the subjects. Thus, the warm-up may not have been intense enough or the rest interval could have been too long.

In summary, warm-up which increases core temperature elevates heart rate during the performance of a given task. Ingjer and Stromme (1979) suggested that the increased body temperature after warm-up may itself be responsible for the majority of higher heart rates and expressed caution in regarding this phenomenon as an indication of a greater preparedness of the circulatory system.

Warm-up and Blood Lactate Concentration

Muscular activity in man is associated with lactate production in active muscle when metabolism is accelerated, and exercise of duration less than three minutes at near maximal intensities causes an abundant production of lactate (Rontoyannis, 1988). High concentrations are found initially at the level of the active muscle and are subsequently found in blood and other tissues. The concentration of lactate in the blood is dependent on the release of lactate from the contracting muscle and the rate of lactate removal from the blood, and is therefore influenced by many factors including diet and exercise, epinephrine and glycogen levels (Brooks, 1986; Gollnick et al., 1986), temperature

(Ingjer & Stromme, 1979), and the enzymatic profile of the muscle ie. fiber type, pattern of fiber recruitment, showing the diversity of the different modulators of lactate metabolism (Katz & Sahlin, 1988; Rontoyannis, 1988).

Increases in blood lactate is correlated with lactate in contracting muscles in many situations and could provide a rough but adequate index of lactate production during short-term exercise (Katz & Sahlin, 1988). Karlsson (1971) showed that the concentration of lactate in muscle and blood are remarkably similar when the intensity of exercise leads to exhaustion in 8 minutes or less. A large part of lactate metabolism is suggested to occur by lactate shuttle, where the lactate produced in fast-twitch glycolytic fibers is shuttled for oxidation to fibers with a high oxidative capacity (Brooks, 1986). Measurements of release and accumulation of lactate would not include the oxidized lactate, resulting in an underestimation of the total lactate production (Katz & Sahlin, 1988).

In studies examining the physiological responses of the body to warm-up, blood lactate values were taken to evaluate the contributions of anaerobic metabolism, specifically anaerobic glycolysis, to warm-up and the subsequent criterion task. Asmussen and Boje (1945) hypothesized that oxidative and anaerobic processes would be accelerated by increases in body temperature but were unable to demonstrate any changes in the formation of lactic acid between warm-up and no warm-up conditions. Stamford et al. (1978) found no changes in $\dot{V}0_2max$ reached i.e. performance, despite elevated pre-test blood lactate levels ranging from 5 to 16 mMol, in an experimental protocol designed to produce varying levels of exhaustion i.e. warm-up. Although performance

times decreased, $\dot{V}0_2max$ was not significantly different from a previously determined standard reference value. Blood lactate concentrations obtained 2.5 minutes post-exercise were not significantly different among the tests. Ingjer and Stromme (1979) found that the highest values for lactate concentration following passive and no warm-up were found 3 minutes after the end of the standard work task, while the highest value following active warm-up was found 30 seconds after the task. As well, blood lactate concentrations following the standard work were lower when the work was preceded by active warm-up as compared with passive or no warm-up. Thus the results of the studies are conflicting.

WARM-UP AND ANAEROBIC PERFORMANCE

The effect of warm-up on performance would depend on a number of variables concerning the warm-up itself. Some researchers have found warm-up to enhance anaerobic performance (de Vries, 1959; Inbar & Bar-Or, 1975; Davies & Young, 1983; Dolan et al., 1985), while others have found it ineffective or detrimental to performance (Thompson, 1958; Margaria et al., 1971; DeBruyn-Prevost, 1980; Genovely & Stamford, 1982). The reason for these discrepancies becomes apparent after the methods employed by the individual studies are examined.

Many factors would affect the influence of warm-up on performance, including the criterion task, type of warm-up used ie. identical to criterion task, specific to task but lower intensity, or general or unrelated to criterion task, the intensity and duration of warm-up, the fitness level of participants, and whether or not the warm-up was active or passive. The results of the majority of studies examing the effect of warm-up on

anaerobic performance are not comparable, as different warm up protocols, criterion tests measuring performance, and types of subjects have been employed. In some cases, information regarding warm-up intensity, type, or duration is not given; therefore, these studies are not reproduceable. In most cases, muscle temperature and/or core temperature have not been measured, leaving the term "warm-up" without strict definition in the study (Howard et al., 1966; Margaria et al., 1971; De Bruyn-Prevost, 1980; Dolan & Sargeant, 1983; Dolan & Sargeant, 1984). Some authors acknowledge this limitation and have used the term 'Prior Exercise', (PE), to label the pre-criterion task activity (Gutin et al., 1976).

The majority of studies examined have studied the effect of active warm-up on anaerobic performance. No studies were found in which the warm-up protocol used was identical to the criterion task. Of the warm-up protocols which were specific to the criterion task but differed in intensity, or duration, the majority resulted in improvement in the criterion task performed. Pacheco (1957) and de Vries (1958) found that speed in swim trials showed a significant improvement following a swim warm-up, Michael et al.(1957) found softball throws were significantly greater following a five minute warm-up of lesser distance, and De Bruyn-Prevost and Lefebvre (1980) found similar results in a criterion cycle ergometer test following a light warm-up on the ergometer.

General warm-up (unrelated to the criterion task) has also been shown to enhance performance. Michael et al. (1957) found that calisthenics and sprint running could significantly improve softball throw distance, and de Vries (1958) found increases in breaststroke and dolphin swim times following a warm-up of calisthenics. A criticism of several of the earlier studies on the effect of warm-up on performance is that warm-up per se is not actually measured, and warm-up intensity is not given or measured - the results

are not reproducible.

Some researchers have manipulated the type, intensity, and duration of warm-up to observe the effects on subsequent performance of a criterion task. Continuous versus discontinuous or intermittent warm-up protocols have been studied. Inbar and Bar-Or (1975) found significantly higher performances on an anaerobic cycle ergometer test following a 15-minute intermittent warm-up protocol on a treadmill. Using children who were ignorant of warm-up habits, a general warm-up was decided upon to reduce the chance of fatigue. De-Bruyn Prevost and Lefebvre (1980) found no improvements in performance with a low intensity, discontinuous warm up protocol, suggesting that such a protocol would be similar to no warm-up at all. Continuous warm-up protocols seem to be the method of choice in most of the studies, but this seems to be the only similarity in these studies.

As mentioned earlier, several of the earlier studies using the continuous method of warm-up failed to report the warm up intensity used (DeVries, 1958; Massey et al., 1960; Howard et al., 1966). The intensities which have been studied range from 30% $\dot{V}0_{2max}$ (De Bruyn-Prevost & Lefebvre, 1980) to 95% $\dot{V}0_{2max}$ (Dolan & Sargeant, 1984) with varying results on performance. De Bruyn-Prevost and Lefebvre (1980) found that a warm-up intensity of 30% $\dot{V}0_{2max}$ improved anaerobic performance on a cycle ergometer task, while 75% $\dot{V}0_{2max}$ resulted in performance decrements. Dolan and Sargeant (1983) found that warm-up intensities less than 60% $\dot{V}0_{2max}$ could elicit increases of 8-15% in power output on an isokinetic cycle ergometer task. At intensities greater than this, an inverse relationship was found between the prior exercise and power output. It has also

been demonstrated that warm-up intensities of 62% $\dot{V}0_{2max}$ and below can substantially increase maximal power output by 12% during a 20 second all-out test on an isokinetic cycle ergometer (Dolan & Sargeant, 1984), and increase total power during a maximal cycle ergometer task (Inbar & Bar-Or, 1975). The majority of evidence seems to indicate that warm-up of an intensity less than 62% $\dot{V}0_{2max}$ can positively influence subsequent anaerobic performance.

Duration of the warm-up activity has ranged from two minutes of jogging in place (Barnard et al., 1973) to 20 minutes on a cycle ergometer (De Bruyn-Prevost & Lefebvre, 1980), to 25 minutes running on a treadmill (Ingjer & Stromme, 1979), and 30 minutes on a cycle ergometer (Asmussen & Boje, 1945). Thus, duration and intensity of warm-up are two key variables in determining the effectiveness of the warm-up protocol.

During high levels of submaximal exercise, there is a depletion of high energy phosphates in the active muscle (Margaria et al., 1971; Dolan & Sargeant, 1983). Since the power developed during the first few seconds of maximal exercise is presumably related to the amount of adenosine triphosphate (ATP) and creatine phosphate (CP) stores in the active muscle, it is possible that prevailing levels of high energy phosphates will differ with varying intensity and duration of warm-up. The results of the criterion task could thus be affected by an improper warm-up protocol, causing fatigue if too intense, and inadequate stimulation for physiological changes and benefits to occur if not intense enough.

Two other areas which could affect anaerobic performance by the warm-up protocol are duration of rest interval, if any, and the fitness level of the subjects. As mentioned

previously, if intensity of warm-up is low, and the rest interval before the criterion task is long, the effect would be as if no warm-up had been given. On the other hand, an intense warm-up with a short or nonexistent rest interval could result in fatigue and impairment of performance. Genovely and Stamford (1982) incorporated a five-minute rest interval into their test protocol and found that warm-up at intensities less than anaerobic threshold (AT) did not contribute to an increase in performance whereas warm-up intensity greater than AT impaired performance. Dolan et al. (1985) found that performance following warm-up and a six-minute rest interval resulted in a significant increase in maximal peak power. De Bruyn-Prevost and Lefebvre (1980) found that a five minute rest period resulted in no modification of performance, whatever the warm-up intensity.

Fitness level of the subjects is another important consideration (Shellock & Prentice, 1985). If the fitness level of the subjects is poor, fatigue may result from the warm-up protocol and result in performance decrements. If the fitness level of the subjects is high, a greater absolute stimulus will be required to elicit the same physiological responses. Most of the studies in which warm-up has benefitted performance have used healthy, relatively young people, generally physical education students and varsity athletes (Michael et al., 1957; DeVries, 1958; Thompson, 1958; Barnard et al., 1973; Bergh & Ekblom, 1979; Davies & Young, 1983).

It appears that the warm-up protocol to be used for varying criterion tasks should also be specific to the task itself (Shellock & Prentice, 1985) while others suggest that the most effective warm-up is an active one that consists of general and specific exercises (Kulund & Tottossy, 1983). A summary of the trends in the types of warm-up that have benefitted performance follows: most of the warm-up protocols which enhance performance are at

least 10 minutes in duration; the intensity of the majority of the warm-ups which improved performance was moderate, between 60 and 80% of $\dot{V}0_{2max}$; low levels of warm-up were not different from rest; beneficial warm-ups are usually specific to the criterion task, although some successful warm-up routines combine specific and general warm-ups; criterion tasks used in studies showing improvement with warm-up have been those of short, explosive, anaerobic nature; specific warm-ups of moderate intensity and duration prior to criterion tasks of an anaerobic nature appear to improve performance in trained individuals.

FLEXIBILITY AND THE BENEFITS OF STRETCHING

Flexibility has been technically defined as the ability to move muscles and joints through their full range of motion (Holland, 1968). Two basic types of flexibility have been described in the literature: static, passive, or extrinsic and dynamic, active, or intrinsic. Static flexibility is the range of motion (ROM) about a joint with no emphasis on speed or movement (Gordon, 1966; Jensen & Fisher, 1979) while dynamic flexibility is defined as the opposition or resistance of a joint to motion - the ability to use a range of joint motion in performance of a physical activity at a normal or rapid speed (Gordon, 1966; Beaulieu, 1980). Flexibility is highly specific and varies from joint to joint within the individual and between individuals (Harris, 1969; Bell & Hoshizaki, 1981). The development of flexibility by stretching will be discussed later.

A number of theoretical benefits of stretching for physical and athletic skills have

been given including a possible decrease in the incidence of musculotendinous injuries (Holland, 1968; Nicholas, 1970; Sapega, 1981; Ekstrand, 1983a), minimization and alleviation of muscle soreness (deVries, 1961a,b), and possible enhancement of athletic performance (Schultz, 1979; Beaulieu, 1981). It is generally felt that muscle tightness may restrict range of motion which can predispose an individual to muscle strain and tendonitis (Nicholas, 1970; Schultz, 1979; Glick, 1980). The effect of stretching on range of motion has been shown to last up to 90 minutes (Moller, 1985). Repetitive stretching with the muscle at a constant length has been shown to produce reduced tension at that length and stretching to constant tension results in lengthening of muscle with subsequent stretches (Taylor, 1985a,b). With stretching, a greater muscle length is necessary to attain the tension for tearing muscle, indicating that stretching may play a large role in muscle injury prevention.

From the point of view of aesthetics, flexibility is a strong requirement in many skilled disciplines, such as dance, gymnastics, and karate, and may be the difference between average and excellence in performance. Flexibility also plays a role in the biomechanics of skilled movement, with increased range of motion allowing the application of force over greater distances and longer periods of time (Alter, 1988). Increased range of motion also permits a greater stretch on the muscle, allowing the production of greater forces as prestretched muscles are more efficient and elastic energy stored in the muscle tissue during stretching is recovered during the subsequent shortening (Asmussen & Bonde-Petersen, 1974).

Shellock and Prentice (1985), Kravitz and Harter (1989), and Safran et al. (1989) have cited the importance of stretching or flexibility training in the reduction and

prevention of injuries in athletics but there seems to be an optimal level of flexibility depending on the sport (Glick, 1980). It has suggested that excessive flexibility may destabilize the joints and increase the likelihood of ligament injury and joint separation or dislocation but Moretz et al. (1982) failed to find any correlation between laxity of a joint and knee injuries. Many teams and individual athletes have noted more musculotendinous injuries with stretching (Benjamin, 1979; Fixx, 1980) and overstretching or improper stretching have been shown to produce tightness or injury (Kulund and Tottossy, 1983). Ireland and Micheli (1987) found no significant relationship between stretcing and the number of injuires or injury patterns in triathletes. Though clinical evidence appears to support the premise that the inclusion of stretching into an exercise or athletic program may reduce the chances of injury (Glick, 1980), conclusive scientific documention is lacking.

The human body contains a number of structures composed of connective tissue, including ligaments, fascia, and tendons which can affect flexibility. Ligaments function to attach bone to bone, acting primarily to support a joint by holding the bones in place, and are comprised mostly of collagenous tissue with elastic fibers woven amongst the parallel fibers. This allows for strength to resist applied forces and flexibility to allow freedom of movement (Elson, 1975). Fascia designates all connective tissue structures which are not otherwise specifically named. The fascia that surrounds and separates the muscle into different groups is named according to where it is found: epimysium surrounds the entire muscle; perimysium encloses muscle fiber bundles called fasciculi; endomysium surrounds each muscle fiber; and the sarcolemma covers the sarcomere, the functional unit of the muscle. The intermeshing of these connective tissues is responsible

for the resistance to stretch in muscle; as the muscle is stretched, the connective tissue become more taut. Fascia gives muscle the ability to change length; during passive motion, the fascia accounts for 41% of the total resistance to flexibility and stretching (Johns & Wright, 1962).

Tendons attach muscles to bones and function to transmit tension to the bones. Because the collagenous fibrils of the tendon are almost all oriented toward the longitudinal axis which is also the direction of normal physiological strain, tendons are well adapted to resist movement in any one direction (Elson, 1975).

Thus, connective tissue exists in the muscle as sarcolemma, around the whole muscle, in surrounding muscle bundles, and in the muscle crossbridges (Safran et al., 1989). The tension which develops in muscle in response to large stretch is due to connective tissue versus the contractile proteins (Borg & Caulfield, 1980). Tension due to the active component in activated muscle must occur within the physiological length of the muscle with explanation to follow, but beyond that, most of the tension produced as a result of a large stretch is due to connective tissue elements (Ralston et al., 1947). As well, connective tissue elements are responsible for most of the biomechanical properties of the musculotendinous unit during the stretch (Casella, 1950). Because the connective tissues play a significant role in limiting flexibility, they must be fully stretched with the muscle relaxed for greater development of flexibility. Elasticity and extensibility of connective tissue have been shown to increase tissue flexibility, resulting in grater force and length of stretch to tear the musculotendinous unit (Gross, 1964; Lehmann, 1970; Cuillo, 1983; Taylor, 1985a,b).

The contractile components of muscle are also a limiting factor for flexibility.

Sarcomeres are the functional units of the myofibrils composed of sliding and interlocking actin and myosin myofilaments. Muscles are hypothesized to contract according to a sliding myofilament theory (Huxley, 1969). In resting muscle fiber, actin and myosin filaments are inhibited from interacting with each other due to the presence of troponin . When a muscle fiber receives a nerve impulse, it causes a release of calcium ions stored in the muscle; in the presence of ATP, the calcium is immediately taken up by the troponin molecules on the actin filaments, triggering changes in the structure of the actin myofilament . An electrostatic bond is created between the actin and myosin filaments, activating an enzyme that functions to provide energy for the sliding of the myosin filament toward the centre of the sarcomere through the movement of myosin cross-bridges. As this occurs, the muscle develops tension and shortens. When the muscle fiber stops receiving nerve impulses, it relaxes - myosin cross-bridges detach from active sites on the actin filament and the internal elastic force that builds within the myofibrils during muscle contraction is released, restoring myofilaments to their original length .

When muscles are stretched, the opposite occurs. Myofilaments reverse the interlocking processes which take place during contraction, and research has shown that the contractile component of the sarcometer is capable of stretching to over 150% of its resting state length, enabling muscles to move through a wide range of motion (Alter, 1988). Muscle fibers are incapable of lengthening or stretching themselves and require a force to be received from outside the muscle itself, including gravity, motion/momentum, antagonistic muscles on the opposite side of the joint, and/or another person or part of the body (Alter, 1988).

Stretch Proprioceptor Organs

Stretch is sensed in the muscle by three types of proprioceptor organs: muscle spindles, Golgi tendon organs, and joint receptors. Muscle spindles are encapsulated structures which run parallel to the muscle fiber (Sherwood, 1989). Spindles send information to the central nervous system about the degree of stretch in the muscle in which they are embedded, providing the muscles with information as to the number of motor units required to work to overcome a given resistance - the greater the load, the greater the stretch and the number of motor units recruited. The spindle is sensitive to the rate of change in length of the muscle and also to the final length attained in the muscle fiber.

The muscle spindle can also be activated by gamma motor neurons which supply the contractile ends of the spindles; these nerves can be directly stimulated by motor centres in the cerebral cortex of the brain (Bowman et al., 1985). Motor nerve impulses from these centres result in contraction at the ends of the spindles, causing the center portion of the spindle to stretch and stimulate the sensory nerve. The ability of the spindle to activate itself provides a sensitive feedback system for the execution of smooth, voluntary movement .

Golgi tendon organs (GTO's) are located in tendon fibers near the junction of muscle and tendon fibers (Sherwood, 1989). GTO's are much less sensitive to stretch than spindles and require a stronger stretch before being activated - because of their location, GTO's are activated largely by the stretch occuring during the contraction of the muscles in tendons in which they are located. When stretched, sensory information is sent to the

central nervous system triggering the contracting muscle to relax. The activation of GTO's results in the inhibition of the muscles and serves as a protective function against the lifting of heavy loads which may produce injury.

Joint receptors supply information to the central nervous system regarding the joint, including joint angles, joint acceleration, and the degree of deformation brought about by pressure, allowing a sense of body awareness, limb position and providing automatic reflexes regarding posture (Fox & Mathews, 1981).

The Stretch Reflex

The basic neural mechanism for maintaining muscle tone and preventing injury is the myotatic or stretch reflex which is initated whenever a muscle is stretched (Holland, 1968; Beaulieu, 1980; Prentice, 1983). When a muscle is stretched, muscle fibers and muscle spindles are also stretched, resulting in impulses being discharged by the spindles. The information is passed to the motor nerve cells which synapse with these afferent spindle fibers in the spinal cord, and impulses are sent back to the same muscle fibers, causing the muscle to contract. For this reason, ballistic stretches which require the muscle to stretch quickly and forcefully and elicit the stretch reflex should be avoided, even though the elastic component of the musculotendinous unit is facilitated by this type of stretch (LeBan, 1962; Warren et al., 1971, 1976). For most effective stretching, parts of the muscle performing the contraction should be totally relaxed so connective tissue can also be stretched (Iashvili, 1983). Thus, slow or static stretching would seem more appropriate for developing flexibility.

Reciprocal Innervation

Muscles usually work cooperatively together in pairs via reciprocal innervation, so that when one set of muscles, the agonistic, are shortening or contracting, the opposing antagonistic muscle group(s) are lengthening or relaxing (Junge, 1981). The nerves supplying the muscle pairs operate in such a way that when one muscle (agonist) of the pair receives an impulse to contract, the other group does not, inhibiting the antagonist group and causing it to relax. Without reciprocal innervation, coordinated muscle activity would not occur.

Inverse Myotatic Reflex

If the intensity of a stretch on the tendon exceeds a critical limit, an immediate reflex occurs to inhibit the motor neurons innervating the muscle and the muscle relaxes, releasing the excess tension (Sherwood, 1989). The impulses from the GTO's are powerful enough to override impulses from the muscle spindles, producing the inverse myotatic reflex which serves as a protective mechanism to prevent the tendon from tearing away from the bone. This reflex may be used to stretch a muscle beyond the point where considerable tension is reached in the muscle and where stretching further may be prevented by the tension of the antagonistic muscle (Prentice, 1982). Autogenic inhibition refers to the relaxation of the antagonist muscle during muscle contractions (Safran et al., 1989).

Stretching refers to the process of elongating or lengthening the muscle, and stretching exercises are performed in a variety of ways depending on the goals, abilities, and training state of the individual. Any stretch held longer than six seconds will activate the inverse myotatic or stretch reflex and cause the muscle being stretched to relax (Prentice, 1982). Although Iashvili (1983) has shown that active flexibility has a higher correlation to the level of sports achievement than passive, passive or static stretching, and proprioceptive neuromuscular facilitation (PNF) techniques appear to be the most widely accepted methods with static methods considered the safest (Shellock & Prentice, 1985).

Static stretching involves holding a final stretched position for a given amount of time (Holland, 1968; Harris, 1969; Cornelius, 1981). The advantages of this method include minimal danger of tissue damage, less energy requirement than other ie. ballistic methods, less muscle soreness (deVries, 1961, 1962), more relief from muscular distress, more time to reset the sensitivity of the stretch reflex, semipermanent to permanent deformation of the musculotendinous unit (LeBan, 1962; Warren et al., 1971, 1976), and greater muscle relaxation via GTO's if stretch held long enough. If GTO's are stretched, inhibition of contraction ie. relaxation occurs in the muscles involved in the stretch.

Both static and ballistic stretching methods have been shown to improve flexibility but some of the arguments against the use of ballistic stretching techniques are: increasing chance of soreness and injury, failure to provide adequate time for various tissues to adapt to the stretch imposed or for the myotatic reflex to occur, and initiation of the stretch reflex via the muscle spindles and increases muscular tension, making it more difficult to stretch out connective tissues (Holland, 1968; Harris, 1969; Lehmann, 1970).

Proprioceptive neuromuscular facilitation (PNF) refers to a range of similar strategies

which may be used to increase the flexibility of a joint. PNF techniques were originally used by physical therapists for treating patients with various types of neuromuscular paralysis (Bobath, 1955; Knott & Voss, 1968). These methods were subsequently adapted for use to develop flexibility (Prentice, 1982). Two of the most widely used techniques are the contract-relax (hold-relax) and contract-relax-contract (hold-relax-contract) methods.

The hold-relax method starts with the muscle in question in a lengthened position; the muscle is first placed under a gentle stretch followed by a maximal voluntary contraction lasting from six to fifteen seconds against resistance, usually supplied by a partner. Following this, the muscle is allowed to relax for a brief period and is then passively stretched through the newly gained range of motion by the partner.

The hold-relax-contract method is similar to the hold-relax method with the exception that the relaxation phase is followed by an active contraction of the agonist, the opposite muscle group of that being stretched. The entire process of both methods can be repeated. PNF techniques offer a wide range of advantages and benefits, including enhanced flexibility over other methods, development of patterns for coordinated motion, and use of several of the neurophysiological mechanisms already discussed. However, several disadvantages make this method undesirable, including greater risk of injury compared to static methods, ranging from pulled muscles to cardiovascular complications, and the need for a knowledgeable and skilled partner (Shellock & Prentice, 1985). Thus the static stretching method seems to be the method of choice for both safety and gains in flexibility.

FLEXIBILITY AND ANAEROBIC PERFORMANCE

Flexibility could theoretically affect anaerobic performance by permanently and progressively increasing the usable range of motion of a joint or joints over a certain time period (Safran et al., 1989). In contrast, the stretching portion of a warm-up is not designed to increase flexibility per se but is theoretically included to relax the muscles to improve performance and act as a preventitive measure to reduce the risk of injury during the activity (Glick, 1980). Lengthening or relaxing the muscle could increase the range of motion of the joint and muscle groups in question and increase the amount of force the muscle could develop over a certain period of time. Stretching the muscle prior to an event could aid in relaxing or lengthening the muscle, making it more supple and decreasing the chance of injury. Unfortunately, there is little literature regarding the role of flexibility in enhancing or hindering performance.

Most exercise specialists recommend the use of some type of warm-up before stretching and before performance to provide the athlete with a period of adjustment from rest to exercise (LeBan, 1962; Rigby, 1964; Shellock & Prentice, 1985; Safran et al., 1989). Thus combining warm-up and stretching, both designed to reduce the chances of injury and improve performance, should theoretically benefit performance. Only one study was found in which stretching was regulated, described, and included as part of a warm-up or experimental protocol. Pacheco (1957) found that isometric stretches of the hip and leg, held for a total of 3 minutes with 15 second per stretch improved performance in a vertical jump test. In a study of joint looseness and performance, Marshall et al. (1980) found significant correlations between performance in a number of tasks testing

agility, strength, power, and endurance and joint looseness - the trait, but not to the act of stretching before performance. It is understandable why some protocols could not be carried out on human subjects but it would be impossible to design a safe test battery.

Two studies were found which included stretching in their warm-up protocol. deVries (1958) included stretching in his calisthenics warm-up and found that a calisthenics warm-up did significantly improve performance times in some 100-yard swimming events. The effects of the calisthenic exercises which were not stretching, and the stretching exercises could not be separated or differentiated in this study.

Howard et al (1966) included unspecified stretching exercises as part of a warm-up in a study which examined the effect of warm-up on heart rate during exercise. Stretching was included in a warm-up combined with jogging, and calisthenics, for a total of 10-15 minutes. The authors found no significant differences between the group which warmed up and the group which did not; again the inclusion of stretching in a multi-faceted warm-up with no description of protocol makes it difficult to assess the effect of warm up or flexibility on performance.

The question of what role flexibility and stretching have in anaerobic performance thus has still not been satisfactorily answered in the literature.

APPENDIX D

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January 31, 1991