# THE EFFECT OF PROLONGED CYCLING AND RUNNING ON SELECTED CARDIOVASCULAR RESPONSES



by

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We accept this thesis as conforming to the required standard

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**ABSTRACT**

To better understand the cardiovascular responses to prolonged exercise 10 male recreational triathletes performed three exercise sessions consisting of a 110 minute control cycle (CC), a 110 minute control run (RC), and a sequential cycle/run session (C/R) (a 70 minute cycle followed by a 40 minute run). Selected physiological responses were monitored every 15 min. After increases at the onset of exercise there were no significant differences in ventilation ($\dot{V}E$), cardiac output ($\dot{Q}$), and plasma concentration (PC) within any of the exercise sessions. For CC, oxygen consumption ($\dot{V}O_2$), arteriovenous oxygen difference ($(a-v)O_2diff$), heart rate (HR), and rectal temperature ($T_r$) all showed significant changes from those observed 3 min into the exercise session. The mean change in HR at the end of the CC was 9 ± 6 beats·min$^{-1}$. For the RC, HR, $T_r$ and stroke volume (SV) changed significantly between 3 and 110 min of the session. The mean change in HR at the end of the RC was 12 ± 6 beats·min$^{-1}$. When comparing sessions the first 40 min of RC did not yield significantly higher HR's than those observed for the run portion of the C/R. In contrast, the last 40 min of the RC elicited significantly greater HR's than the run portion of the C/R (means = 173 ± 8 beats·min$^{-1}$ and 162 ± 8 beat·min$^{-1}$,

respectively. The smaller magnitude of cardiovascular drift during the CC was possibly related to the lower thermal demand of the activity reflected in the mean $T_r$ and stabilization at 60 min of the session. Although the absolute change in HR was greater for the RC than for the CC when expressed in terms of percent change both the RC and the CC sessions were very similar (7% and 6% change, respectively). The results indicate that the RC elicited greater cardiovascular and thermal responses than the CC and the C/R sessions.

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There are several people I would like to acknowledge for their time and effort they have put into this project. The first is my supervisor Dr. David Docherty. Thank-you for giving me the direction I needed to complete what I was beginning to think was an impossible project. Second, I would like to thank all my fellow graduate students and the work studies, in particular Wendy Pethick and Roisheen Doherty, for the long hours they put into my data collection. Last but not least, Gladys Whittal, who kept me in graduate school, literally.

# DEDICATION

I had to practice many values in writing this thesis. Among the many, these were the most important. The first was to be diligent. The second was to persevere, and the third was to be honest to myself of my capabilities.

These are values my mother taught me.

For my mother

# INTRODUCTION

The cardiovascular adjustment to prolonged exercise is characterized by a progressive rise in heart rate over time even as work rate remains constant (Senay & Pivarnik, 1991). This phenomenon is often referred to as cardiovascular drift (CD). Shaffrath and Adams (1984) define CD as the progressive circulatory changes that take place with prolonged single load exercise. Their term for CD was limited to the commonly reported subset of circulatory changes that take place during this type of exercise. Circulatory changes include such parameters as increased heart rate, decreased stroke volume, maintained cardiac output and decreased mean arterial pressure. These factors are commonly associated with exercise-induced hyperthermia.

CD has also been described by Kalis, Freund, Joyner, Jilka, Nittolo and Wilmore (1988) as "$O_2$ drift", a phenomenon that similarly occurred during constant-rate submaximal exercise. These authors suggested that CD results from both circulatory and non-circulatory factors. The circulatory factors include those involved with exercise-induced hyperthermia. The non-circulatory factors include a reduction in the mechanical efficiency of the heart, increased ventilation, increased circulating catecholamines, altered substrate utilization, and increased circulating lactate.

CD has been most commonly shown to be reflected by

changes in the following variables: decreased stroke volume; decreased blood pressure; decreased plasma volume; and, increased oxygen consumption. These variables are associated with the secondary rise in heart rate observed during prolonged exercise (Adams, Fox, Fry, & MacDonald, 1975; Brenglemann, 1983; Harrison, Edwards, & Leitch, 1975; Rowell, 1975).

The cardiovascular response to prolonged single load exercise may be different for different modes of exercise, such as cycling and running. As previously described, CD may be observed during a single prolonged event, (60 minutes or longer), but few studies have examined the CD response during exercise consisting of more than one mode.

It has been observed that heart rate increases with CD even though power output remains the same. Therefore, heart rate achieved at the end of a constant load exercise bout would normally be higher as compared to the beginning. This has a number of implications to both single mode and combined mode exercise. When CD occurs within a single mode exercise in which power output is maintained the heart rate observed at the onset of exercise will be different (lower) than the heart rate observed at the end. Similarly CD may occur during sequential exercise but the CD that occurs in the first event may impact on the cardiovascular response at the onset of the later event as well as at the end.

From a practical perspective the portable heart rate

monitor is becoming a common training aid and athletes in multi-sport events, such as the triathlon and duathlon, often use them to enhance both training and performance. The use of such information due to the phenomenon of CD may provide misleading feedback to the athlete. For example, an athlete trying to maintain a specific heart rate may find that as the activity continues their power output will decrease, (running or cycling velocity may decrease). If power output is to be maintained the athlete must allow his/her heart rate to increase. However, the magnitude of the increase in heart rate that would allow maintenance of power output has not been established.

The aims of this research were to investigate the cardiovascular responses during prolonged cycling and running of 110 minutes as single mode activities and the effects of prior cycling (70 minutes) on the cardiovascular responses during running (40 minutes).

The specific purposes of this study were to investigate:

1. The magnitude of CD during a single bout of exercise, of cycling and running, 110 minutes in duration, performed in standardized conditions at an intensity between 60 and 70% $\dot{V}O_2max$.

2. The effect of prior cycling exercise (70 minutes) on the

cardiovascular responses to running exercise (40 minutes) when the two are performed consecutively.

## RESEARCH QUESTIONS

This study examined the cardiovascular responses to prolonged running and cycling. As well, cardiovascular responses to prolonged exercise consisting of more than one mode were investigated in an attempt to answer the following questions.

1. What is the magnitude of cardiovascular drift elicited over a separate 110 minute cycle and 110 minute run at the same intensity relative to $\dot{V}O_2max$ in that mode.

2. What are the effects of prior cycling exercise of 70 minutes on the cardiovascular responses to 40 minutes of running when compared to running alone?

3. Is the magnitude of cardiovascular drift greater for prolonged exercise consisting of one mode or two modes?

## DEFINITIONS OF TERMS

| | |
|---|---|
| Cardiovascular drift: | the progressive circulatory and thermoregulatory changes that take place with constant load prolonged exercise at an intensity greater than 50% $\dot{V}O_2$max (Senay & Pivarnik, 1991) |
| Prolonged exercise: | continuous exercise which lasts for 60 minutes or longer (Senay & Pivarnik, 1991) |
| $\dot{V}O_2$max | maximal volume of oxygen consumed when two of the following criteria are observed: a plateau in oxygen consumption with an increase in power output, a respiratory exchange ratio (RER) in excess of 1.15, and volitional exhaustion (Thoden, 1991) |
| Plasma Concentration | An estimation of plasma volume from from the values of haematocrit and haemoglobin according to the methods of Dill and Costill (1974). |

## DELIMITATIONS

To assist with the investigation of the experimental hypotheses the scope of this study was restricted to the following:

1. Ten subjects;
2. Males ranging in age from 23-43 years old;
3. Subjects were triathletes or duathletes with two years of competitive experience or more;
4. All subjects were from the Vancouver Island area; and
5. Data collection occurred between January, 1994 and February 1994.

## LIMITATIONS

Every attempt was made to control for outside influences which may have affected the validity or reliability of the results. This study was conducted within the context of the following limitations:

1. Subjects who took part in this investigation did so on a voluntary basis and completed all measurements as requested. It was assumed that all subjects exerted maximal effort during the tests, adhered to instructions regarding rest prior to laboratory measurements, and complied with nutritional guidelines;
2. Determination of maximal oxygen consumption ($\dot{V}O_2max$). Wherever possible $\dot{V}O_2max$ was determined by the given

criteria. If subjects were unable to continue the test even when no other criteria was met, the value at exhaustion was used as the maximal value;

3. Results are generalizable to male triathletes and duathletes who have at least two years or more experience.

# METHODS

## Subjects

The sample consisted of male triathletes and duathletes (n=10). Each subject underwent an orientation session in the laboratory to become familiarized with the testing protocols and equipment. As well, each subject signed an informed consent in compliance with the University of Victoria Committee on Research and Other Activities Involving Human Subjects.

## Experimental Design

Each subject participated in five laboratory sessions:

1. A cycle ergometer test (CT) to establish ventilatory threshold (VT) and maximal oxygen consumption ($\dot{V}O_2max$).
2. A treadmill run test (RT) to establish VT and $\dot{V}O_2max$.
3. A cycle control test (CC) which consisted of 110 minute continuous cycling.
4. A run control test (RC) which consisted of 110 minutes of continuous running.
5. A sequential 70 minutes of cycling and 40 minutes of running (C/R).

Subjects were randomly assigned into two groups with group A beginning with the cycle testing and group B with the run on the treadmill. Tests were administered over a one month period. Forty-eight to 72 hours separated the

$\dot{V}O_2$max tests. Control tests were performed over the following three week period with a minimum of 72 hours and a maximum of 2 weeks separating each control test. Subjects performed each of their tests at approximately the same time of day on each of their test days.

Subjects reported to the laboratory 30 minutes prior to testing on the control and C/R days and were weighed in order to monitor weight fluctuations. They were also instructed to be in the post-absorbtive state. Due to the length of the study subjects were permitted to maintain their level of training during this time although they were directed to refrain from any training for 48 hours prior to testing. To accomplish glycogen repletion on a daily basis the subjects were instructed to consume between 8-10 g carbohydrate $\cdot kg^{-1}$ per day, approximately 60-70% of their total caloric intake for the duration of the study (Applegate, 1989). Subjects were provided with a log book for the purpose of dietary and weight record. When necessary the subjects limited themselves to a light meal 1-4 hours prior to exercise testing and did not consume beverages containing caffeine.

## Procedures

### $\dot{V}O_2$max and Ventilatory Threshold tests

CT consisted of single test to measure $\dot{V}O_2$max and VT on

a Monarch cycle ergometer. All subjects warmed-up for five minutes at a power output of 80 Watts with pedal frequency maintained at 80 revolutions per minute. The test began at a resistance of 150-175 W. The power output was increased by 45 W every two minutes until ventilatory threshold was observed. At that time the resistance was increased by 45 W every minute until $\dot{V}O_2$max was reached.

$\dot{V}O_2$max and ventilatory threshold for running were measured on a treadmill using a continuous incremental protocol. Subjects were allowed a five minute warm-up (walking or running). Starting speed was determined after consideration of the individual's 10 km race pace and was increased by 13.3m$\cdot$min$^{-1}$ every two minutes until ventilatory threshold was observed. At this time the grade was increased by one percent every minute until the attainment of $\dot{V}O_2$max.

The $\dot{V}O_2$max test was terminated when two of the following criteria were obtained: a plateau in oxygen consumption with an increase in power output, a respiratory exchange ratio (RER) in excess of 1.15, and volitional exhaustion (Thoden, 1991). The primary criteria for determination of ventilatory threshold was: a significant change in $\dot{V}E/\dot{V}O_2$ with no change in $\dot{V}E/\dot{V}CO_2$; a non-linear increase in $\dot{V}E$; and an RER greater than 1.00 (Wasserman, Whipp, Koyal, & Beaver, 1973). The criteria were determined by the primary investigator by visual inspection.

**Control Tests**

From the incremental exercise tests, power outputs were determined for the CC and RC. The intensity chosen for the CC corresponded to the power output achieved at 60 percent $\dot{V}O_2max$. The power output for the RC was set between 60 and 70% $\dot{V}O_2max$. For the RC speed was set at 60% $\dot{V}O_2max$ unless the subjects perceived this as too slow. At this time the speed was increased by $13.3m \cdot min^{-1}$. The identical power ouputs were used for the C/R session.

CC consisted of 110 minutes of continuous cycling on the cycle ergometer. RC consisted of 110 minutes of treadmill running. C/R consisted of a consecutive 70 minute cycle and a 40 minute treadmill run separated by a standardized 5 minute transition time.

**Physiolocial Measures**

Subjects prepared for the CC, RC and C/R sessions like they would for any competition. The cycling and running events were performed in a laboratory. Fans were directed towards the subjects to simulate actual cycling and running conditions.

During exercise tests subjects breathed through a low resistance respiratory valve. Oxygen ($O_2$) and carbon dioxide ($CO_2$) were collected and analyzed by an Horizon cardiorespiratory system. The Horizon was calibrated with primary standard gases immediately prior to all $\dot{V}O_2max$ tests

and throughout control sessions. The physiological measures, minute ventilation ($\dot{V}E$), oxygen consumption ($\dot{V}O_2$), RER, and HR were recorded every 20 seconds during maximal tests and every 15 minutes during the cycle and run control sessions and the sequential cycle/run while, core temperature ($T_r$), and plasma concentration (PC) were measured pre- and post-test as well as every 15 minutes during the CC, RC and C/R. Cardiac output ($\dot{Q}$) was measured every 15 minutes during the control and C/R sessions.

HR was recorded by a Polar Vantage Pro II heart rate monitor. Core temperature was measured using a Yellow Springs telethermometer connected to a rectal thermistor inserted approximately 10cm past the anal sphincter. A 20uL blood sample was obtained by finger prick prior to exercise in the same postural position in which the exercise session took place. All exercise samples were taken in the position of the mode of exercise. Haematocrit was determined in duplicate with a micro-haematocrit centrifuge and was corrected for the plasma trapped with the packed red cells. Percent changes in plasma concentration, red cell volume, and plasma concentration were estimated from the values for haemoglobin and haematocrit according to the method of Dill and Costill (1974) (see appendix C). Haemoglobin was determined in duplicate by the cyanmethemoglobin method.

Cardiac output ($\dot{Q}$) was determined by the $CO_2$ rebreathing technique. In determining $\dot{Q}$ the subjects

breathed from a five to seven litre anaesthesia bag containing a mixture of 11% to 14% $CO_2$, depending on the metabolic rate, until a "plateau" in the partial pressure of $CO_2$ between the lung and the bag was attained (Jones, 1988). A difference of less than 1mmHg during the first six to eight seconds of the rebreathing maneuver was considered indicative of an equilibrium in the $CO_2$ concentration between mixed venous blood and alveolar gas. The steady state in the partial pressure of end-tidal $CO_2$ ($P_{ET}CO_2$) obtained prior to the initiation of the maneuver was considered to be an indication of the arterial $PCO_2$. The value of $\dot{Q}$ was calculated from the indirect Fick equation, using the values of $CO_2$ production, and the estimated difference in the venous and arterial $CO_2$ concentrations under the exercise conditions. All these calculations were performed using a computerized software package that was available with the metabolic cart. When a plateau did not occur the estimation of $PCO_2$ was made by extrapolation of the line joining the points for expired $PCO_2$ at 8 and 12 seconds of rebreathing to that at 20 seconds. This was indicative of the equilibrium value within 2 mmHg (Rebuck, 1973). Test-retest measurements were performed on a separate group of subjects exercising at approximately the same intensity (60% of $\dot{V}O_2max$) and produced an intraclass reliability coefficient of 0.89. Marks, Katch, Rocchini, Beekman, and Rosenthal (1985) reported reliability

coefficient ranging between 0.86 to 0.96. The present value falls within this range. Marks et al. (1985) also reviewed several investigations which utilized the $CO_2$ rebreathing method for determination of $\dot{Q}$ and reported a mean validity coefficient of 0.82 compared to the direct Fick method.

Gatorade was available to the subjects **ad libitum** and was measured for total intake during the CC, RC, and C/R sessions. Towelled dry nude body weight was measured before and after all sessions, as well as during the transition of the cycle/run. Body water loss was calculated from total water intake and the change in body weight.

**Statisical Procedures**

Statistical differences between the CC, RC, and C/R sessions were determined by analysis of variance (ANOVA) for repeated measures. Level of significance was set at $p<0.05$. Where statistical significance was noted, critical differences were calculated using a Newman-Keuls post hoc test.

Pearson product moment correlations were conducted to determine the relationship between the change scores for all variables at each time interval compared to the intial 3 minute value for all sessions.

# RESULTS

The mean (SD) for the subjects physical characteristics and physiological responses to the cycle and run $\dot{V}O_2max$ tests are presented in Table 1. The sample consisted of triathletes and duathletes of varying abilities who had been training for these type of events for an average of 5.0 (1.5) years. The mean age of the sample was 30 (7) years and the mean body mass was 74.6 (6.8) kg. The mean $\dot{V}O_2max$ for the cycle was 4.03 (0.43) $L \cdot min^{-1}$ and the mean $\dot{V}O_2max$ for running was 57.5 (5.3) $mL \cdot kg \cdot min^{-1}$.

Table 2 presents the mean power output (PO) (SE) expressed in Watts for the CC and the cycle portion of the C/R. The mean PO for the CC was 175 (30) W. The PO at 15 min was not significantly different from values reported at any other time except for 110 min where there was a significant increase. The mean PO for the cycle portion of the C/R was 175 (30) which was not significantly higher than the mean reported for the CC.

Figure 1 represents the mean heart rate (HR) responses for the CC, RC, and C/R sessions. The mean HR for the CC was 150 (12) $beats \cdot min^{-1}$. During the CC the only significant differences observed between consecutive time intervals were between first exercise HR recorded at 3 min and those at 15 and 110 min. The mean change in HR observed from 3 to 15 min was 8 (5) $beats \cdot min^{-1}$. The mean change in HR from 3

Table 1: Mean (SD) of physical characteristics of subject pool of recreational triathletes and duathletes (n=10).

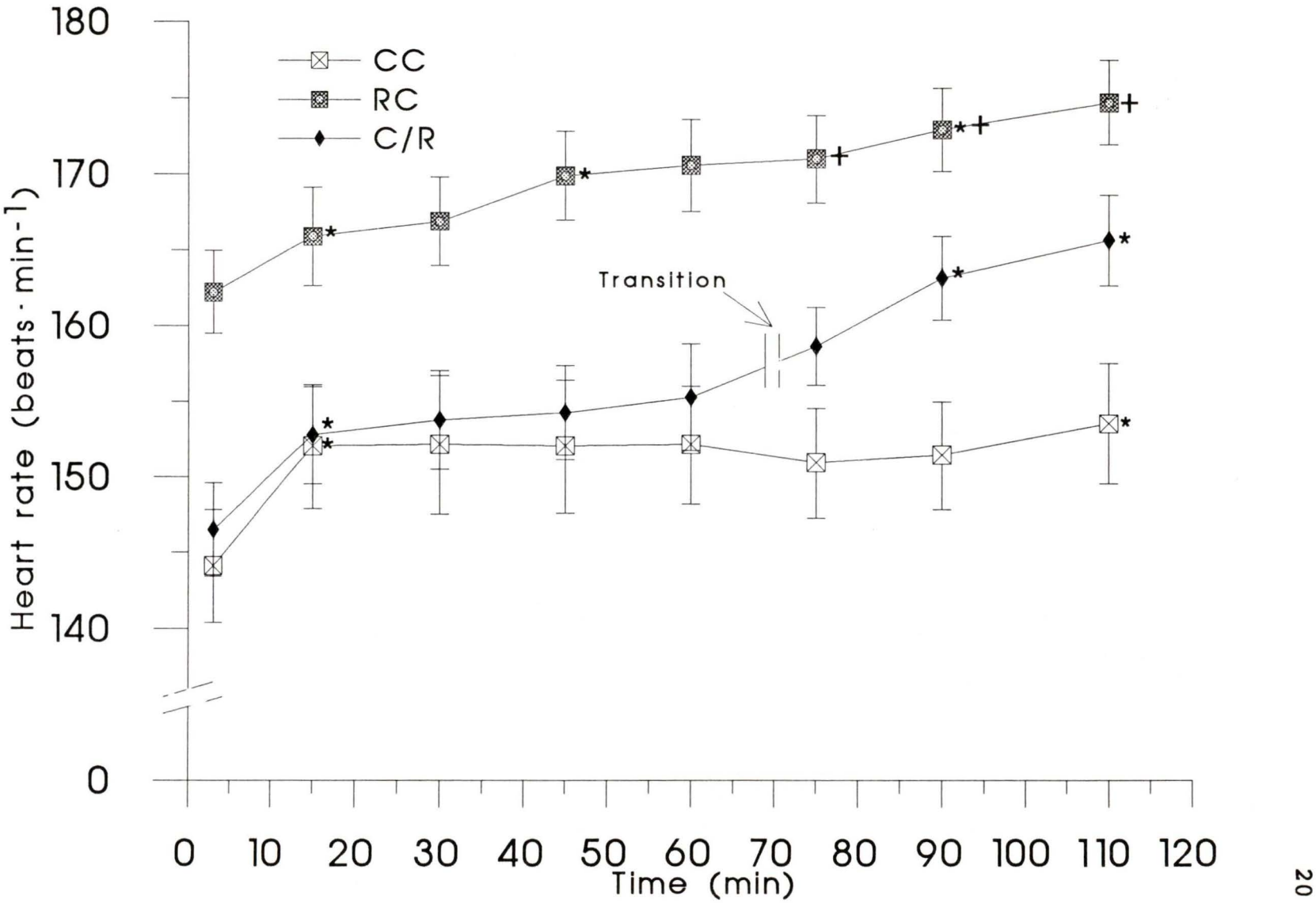
| | Mean | Range |
|---|---|---|
| Age (years) | 30 (6.5) | 23 - 43 |
| Body Mass (kg) | 74.6 (6.8) | 62.5 - 86.0 |
| Height (cm) | 176.7 (5.5) | 170.1 - 186.4 |
| Sum of Skinfolds (mm) | 86.0 (38.8) | 37.7 - 146.2 |
| BSA ($m^2$) | 1.88 (0.10) | 1.70 - 2.01 |
| $\dot{V}O_2$max Cycle ($L \cdot min^{-1}$) | 4.03 (0.43) | 3.42 - 4.84 |
| $\dot{V}O_2$max Cycle ($mL \cdot kg \cdot min^{-1}$) | 54.0 (5.4) | 45.8 - 64.9 |
| $\dot{V}O_2$max Run ($L \cdot min^{-1}$) | 4.29 (6.8) | 3.65 - 5.25 |
| $\dot{V}O_2$max Run ($mL \cdot kg \cdot min^{-1}$) | 57.5 (5.3) | 48.9 - 70.3 |

Table 2: Cycling power output (SE) expressed in watts for the control cycle and the cycle portion of the sequential cycle/run sessions.

| Control | | Cycle/run | |
|---|---|---|---|
| Time | X | Time | X |
| 15 | 169 (10.9) | 15 | 176 (11.7) |
| 30 | 168 (9.7) | 30 | 176 (11.5) |
| 45 | 166 (11.7) | 45 | 176 (11.5) |
| 60 | 171 (10.4) | 60 | 174 (11.1) |
| 75 | 168 (10.0) | 70 | 182 (9.5)* |
| 90 | 169 (10.2) | | |
| 110 | 209 (12.6)* | | |
| 0 - 110 | 174 (13.2) | 0 - 70 | 177 (12.3) |

* Demonstrates statistically significant difference ($P<0.05$) within the exercise session.

Figure 1: Mean heart rate responses for the control cycle, run, and cycle/run sessions, n=10 for CC, n=9 for RC, and n=8 for C/R, * significantly different from prior time interval ($p<0.05$), + significantly different from C/R session at the same time interval. S.E. indicated by ⊥.

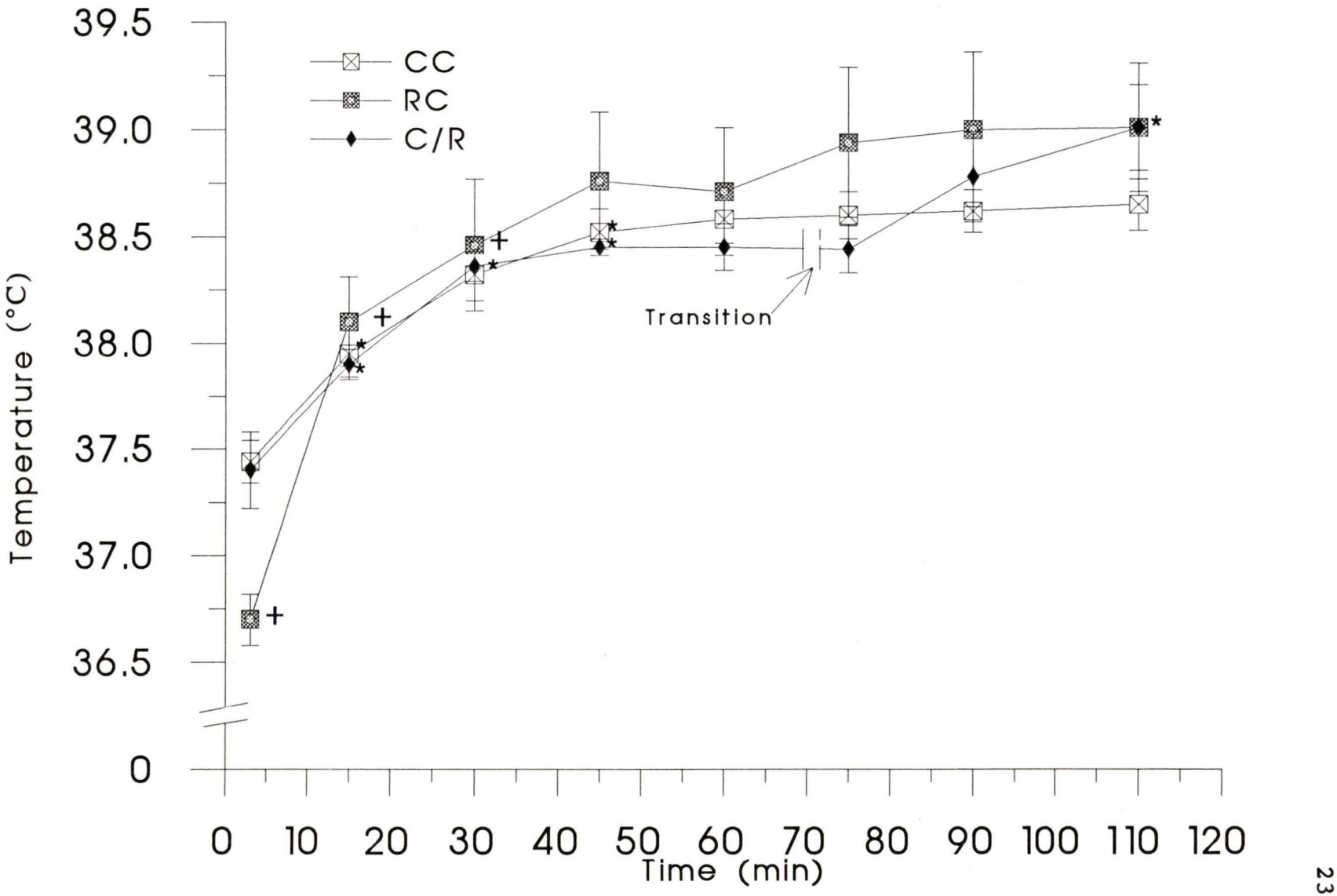

180
170
160
150
140
0
Heart rate (beats · min-1)
CC
RC
C/R
Transition
0
10
20
30
40
50
60
70
80
90
100
110
120
Time (min)

to 110 min was 9 (5) beats·min$^{-1}$. The mean HR for the RC was 169 (9) beats·min$^{-1}$. During the RC significant differences were observed at all times when compared to the HR at 3 min. A significant drift in HR occurred over the course of the RC. The mean difference in HR at 110 min compared to 3 min was 12 (6) beats·min$^{-1}$. Significant differences between successive HR during the RC were observed between 3 and 15, 30 and 45, and 75 and 90 min. The change in HR observed at 45 min correlated significantly to the mean change in $\dot{Q}$, r= 0.70, and mean change in (a-v)$O_2$diff, r = -0.72 ($p<0.05$). The HR change that occurred at 110 min of RC correlated significantly to the mean change in $\dot{V}E$, r= 0.80, SV, r = 0.76, and $\dot{Q}$, r = 0.84.

The mean HR for the cycle portion of the C/R session was 152 (9) beats·min$^{-1}$. This was not significantly different from the CC. The mean HR for the run portion of the C/R session was 162 (8) beat·min$^{-1}$ which was significantly lower than the mean HR observed for the last 40 min of the RC of 172 (8) beats·min$^{-1}$. The mean HR observed for the first 30 min of the RC of 165 (8) beats·min$^{-1}$, was not significantly different from the mean HR observed for the run portion of the C/R session.

Figure 2 shows rectal temperature ($T_r$) responses observed during the CC, RC, and C/R. $T_r$ averaged 38.3

Figure 2: Mean temperature responses for the control cycle, run, and cycle/run sessions, n=10 for CC, n=9 for RC, and n=8 for C/R, * significantly different from prior time interval ($p<0.05$), + significantly different from last 40 min of C/R session. S.E. indicated by $\perp$.


39.5
39.0
38.5
38.0
37.5
37.0
36.5
0
Temperature (°C)
CC
RC
C/R
Transition
0
10
20
30
40
50
60
70
80
90
100
110
120
Time (min)

(0.5), 38.6 (1.0), and 38.0 (0.3) °C, respectively. In all sessions $T_r$ recorded at 3 min differed significantly from $T_r$ recorded at 110 min. There were no significant differences observed for $T_r$ at 110 min between the sessions. The mean difference in $T_r$ from beginning to end for the RC was 1.7°C (0.5°C). During the CC $T_r$ increased until 45 min and then levelled off and maintained a mean $T_r$ of 38.6 (.3) °C until the end of the session. The mean difference in $T_r$ from values recorded at 3 and 45 min was 1.1 (0.4) °C whereas the mean difference in $T_r$ from 45 to 110 min was only 0.1 (0.4) °C. The mean $T_r$ for the first 30 min of the RC (36.5 (0.6) °C) was significantly lower than the mean $T_r$ for the run portion of the C/R (mean = 38.7 (0.5) °C).

Figures 3 and 4 represent cardiac output ($\dot{Q}$) and stroke volume (SV) responses to the exercise sessions. No significant differences were observed between $\dot{Q}$ recorded at 3 min and those observed at 110 min for any of the sessions.

The $\dot{Q}$ observed during the CC remained stable. The mean response observed within the first 3 min of exercise was 24.3 (4.0) $L \cdot min^{-1}$. This was not significantly different from all successive measurements. $\dot{Q}$ was similar for the RC with a mean value of 22.7 (3.0) $L \cdot min^{-1}$. Although no significant differences were observed the mean decrease in $\dot{Q}$ of 1.3 (3.7) $L \cdot min^{-1}$ during the RC correlated significantly

Figure 3: Mean cardiac output responses for the control cycle, run, and cycle/run sessions, n=10 for CC, n=9 for RC, and n=8 for C/R. S.E. indicated by ⊥.

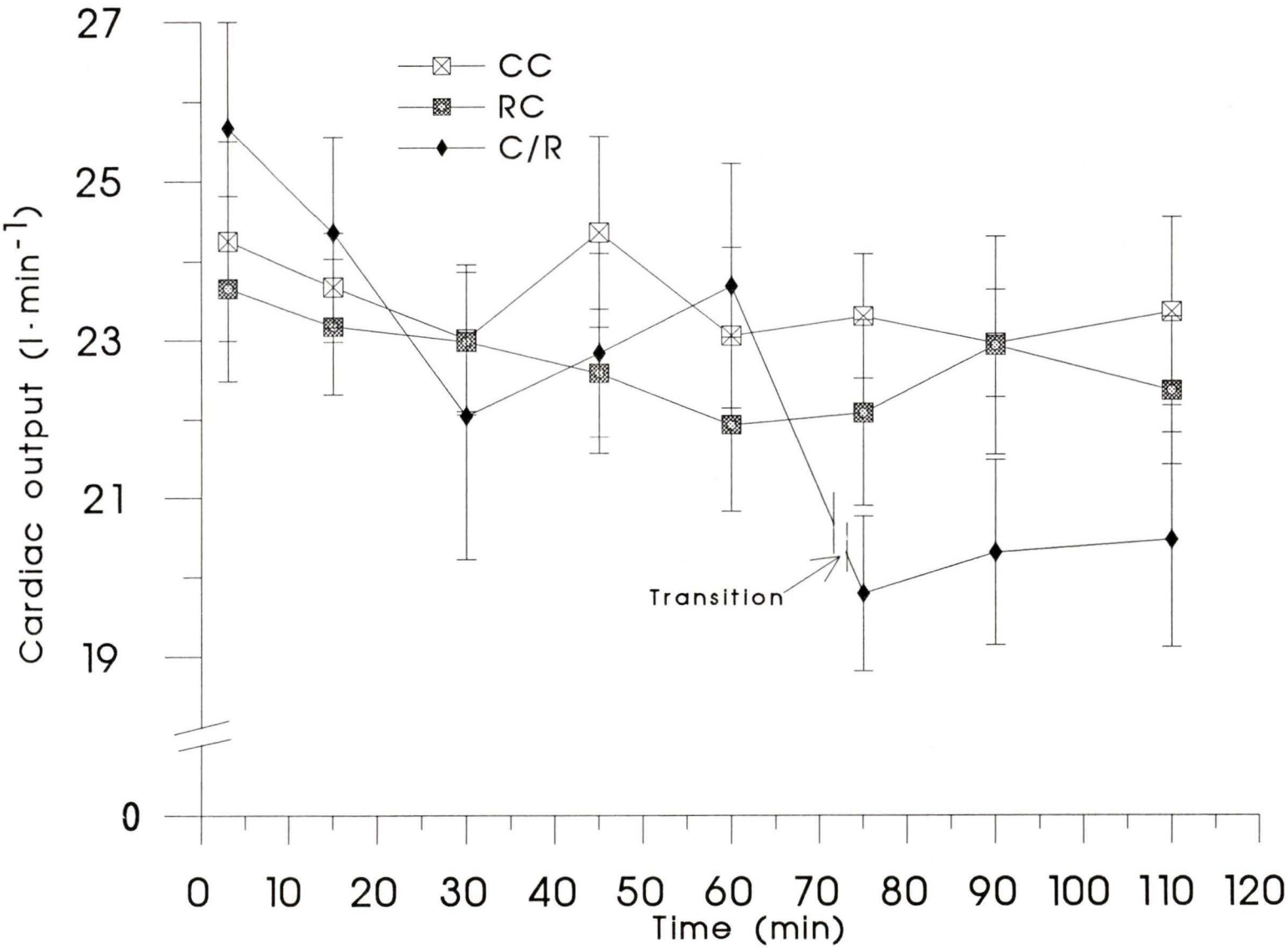
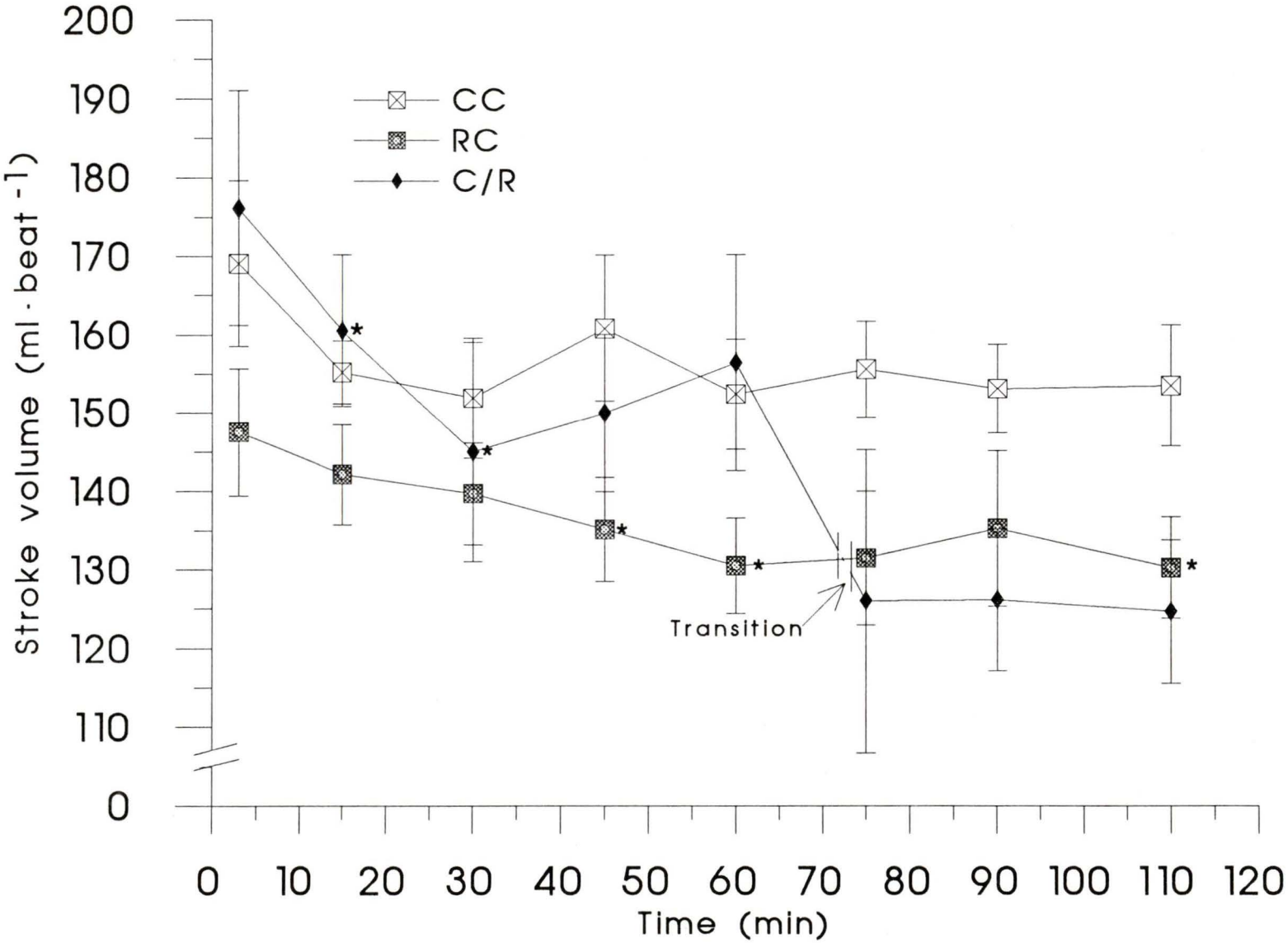

CC
RC
C/R
Transition
Cardiac output (l·min-1)
27
25
23
21
19
0
0 10 20 30 40 50 60 70 80 90 100 110 120
Time (min)

Figure 4: Mean stroke volume responses for the control cycle, run, and cycle/run sessions, n=10 for CC, n=9 for RC, and n=8 for C/R, * significantly different from value at 3 min of session ($p<0.05$). S.E. indicated by ⊥.

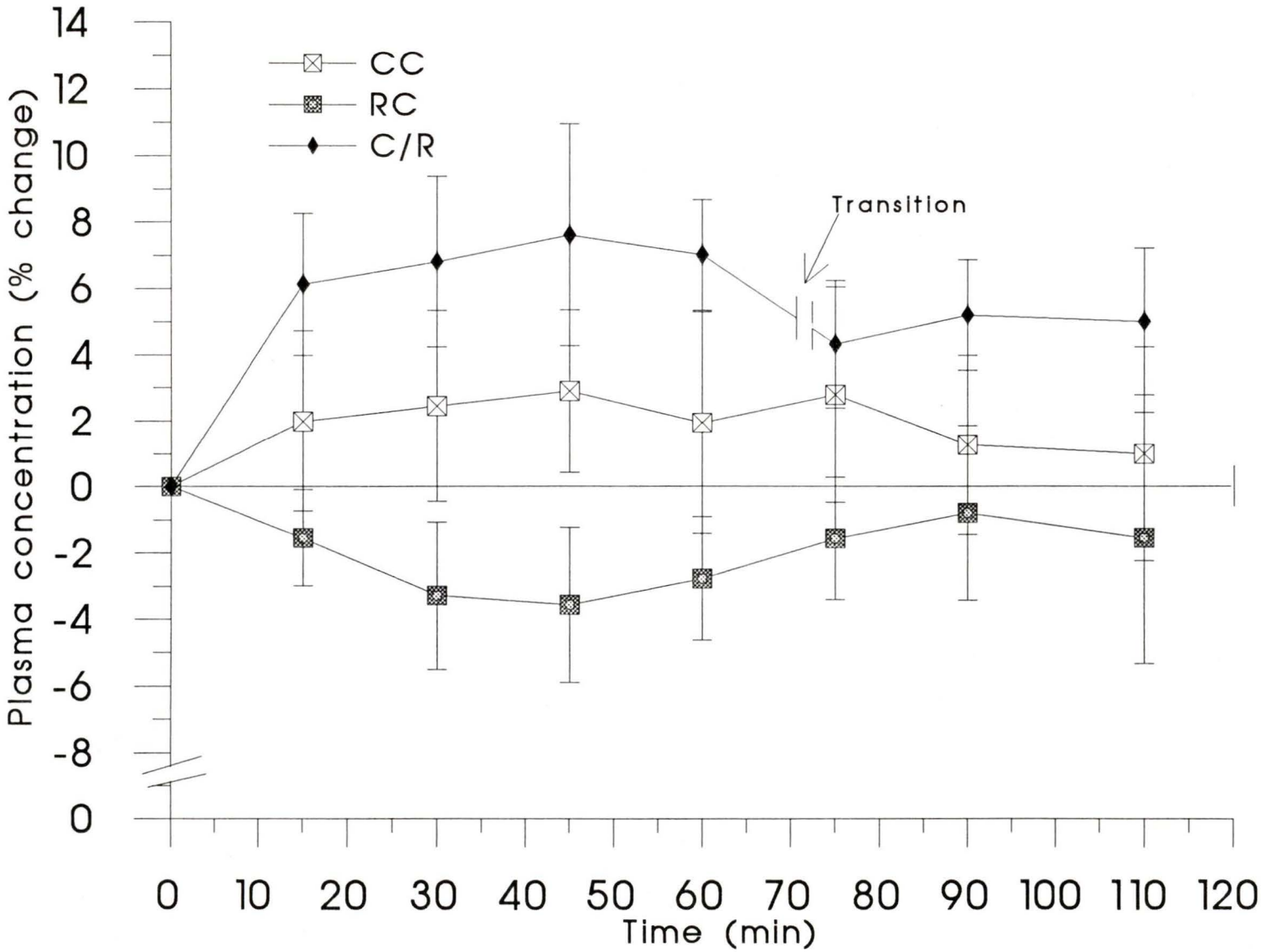

200
190
180
170
160
150
140
130
120
110
0
Stroke volume (ml · beat -1)
CC
RC
C/R
Transition
0 10 20 30 40 50 60 70 80 90 100 110 120
Time (min)

to the mean increase in HR of 12 (6) beats·min$^{-1}$ at the same time (r=0.84). As well, CC $\dot{Q}$ averaged 23.5 (3.1) L·min$^{-1}$ which was not significantly different from the mean $\dot{Q}$ of 23.7 (4.4) L·min$^{-1}$ for the cycle portion of the C/R. During the first 30 min of the RC $\dot{Q}$ averaged 23.3 (2.8) L·min$^{-1}$ which was not significantly different from the $\dot{Q}$ observed during the run portion of the C/R.

During the CC the mean SV of 156.7 (23.4) mL·beat$^{-1}$ remained stable with no significant difference observed within the session. However, during the RC significant decreases in SV from 3 min were observed at 45, 60 and 110 min. The mean decrease in SV of 12.4 mL·beat$^{-1}$ that occurred at 45 min of the RC had a significant correlation (r=0.88) to the change in $\dot{Q}$ and the change in (a-v)$O_2$diff (r=-0.70) at the same time. Similarly the mean decrease in SV of 17.1 (19.2) mL·beat$^{-1}$ at 60 min was also significantly correlated to the change in $\dot{Q}$ and the change in (a-v)$O_2$diff (r=0.95 and r=-0.71, respectively). At 110 min the mean decrease in SV of 17.3 (18.6) mL·beat$^{-1}$ was significantly correlated to the mean change in HR, $\dot{Q}$, and (a-v)$O_2$diff with correlation coefficients of r=0.76, 0.97 and -0.90, respectively.

Percent changes in plasma concentration (PC) are presented in Figure 5. During CC PC was maintained at 55.2 (4.3) mL. No significant changes were observed when resting values were compared to the 15 min time increments

Figure 5: Mean percent change in plasma concentration for the control cycle, run, and cycle/run sessions, n=10 for CC, n=9 for RC, and n=8 for C/R, * significantly different from value at 3 min of the session ($p<0.05$). S.E. indicated by ⊥.


CC
RC
C/R
Transition
Plasma concentration (% change)
14
12
10
8
6
4
2
0
-2
-4
-6
-8
0
0
10
20
30
40
50
60
70
80
90
100
110
120
Time (min)

throughout the session. For the CC the mean percent change was 1.4 (8.5). The PC responded similarly during the RC ($\bar{x}$ = 53.5 (3.2) mL) with no significant differences occurring between the resting values and the 15-110 exercise values as well as between consecutive time intervals. The mean percent change during the RC was -0.9 (0.8). During the cycle portion of the C/R PC increased significantly between the resting value and 30 min and continued to remain elevated until the end of the cycle portion. Prior cycling exercise appeared to have no significant effect on PC responses during the run with no significant differences noted between the first 30 minutes of the RC and the run portion of the C/R. The mean values of 53.5 (2.6) and 56.1 (2.9) mL respectively. The mean percent change for PC during the cycle and run portion of the C/R was 5.0 (1.0) and 3.2 (1.1), respectively.

Figures 6 and 7 represent the mean oxygen uptake ($\dot{V}O_2$) and ventilation ($\dot{V}E$) responses for the RC, CC, and the C/R. The mean $\dot{V}O_2$ for the CC was 2.42 (0.35) $L \cdot min^{-1}$. Significant increases from values recorded at 3 min were noted for $\dot{V}O_2$ at 75 and 110 min. The mean $\dot{V}O_2$ for the cycle portion of the C/R of 2.59 (0.36) $L \cdot min^{-1}$ was not significantly different from the mean for the CC.

The mean $\dot{V}O_2$ observed for the RC was 2.80 (0.41) $L \cdot min^{-1}$. $\dot{V}O_2$ for the run portion of the C/R session was 2.83 (0.29) $L \cdot min^{-1}$.

Figure 6: Mean oxygen consumption responses for the control cycle, run, and cycle/run sessions, n=10 for CC, n=9 for RC, and n=8 for C/R, * significantly different from value at 3 min of session ($p<0.05$). S.E. indicated by $\perp$.

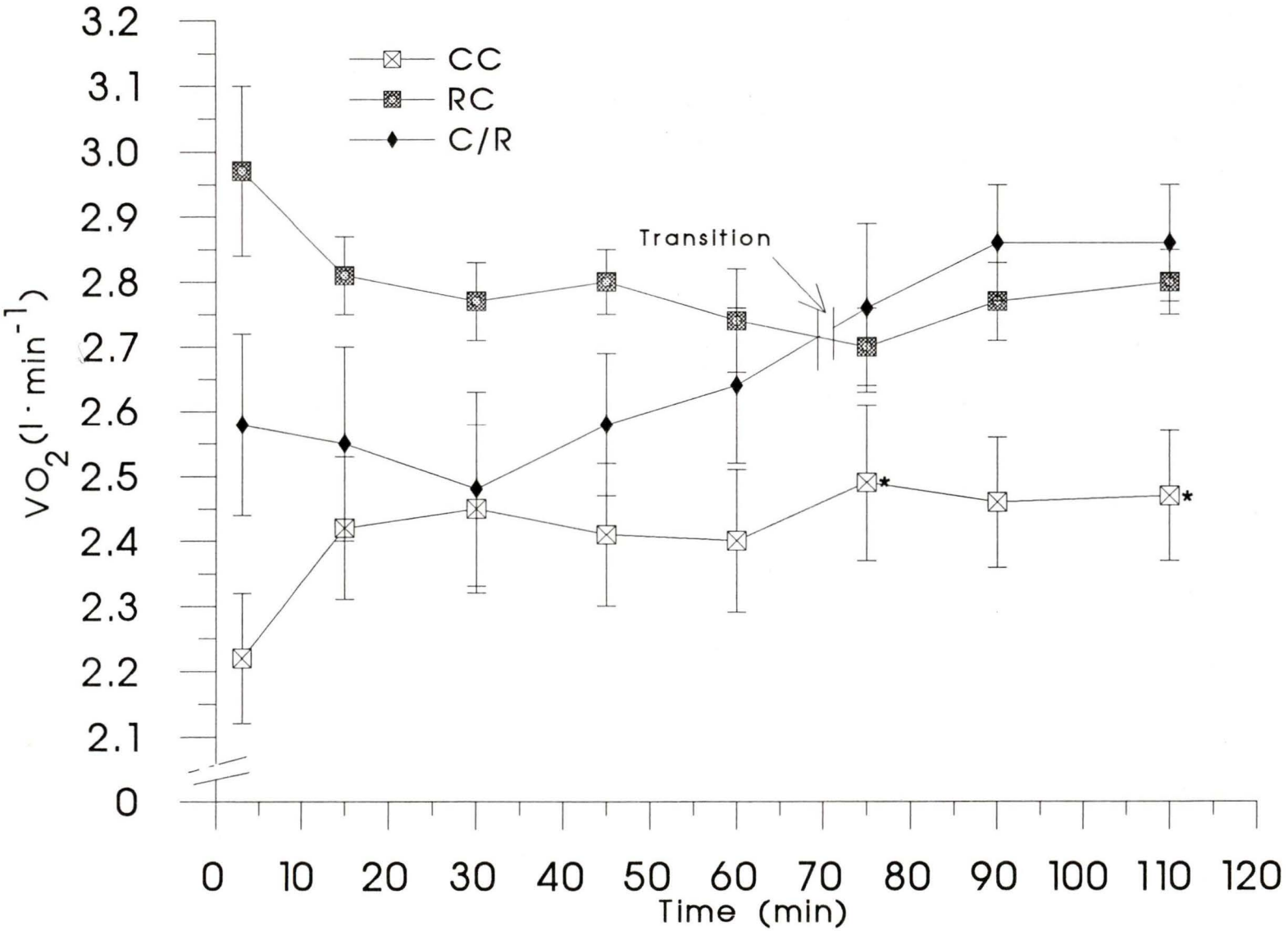
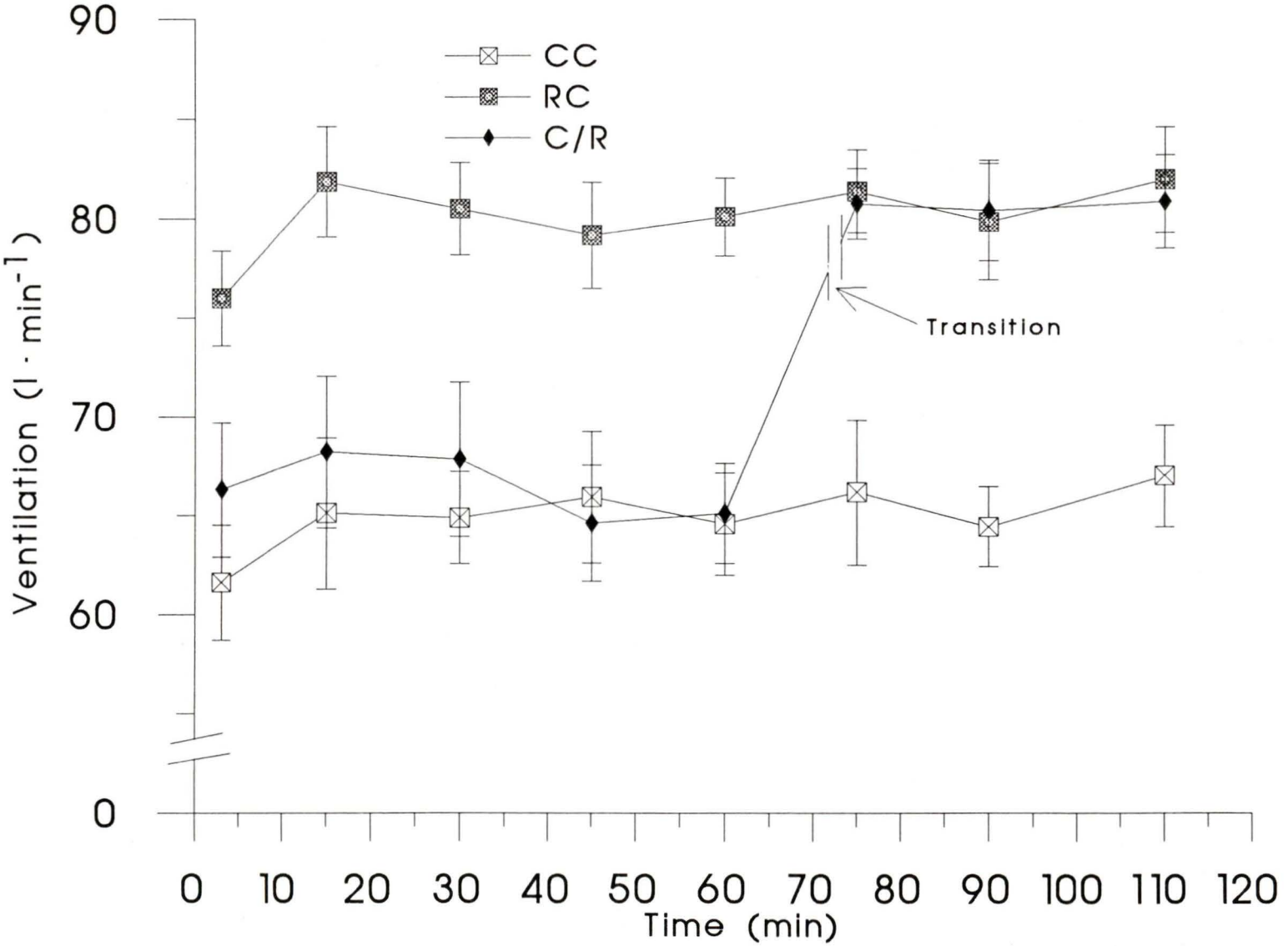

CC
RC
C/R
Transition
$VO_2 (l \cdot min^{-1})$
3.2
3.1
3.0
2.9
2.8
2.7
2.6
2.5
2.4
2.3
2.2
2.1
0
*
*
0
10
20
30
40
50
60
70
80
90
100
110
120
Time (min)

Figure 7: Mean ventilatory responses for the control cycle, run, and cycle/run sessions, n=10 for CC, n=9 for RC, and n=8 for C/R. S.E. indicated by ⊥.

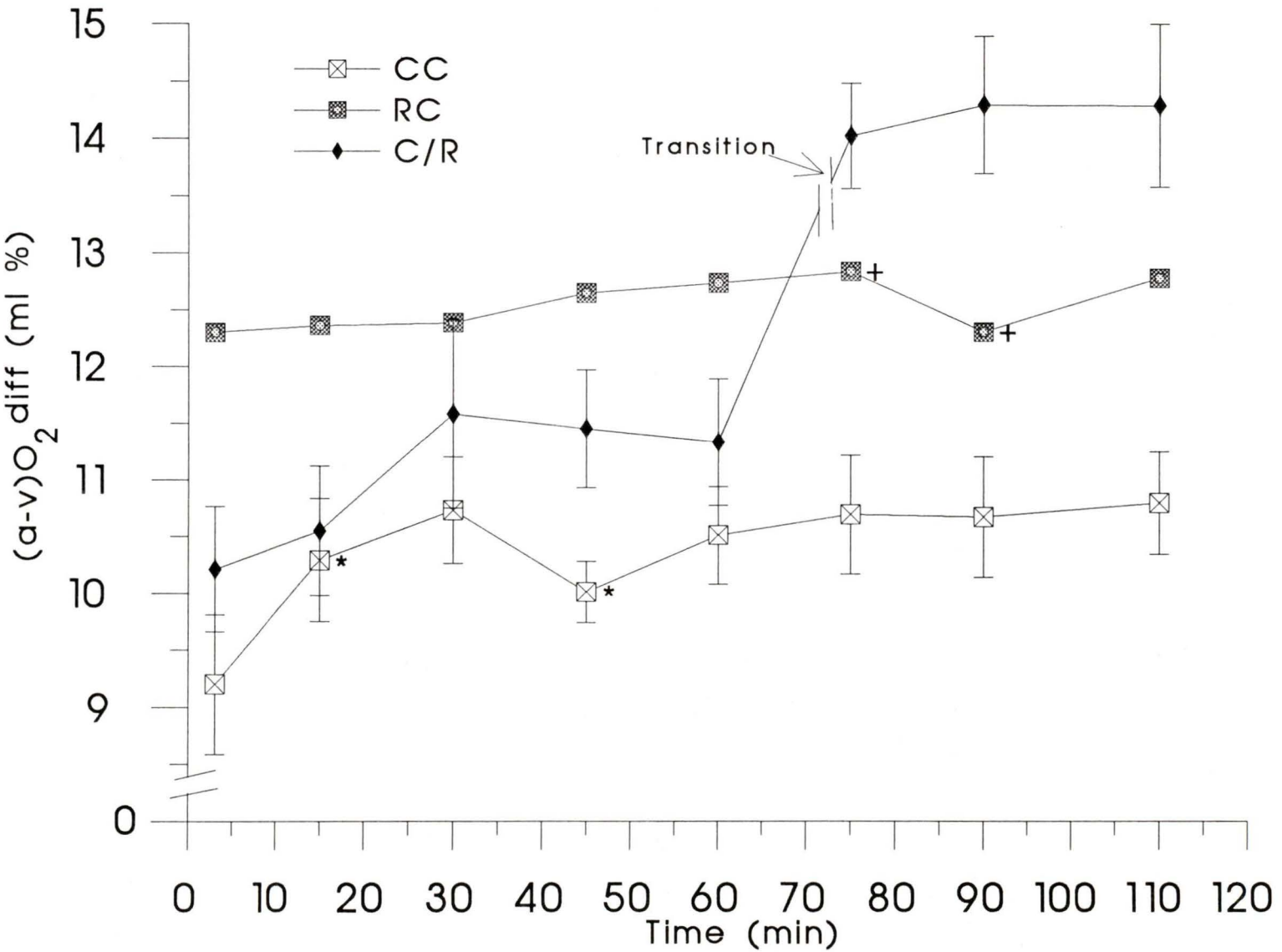

90
80
70
60
0
Ventilation (l · min-1)
CC
RC
C/R
Transition
0
10
20
30
40
50
60
70
80
90
100
110
120
Time (min)

Mean $\dot{V}E$ for the control cycling session was 65.0 (9.1) $L \cdot min^{-1}$. $\dot{V}E$ remained stable throught the CC with no significant differences noted within the session. The RC $\dot{V}E$ behaved similarly with a mean of 80.1 (7.3) $L \cdot min^{-1}$. As well no significant differences were noted during the session. Both the cycle and run session $\dot{V}E$ were not significantly different from $\dot{V}E$ response in the same mode of C/R.

The mean arteriovenous oxygen difference ($(a-v)O_2$diff) for the CC was 10.4 (1.4) $mL \cdot 100mL^{-1}$ of blood (see Figure 8). The $(a-v)O_2$diff showed a significant rise between 3 and 15 min of CC with a mean difference of 1.1 (1.2) $mL \cdot 100mL^{-1}$. There was a significant drop in $(a-v)O_2$diff at 45 min of CC. This change significantly correlated to the changes occuring in SV and $\dot{Q}$ at the same time (r=-0.95 and -0.89 respectively). The $(a-v)O_2$diff remained stable throughout the RC with a mean of 12.6 (1.4) $mL \cdot 100mL^{-1}$. During the C/R $(a-v)O_2$diff averaged 11.0 (1.7) $mL \cdot 100mL^{-1}$ during the cycle portion and 14.2 (1.6)$mL \cdot 100mL^{-1}$ during the run portion. The mean $(a-v)O_2$diff at 75 and 90 min of the run portion of the C/R ($\bar{x}$ = 14.0 (1.3) and 14.2 (1.7) $mL \cdot 100mL^{-1}$, respectively) were significantly higher than the $(a-v)O_2$diff at the same times of the RC ($\bar{x}$ = 12.8 (1.6) and 12.6 (1.6) $mL \cdot 100mL^{-1}$).

During the CC respiratory exchange ratio (RER)

Figure 8: Mean arteriovenous oxygen difference responses for the control cycle, run, and cycle/run sessions, n=10 for CC, n=9 for RC, and n=8 for C/R, * significantly different from prior time interval ($p<0.05$), + significantly different from C/R at same time interval ($p<0.05$). S.E. indicated by ⊥.


15
14
13
12
11
10
9
0
CC
RC
C/R
Transition
(a-v)O2 diff (ml %)
0
10
20
30
40
50
60
70
80
90
100
110
120
Time (min)

significantly decreased between 15 and 30, and 75 and 90 min (see Table 3). The mean RER for the CC was 1.01 (0.04). RER during the RC showed no significant differences when the value at 3 min was compared to all other time intervals. As well, no differences were found when consecutive time intervals were compared. The mean RER for the RC was 1.00 (0.04). During the C/R a significant decrease from 1.02 (.03) to 0.99 (.04) was found between 15 and 30 min of the cycle portion. There were no differences between the RER elicited during first 30 min of the RC and the run portion of the C/R ($\tilde{x}$ = 1.01 (0.05) and 0.95 (.05), repectively).

The subjects ingested an average 90.2 (15.8) mL of Gatorade every 10 minutes throughout the CC. This was not significantly different from the average of 86.1 (40.9) consumed every 10 min of the RC. Post-CC weight averaged 74.2 (6.9) kg with a mean decrease of 0.8 (0.6) kg. This weight was significantly greater than the post-RC weight of 73.6 (6.8) kg. The mean decrease observed for the RC was 1.3 (0.6) kg. The mean decrease observed for the RC was also greater than the decrease observed during the C/R session of 1.1 (0.4) kg.

Table 3: Mean (SE) RER responses for the CC, RC, and C/R sessions, n=10 for CC, n=9 for RC, and n=8 for C/R, * significant difference from prior time interval ($p<0.05$).

| | Time (min) | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Session | 3 | 15 | 30 | 45 | 60 | 75 | 90 | 110 |
| CC | 1.04±.06 | 1.04±.04 | 1.01±.03* | 1.01±.03 | 1.00±.03 | 1.00±.03 | 0.99±.03* | 0.99±.04 |
| RC | 1.00±.05 | 1.01±.04 | 1.02±.05 | 1.03±.03 | 1.00±.03 | 0.99±.04 | 1.00±.04 | 1.00±.02 |
| C/R | 1.02±.06 | 1.02±.03 | 0.99±.04* | 0.99±.04 | 0.97±.03 | 0.96±.05 | 0.96±.05 | 0.94±.04 |

# DISCUSSION

## Cardiovascular Responses to Prolonged Cycling and Running

It has been established that the phenomenon of cardiovascular drift (CD) is associated with prolonged exercise (Raven & Stevens, 1991; Rowell, 1974; Davies & Thompson, 1986). The physiological responses to prolonged exercise at an intensity greater than 50% maximal oxygen consumption is characterized by a progressive increase in heart rate (HR) corresponding to a decrease in stroke volume (SV) in order to maintain cardiac output ($\dot{Q}$), and an increase in oxygen consumption ($\dot{V}O_2$) (Nadel, Cafarelli, Roberts, & Wenger, 1979; Sawka, Knowlton, & Critz, 1979).

The major finding of this investigation is an apparent greater magnitude of cardiovascular drift elicited during the 110 minute control run session. This is indicated by the increase in HR over the entire course of the session of 12 beats·min$^{-1}$ with significant increases between 30 and 45 minutes and 75-110 minutes. The increase in HR during the control run corresponds with a decrease in SV of 17.3 ml·beat$^{-1}$. $\dot{Q}$ was maintained throughout the control run at 22.7 l·min$^{-1}$. The decrease in SV, can account for the increase in HR over the control run. This is consistent with the results of Saltin and Sternberg (1964), and Ekelund (1967) who also observed a constant $\dot{Q}$ over a period of exercise of one hour or longer. The maintenance of $\dot{Q}$ during the RC despite a greater blood flow to the skin as a result

of the greater thermal demand of running suggests that the extra volume of blood needed for temperature regulation is shunted from the inactive muscle and organs. The maintenance of $\dot{V}O_2$ is unusual.

The increase in HR along with the corresponding decrease in SV during the control run (RC) may have been precipitated by the thermoregulatory demand of the exercise session. Davies and Thompson (1986) observed similar responses during a four hour run between 65 and 70% $\dot{V}O_2$max in which heart rate increased 17%. The changes in SV may be due to adjustments in the distribution of the blood volume such as the shunting of blood to the cutaneous vessels for heat dissipation and a dilation of peripheral vessels (Rowell, 1974). During prolonged exercise when there are significant increases in body temperature both the exercising muscle and the skin require a high flow rate of blood (Roberts & Wenger, 1980). The ability of the body to supply the skin and the muscle with sufficient blood flow depends largely on its ability to maintain an adequate cardiac filling pressure (Rowell, 1974). During moderate exercise in a warm environment, the demands from the skin and the muscle are increased and the ability of the heart to meet this demand becomes compromised (Rowell, 1974; Brenglemann, 1983). The body's defense against a decreased central filling pressure is an increase in HR to compensate for the decrease in SV, in combination with a redistribution

of blood away from the inactive organs (Rowell, 1974).

In this investigation the change in HR was found to have a significant negative correlation (r=-0.77) with the change in rectal temperature ($T_r$). This suggests that as HR increases the change in $T_r$ decreases. A possible explanation for this is that as a result of the increasing HR more blood can reach the periphery, therefore promoting whole body cooling and partially alleviating the rapid increases in core temperature. The fact that $T_r$ continues to increase despite this correlation suggests that the blood flow to the skin was not great enough to prevent whole body heating and that HR increased to maintain $\dot{Q}$ as a result of a falling SV. This responses was similar to the findings of O'Toole, Hiller, Douglas, Pisarello and Mullen (1987). In their study subjects exercised on a cycle ergometer for five hours which was immediately followed by a 3-hour treadmill run. HR increased by 3% during the cycle in conjunction with a fall in SV of 7%. During the run HR increased 7% in conjunction with a 5% decrease in SV. Senay and Pivarnik (1991) reported some instances when body temperature was maintained but HR continued to increased. The redistribution of blood associated with thermal demands cannot always explain the increases in HR.

In contrast, during the 110 minute control cycling (CC) session HR increased 9 beat$\cdot$min$^{-1}$ during the session. Much of the overall increase occurred within the first 15

min of cycling. During the first 15 min of cycling SV fell by 13.9 $ml \cdot beat^{-1}$. The mean change in HR observed at this time was already 8 $beats \cdot min^{-1}$. Similar to the RC the decrease in SV during the first 15 min of the CC can account for the increase in HR that takes place at the same time. HR virtually plateaued from 15 min to 110. Like HR $T_r$ also appeared to stablize but not until 60 min into the exercise session. $T_r$ increased 1.1°C during the first 60 min and only 0.1°C more from 60 to 110 min. This suggests that blood flow to the exercising muscle and the periphery may have reached a level in which blood flow to the periphery was sufficient to offset further heat accumulation, as reflected by the stablization in $T_r$ in the later part of the cycle.

$\dot{Q}$ was maintained throughout the cycle as well as SV. This is unusual considering that there was still a significant thermal demand during the cycle. Whether or not the increase in HR during the first 15 minutes of the cycle was thermally mediated is questionable. Although there was a significant increase in $T_r$ of 0.5°C during the first 15 minutes $T_r$ continued to increase another 0.6°C from 15 through to 60 min. The increase in temperature from 15 to 60 min is not associated with any further increase in HR. The implication of this result may be that the increases in HR during the first 15 min are due to mechanisms other than those that are thermally mediated.

Decreases in plasma volume associated with dehydration have also been linked as contributors to CD (Hamilton, Gonzalez-Alonso, Montain, & Coyle, 1991). Reductions in body water due to sweating are considered to amplify hyperthermia by reducing venous return, thereby decreasing the body's ability to transfer heat from the core to the periphery (Nadel, Fourtney & Wenger, 1980). In conditions such as this, vasoconstriction in the skin can occur to offset the fall in central venous pressure. As a result, heat dissipation is reduced and hyperthermia may ensue. Hamilton et al. (1991) reported that fluid replacement which maintained euyhydration prevented stroke volume from declining over a 2-hour cycle. Montain and Coyle (1992) reported decreases in plasma volume of 2-3% during the last 80 minutes of a 2-hour cycle. They concluded that decreases in plasma volume and body weight were directly related to the increases in HR and declining stroke volume during the exercise session. In the present study, the greatest change in PC during the run took place between 30 and 60 minutes of exercise and represented a mean decrease of 3.4%. Although this was not a statistically significant reduction it falls within the guidelines set for effects of dehydration by Montain and Coyle (1992). After the RC the mean weight loss recorded was 1.3 kg. This represented a significant decrease in body weight of 1.7%. It has been shown that even small amounts of dehydration, 1% reduction in body

weight, similar to the dehydration in the present study can amplify increases in core temperature resulting in increases in HR (Elkblom, Greenleaf, Greenleaf, & Hermansen, 1970). In contrast, Noakes, Myburgh, du Plessis, Lang, Lambert, van der Riet and Schall (1991) reported that dehydration does not effect core temperature until a 3% reduction in weight has occurred. The present data do not support this. There was 1.7°C increase in rectal temperature while the reduction in body weight was equal to only 1.7% indicating that there was an alteration in heat storage even with a weight loss of less than 3%.

The reduction in plasma concentration (PC) during the first 15 min of running may also be attributed to the initial osmotic and hydrostatic changes that take place within the exercising muscle (Senay & Pivarnik, 1991). Within the first few steps of running hydrostatic pressure falls in the muscle. This gradient from the muscle to the capillaries is opposed by an increased osmotic gradient from the capillary to the muscle. Fluid will then remain in the muscle due to the increased osmotic gradient. Slowly, over the course of prolonged running, this fluid may be returned. As the subjects continue to exercise the muscle compartment may equilibrate or the exercise may cause a similar loss of fluid so that a recovery in plasma volume will not be realized (Galbo, Holst, & Christensen, 1975).

During the RC fluid lost as sweat was unlikely the sole

contributor to the decrease in PC. Nielson, Sjogaard and Bonde-Petersen (1984) observed similar changes in plasma volume in a consecutive swim and cycle. They observed a fall in plasma volume of 5% within the first 10 minutes of the exercise session. It is unlikely that the increase in HR was related to the reductions in plasma volume. The mechanism in which decreases in plasma volume are believed to cause increases in HR involve the low pressure arterial baroreceptors (Nadel, Fortney & Wenger, 1980). The parallel change in PC occurring in this study was probably not great enough to stimulate the low pressure baroreceptor reflex from the atria and the pulmonary circulation to cause the observed increase in HR.

During the first 15 min of the control cycle PC slightly increased (2.0%) at the onset of exercise which was maintained for the entire CC. This observation is unusual in consideration of the significant mean weight loss similar to the RC of 0.8 kg. In addition, the fluid intake for the CC was not significantly different from the control run either which makes even small increases in PC unusual. The small increase in PC might be explained by initial hydrostatic and osmotic events that are opposite to those experienced during running. Although, cycling is considered an upright activity it may be that the body position of the subjects in the present study was atypical. All subjects assumed an aerodynamic position during cycling which, in

some cases, could bring the torso to or below the level of the heart. The effect of this position on the osmotic and hydrostatic forces that would normally occur during cycling is speculative.

During the RC oxygen consumption ($\dot{V}O_2$) remained stable for the entire session. These findings are similar to those of Davies and Thompson (1986) who also found that $\dot{V}O_2$ did not significantly increase for the first 110 minutes of a prolonged cycle. Similar results were observed by Nielson et al. (1984) who found no significant increases in $\dot{V}O_2$ during a consecutive 20 min swim, 90 min swim and 90 min cycle. The responses in the present study were unexpected, especially considering thermal demand and changes in SV and HR that occurred. O'Toole et al. (1987) observed increases in $\dot{V}O_2$ over a 5-hour cycle that corresponded to changes in HR, SV, and $\dot{Q}$ for the female subjects whereas the males $\dot{V}O_2$, SV, and $\dot{Q}$ remained stable. Davies and Thompson (1986) also observed elevations in $\dot{V}O_2$ but they only became statistically significant after 110 minutes of cycling. The late increase in $\dot{V}O_2$ was attributed to increases in relative workload caused by a decrease in body weight. This, however, cannot explain the lack of drift in $\dot{V}O_2$ in the present study because the subjects experienced a significant mean weight loss of 1.3 kg. As well the significant thermal demand of the session did not seem to effect $\dot{V}O_2$.

Unlike the RC some significant increases did occur in

$\dot{V}O_2$ during the CC. Increases were noted between 60 and 75 and 90 and 110 min. Increases in $\dot{V}O_2$ during prolonged exercise have been observed by others (Saltin & Sternberg, 1964, Hagberg, Mullen, & Nagle, 1978). Several factors could account for this rise in $\dot{V}O_2$. Decreased mechanical efficiency, increased levels of circulating catecholamines, hyperventilation, increased energy requirements of the peripheral circulation, and the $Q_{10}$ effect have all been cited as possible causes of increased $\dot{V}O_2$ during prolonged exercise (Rowell, Brenglemann, Murray, Kraning, & Kusmi 1969; Saltin & Sternberg, 1964; Hagberg et al., 1978).

Increased upper body movement and use of the arms to aid in pedalling may have caused the elevation in $\dot{V}O_2$ during the control cycle. The subjects in this study were able to maintain the required power output but during the last 20 minutes of exercise a significant increase of 40 W was observed. This might have been due to anticipation of the completion of the session. Power output was changed by increasing revolutions per minute (RPM). By doing this the subjects effectively maintained a higher power output for the last 20 min of the session than was actually required. This, however, cannot explain increases in $\dot{V}O_2$ earlier in the session (60 minutes). In the later part of the session some subjects appeared to use more of their upper body to maintain the required power output. The observed power output of 171 W at this time was not significantly different

from any other time interval except the value noted at 110 minutes. A more plausible explanation may link the increases $\dot{V}O_2$ to an augmented neural drive related to the maintenance of muscle force output. There may have been an elevated neural drive because an increased number of muscle fibers was required to maintain the power output (Davies & Thompson, 1986).

The fact that $\dot{V}E$ did not change during the RC suggests that hyperventilation probably did not lead to the increased $\dot{V}O_2$ (Powers, Howley, & Cox, 1982). During submaximal exercise a gradual increase in plasma catecholamine content is observed. As exercise becomes prolonged the response may become more pronounced as a result of reductions in blood glucose concentration (Mazzeo, 1991). Kalis, Freund, Joyner, Jilka, Nittolo and Wilmore (1988) have demonstrated that a drift in $\dot{V}O_2$ can be prevented with non-selective ß-blockade. This suggests that increases in circulating catecholamines during prolonged exercise may act via ß receptors to cause the increase in $\dot{V}O_2$. Catecholamines are also believed to increase in conjunction with increases in core temperature. In the present study catecholamine levels were not measured therefore the impact of them on $\dot{V}O_2$ is speculative.

Respiratory exchange ratio (RER) actually decreased slightly from the beginning to the end of the cycle control session. The use of fat as a substrate is known to increase the oxygen cost of exercise (Millard-Staggord, Sparling,

Rosskopf, Hinson, & Dicarlo, 1990). In the present study there is a significant decrease in RER from 60 through to 110 minutes of the control cycle from a mean RER of 1.00 at 60 to 0.98 at 110 minutes. Although statistically significant these RER values indicate that carbohydrate, in the form of glycogen, is still the primary fuel source.

The increase in $\dot{V}O_2$ during the control cycle may be partially explained by the increase in core temperature. Grimby (1962) found that for each increase in core temperature of 1.3°C, $\dot{V}O_2$ increases 5.5%. The mean increase in $T_r$ at 60 minutes was 1.1°C. If the relationship holds true for this data there would be an increase in $\dot{V}O_2$ of 4.7% at 60 minutes. However the increase in $\dot{V}O_2$ between 60 and 75 minutes was only approximately 1%.

The difference in the magnitude of drift between the CC and the RC is probably attributable to the greater absolute and relative workload of the running exercise compared to cycling. Shaffrath and Adams (1984) made a similar observation in their investigation of CD. They concluded that workload, particularly relative workload, contributed to the thermal demand by increasing metabolic heat production. They also suggested that workload may contribute to CD by means other than metabolic heat production. Relative workload is instrumental in determining the magnitude of splanchnic vasoconstriction thereby establishing the size of the reservoir of vascular

volume available to meet any ensuing demand. Although both sessions were at an intensity of approximately 60% $\dot{V}O_2$max, of the specific mode of exercise, the absolute workload may have been greater during the RC. Running utilizes a greater muscle mass than cycling and, therefore the work being accomplished during running would be greater.

It appears that CD is highly related to the competition between the exercising muscles and the cutaneous circulation for the circulating blood volume (Johnson, Niederberger, Rowell, Eisman, & Brenglemann, 1963). The increases in $T_r$ seem to only partially explain the increases in HR and that additional increases in HR and $\dot{V}O_2$ may be linked to centrally mediated control of the cardiovascular adjustments to prolonged exercise (Perski, Tzankoff, & Engel, 1985; Johnson, 1987). Practically, it is of importance to know whether or not CD effects performance. Subjects in this study were able to maintain power output during the control cycle and complete the control run despite the changes in HR that occurred. In prior studies, in which exercise duration was longer and the magnitude of drift greater than in the present study, subjects were still able to complete the required exercise (Davies & Thompson, 1986; Hamilton et al., 1991; Neilsen et al., 1984; MacDougall, Redden, Layton, & Dempsey, 1974). There appears to be no direct evidence that CD impairs performance.

**The Effects of Prior Cycling on the Cardiovascular responses to 40 Minutes of Running**

Few investigations have studied the cardiovascular responses to consecutive exercise consisting of more than one mode (O'Toole et al. 1987; Davies & Thompson, 1986; Kreider et al., 1988). The major focus of these investigations was to study the overall cardiovascular effect rather than the effects of the first exercise on the second.

In the present study the cardiovascular responses to a consecutive 70 minute cycle and 40 minute run were compared to the control sessions. The major finding of this investigation was that there appeared to be an accumulated thermal demand applied to running when immediately preceded by cycling. The 70 minute cycle elicited a 1.0°C increase in $T_r$ at the onset of the 40 minute run. Subjects began the run portion of the C/R session at 38.4°C versus 37.6°C at the onset of the control run. This result would suggest that the cardiovascular responses of the run portion of the C/R would be expected to correspond to cardiovascular responses observed in the latter portion of the RC. This was not the case. HR, SV, $\dot{Q}$, $\dot{V}E$, $(a\text{-}v)O_2$diff, $\dot{V}O_2$, and PC observed during the run portion of the C/R were not significantly different from the values observed during the first 40 minutes of the control run. In contrast when cardiovascular responses were compared between the last 40 minutes of the RC and the run portion of the C/R session

some differences were noted. HR's observed for final 40 minutes of the control run were significantly higher than those observed for the run portion of the C/R ($\bar{x}$ = 173 and 165 beats·min$^{-1}$ respectively). The total thermal demand of the C/R was less than that for running alone, although this was not significant ($p<0.06$). Mean $T_r$ at the cessation of the cycle/run was 39.0°C whereas mean $T_r$ upon completion of the control run was 39.2°C. The difference is minimal and it is unlikely that a 0.2°C difference would cause the observed difference in HR. What does appear to differentiate the sessions is the amount of time spent at such an elevated temperature. During the last 90 min of the RC $T_r$ remained at or above 38.4°C whereas for the C/R $T_r$ achieved this level at 75 min (at the onset of run portion). It may be that this additional thermal demand for a greater portion of the RC was directly related to the higher HR's observed for the session. In contrast $(a-v)O_2$diff for the control run was significantly lower than during the run portion of the C/R. The mean $(a-v)O_2$diff for the last 40 minutes of the RC was 12.7 ml·100ml$^{-1}$. The run portion of the C/R session elicited a mean $(a-v)O_2$diff of 14.2 ml·100ml$^{-1}$. As well, the run portion of the C/R session elicited a slightly higher $\dot{V}O_2$ than that elicited during the RC ($\bar{x}$ = 2.83 l·min$^{-1}$ and 2.76 l·min$^{-1}$, respectively). Although this increase was not significant it may still be linked to the higher $(a-v)O_2$diff during the run portion of

the C/R. A higher oxygen extraction during the C/R may be the result of two circulatory changes. First, oxygen extraction by the working muscles may be increased. Second, a redistribution of blood away from the non-exercising muscle and the splanchnic organs may occur. The splanchnic, hepatic and renal circulations extract only small amounts of oxygen from the blood. Vasoconstriction of these areas would allow a greater portion of $\dot{Q}$ to perfuse the working muscle resulting in the potential for a greater oxygen extraction (Rowell, 1974). A higher $(a\text{-}v)O_2$diff as a result of redistribution is unlikely in this case due to the significant thermal demand of the exercise. Blood flow to the skin will lose little oxygen so an increased $(a\text{-}v)O_2$diff for this reason is unlikely (Williams, Bredell, Wyndham, Strydom, Morrison, Peter, Flemming, & Ward, 1962). $\dot{Q}$ is another factor that may have influenced $(a\text{-}v)O_2$diff during the run portion of the C/R. $\dot{Q}$ appears to drop at the onset of running in the C/R session. Although this decrease was not statistically significant, $\dot{Q}$ dropped from a mean value of 23.5 $l \cdot min^{-1}$ at the end of the cycle to a mean of 19.8 $l \cdot min^{-1}$ at the beginning of the run portion and maintained this level for the duration of the run. In response, the $(a\text{-}v)O_2$diff may have had to increase to sustain the work. Kreider et al. (1988) reported similar results in a simulated triathlon session. Oxygen extraction was greater when running was preceded by cycling by 1.9 $ml \cdot 100ml^{-1}$ and

was attributed to the increases in $VO_2$ that coincided with the onset of running.

As with single mode exercise the CD response elicited during a sequential 70 minute cycle and 40 minute run did not appear to effect performance in either mode. In addition, even the cumulative thermal demand imposed during the run portion of the cycle/run did not impair performance during that portion.

# CONCLUSIONS

**What is the magnitude of cardiocascualr drift elicited over a separate 110 minute cycle and 110 minute run?**

Cardiovascular drift as reflected by HR had increased by 8 beats·min$^{-1}$ at 60 min of the RC and 12 beats·min$^{-1}$ by the cessation of the 110 min session. This represents a total increase of 7 %. CC did not elicit as great a magnitude of cardiovascular drift although a significant rise in HR was noted. Like the RC at 60 min of the CC HR had also increased 8 beats·min$^{-1}$, but from 60 min through to 110 min HR only increased another 1 beat·min$^{-1}$ for a total drift of 9 beats·min$^{-1}$. This represented a 6 % increase in HR for the session. The greater drift in the second half of the RC can be explained by the continuing increases in rectal $T_r$ that were observed for the session, whereas during control cycling the stablization of $T_r$ appeared to correspond to the stabilization in heart rate. Although the RC elicited a greater absolute increase in HR the increase in terms of percent change in HR from the initial steady state value were very similar between sessions.

An important factor in explaining the difference in the CD observed between the RC and the CC may be related to the differences in absolute and relative workload maintained throughout each of the sessions rather than the mode of exercise. The workload for cycling was probably less than

that for running which resulted in a smaller metabolic heat production during the cycling. A smaller metabolic heat production would lead to a decreased thermal demand and less magnitude in CD as reflected by heart rate.

**What are the effects of prior cycling exercise of 70 minutes on the cardiovascular responses to 40 minutes of running when compared to running alone?**

Prior cycling exercise appeared to have very little effect on the cardiovascular responses to a 40 minute run. HR, SV, $\dot{Q}$, $\dot{V}O_2$, $(a\text{-}v)O_2$diff, $\dot{V}E$ and PC appeared to be unaffected by 70 min of prior cycling. There was no significant difference in these variables when reponses for the first 30 min of the control run were compared to reponses observed for the run portion of the C/R. In contrast, there appeared to be a cumulative effect on the temperature response to cycling and running. The $T_r$ observed at the start of the run portion of the C/R was significantly higher than the $T_r$ observed at the beginning of the RC because it was preceded by 70 min of cycling. However, by the end of the C/R no significant difference was noted for $T_r$ between the sessions.

**Was the magnitiude of cardiovascular drift greater or smaller for exercise consisting of one mode than for exercise consisting of two modes?**

For the present study the magnitude of the CD during RC appeared greater than that experienced during the C/R. HR's were significantly higher during the last 40 min of RC than during the run portion of the C/R. This response may reflect the greater metabolic load produced by running (due to the larger muscle mass required). In addition, the RC and the run portion of the C/R may have required the subject to exercise at a higher absolute and possibly relative workload, than during the CC and cycle portion of the C/R.

In contrast, C/R elicited significantly higher (a-v)$O_2$diff during the run portion compared to the final 40 min of the RC. This may have been related to the slightly higher $\dot{V}O_2$ and slightly lower $\hat{Q}$ observed during the run portion of the C/R.

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# APPENDIX A

Review of the Literature

With the increasing popularity of endurance and ultra-endurance sport there is a need to understand the adjustments and changes that the body undergoes during the competition and training for these types of events. One such physiological response that occurs during prolonged exercise is cardiovascular drift (CD). Cardiovascular drift is characterized by a constant decrease in stroke volume, increased heart rate and decreased mean arterial pressure (Adams, Fox, Fry & MacDonald, 1975; Brenglemann, 1983; Harrison, Edwards & Leitch, 1975).

In the past it was the marathon that was the "ultimate" challenge of endurance. Over the last 15 years there has been an increased popularity of ultra-distance multi-sport events such as the triathlon and the duathlon. The compounding complexity in understanding the effects of this type of activity on the body is that they are longer in duration and the change in events adds to the problem of understanding the physiological responses and adaptations that occur.

It is the purpose of this review to discuss briefly the cardiovascular adjustments to prolonged exercise, more specifically the phenomenon of CD and the influences of body temperature, cutaneous blood flow, blood volume, visceral blood flow, autonomic nervous system and myocardial contractility. The impact of CD will be assessed for those

athletes that use heart rate monitors for training and racing and the special considerations for triathletes and duathletes such as hydration and posture.

## Cardiovascular Adjustments to Prolonged Exercise

The cardiovascular response to prolonged exercise is first characterized by an initial change in steady state (metabolic and respiratory variables stabilize) to match the metabolic demands of the exercise. This initial adjustment is followed by a phase of CD. The level of CD is related to the individual's $\dot{V}O_2max$, the relative workload, the environmental conditions, and the level of hydration (Raven & Stevens, 1991). If the intensity of exercise is intense enough and the duration long it is possible that myocardial fatigue may occur.

During prolonged exercise, the blood supply to active muscle is increased, while at the same time the body tries to maintain thermal balance. It appears that blood translocation to the skin is the primary thermoregulatory response during prolonged exercise. After the first few minutes of exercise, blood flow to the visceral organs decreases and shows only minor changes as exercise progresses.

## Cardiovascular Drift

The progressive circulatory changes that take place

with prolonged exercise have been termed "cardiovascular drift" (CD). Raven and Stevens (1990) define CD as the cardiovascular adjustments that occur during constant load exercise for 60 minutes or longer at an intensity greater than 50% $\dot{V}O_2$max.

CD has been characterized by a constant cardiac output produced by an increase in heart rate with a decrease in stroke volume. Central venous pressure, pulmonary pressure and systemic arterial pressure all fall gradually and oxygen consumption gradually rises throughout prolonged exercise.

Unlike high-intensity short-term exercise during prolonged exercise the athlete is faced with two problems. Thermal stress causes cutaneous vasodilation, which displaces a large portion of the total blood volume into the cutaneous veins which lowers central blood volume, cardiac filling pressure, and stroke volume. High cutaneous blood volume tends to reduce the blood supply to the working muscle, which threatens to hinder performance.

There are several explanations for CD, 1) body water loss that may occur throughout prolonged exercise due to sweating, 2) peripheral vasodilation and a shift in blood volume from the central circulation to the periphery, 3) stroke volume and venous pressures may fall secondary to an increased heart rate, 4) a reduction in myocardial contractility (Johnson, 1987; Neilson, Sjogaard, & Bonde-Petersen, 1984; Hamilton, Gonzalaz-Alonso, Montain & Coyle,

1991; Shaffrath & Adams, 1984).

The degree of change observed in these responses appears related to environmental temperature and relative workload. Severity of the exercise affects the ability of the circulation to adjust to the heat. It has been shown that CD is increased by heat stress and reduced by body cooling (Johnson, 1987; Freund, Joyner, Jilka, Kalis, Nittolo, Taylor et al., 1987 and Roberts & Wenger, 1980). During prolonged exercise the athlete is faced with two physiological problems. First, thermal stress invokes cutaneous vasodilation which displaces the circulating blood volume into the cutaneous veins. This lowers central blood volume, cardiac filling and stroke volume. Second, the working muscles must receive adequate perfusion for the delivery of oxygen in order to perform the exercise (Raven & Stevens, 1990). It appears that whole body cooling plays an important role in determining the magnitude of CD. However, it is unclear whether CD is centrally of peripherally mediated.

Influence of Cutaneous Blood Flow

The principal event associated with CD is the translocation of the circulating blood volume into the cutaneous circulation. Skin blood flow (SBF) transfers heat from the core to the surface of the body. Lethal body temperatures would be reached if heat was prevented from

leaving the core. SBF reduces the temperature gradient at the skin for the transfer of heat to take place. If skin temperature cannot be kept low more SBF is required for the same heat transfer. The increase in SBF can be linked to several central mediated mechanisms of CD. Rowell (1974) suggested that the progressively decreasing stroke volume was due to increased core temperature that resulted in vasoconstriction of active skeletal muscle and peripheral displacement of blood volume to the cutaneous blood vessels. There may be reduced ventricular filling due to the redistribution of blood volume to the periphery as indicated by a decrease in blood pressure (BP) with prolonged exercise. Under normal conditions BP is shown to increase with exercise due to the resulting increase in cardiac output or more specifically heart rate and stroke volume. It has been proposed that the thermal stress experienced during prolonged exercise causes dilation of the arterioles and veins in the skin (Johnson et al., 1973). As a result a larger portion of the total blood volume goes to the periphery. Which results in a decrease in the cardiac filling pressure. This results in a smaller stroke volume which causes an increase in heart rate (Rowell, 1974). Johnson, Neiderberger, Rowell, Eisman and Brenglemann (1973) concluded that during prolonged exercise in which significant body heating occurs, the capillaries in the skin retain the ability to vasoconstrict in response to the

activation of the baroreceptor reflexes. This enables cardiac output to be maintained. Consequently, there is competition between the vasodilator response to prevent body heating and the baroreflex to maintain arterial pressure. The result of this competition is a compromised dilator response, which cannot effectively keep the body cooled, and a compromised mean arterial pressure which cannot maintain optimal stroke volume.

Rowell (1974) suggested that blood volume may not only be effected by increased skin blood flow but it may partially be determined by venous compliance. An increase in venous compliance in the skin would result in an even greater SBF and conversely the venous volume will also increase at a given rate of SBF. Relaxation of the upstream cutaneous arterioles provides an increased distending pressure for the compliance vessels of the venous system (Johnson, 1987). The peripheral increase in blood volume depletes other volume reservoirs that would otherwise provide filling to the heart, thus reducing stroke volume. Shaffrath and Adams (1984) support the hypothesis that CD originates with the displacement of the central blood volume into the cutaneous compliance vessels.

## INFLUENCE OF SPLANCHNIC, HEPATIC AND RENAL BLOOD FLOW

If cardiac output is not increased during heat stress the only means of increasing skin blood flow is to decrease

to the volume of blood perfusing the inactive tissues such as the liver, kidneys and splanchnic organs.

The increased sympathetic activation during prolonged exercise is in part responsible for the reduced blood flow to these areas (Hartley, Mason, Hogan, Jones, Kotchen, Mougay, et al., 1972). Even with this reduced blood flow to these inactive tissues, stroke volume continues to decrease during CD.

Shaffrath and Adams (1984) determined that relative workload plays an integral role in the magnitude of splanchnic, hepatic and renal passive redistribution of blood volume that accompanies prolonged exercise. This in turn will effect the magnitude of the passive redistribution of blood volume and thus establish the size of the reserve of vascular volume available to meet the subsequent demand. This concept of workload influencing vascular volume may explain the appearance of CD in cool environments and the large heat stress necessary to induce CD when the workload is low (Shaffrath and Adams, 1984).

If blood volume is reduced, as it often is during prolonged exercise, splanchnic blood flow can be further reduced at lower relative intensities. The reduction in flow can be seen at lower exercise intensities during exercise in the heat when reductions in blood volume occur (Rowell, 1974).

BLOOD VOLUME CHANGES

Progressive changes in blood volume occur during the first 10 minutes of prolonged exercise that are attributed to the initial osmotic and hydrostatic changes within the exercising muscle. As exercise becomes prolonged venous return can become compromised by, 1) the filtration of fluid out of the vascular bed, 2) increased cutaneous blood flow and volume due to the increased requirement for heat transfer and, 3) decreased blood volume as a result of evaporation of sweat for body cooling (Nadel, Fortney, & Wenger, 1980)

The primary physiological adjustment to a reduction in blood volume is the upward shift in the threshold for cutaneous vasodilation. Therefore, the body must reach a higher internal temperature before it can be attenuated by an increase in cutaneous blood flow. Once cutaneous vasodilation occurs the changes in blood flow are proportional to increases in core temperature regardless of whether or not there was a reduction in blood volume (Nadel et al., 1980).

Relatively small changes to the sensitivity of the thermoregulatory response occur with reductions in blood volume and more pronounced changes are seen as to threshold of response. This means that if blood volume were to decrease during prolonged exercise the overall thermoregulatory response would not be altered rather, the

temperature in which it would be activated would be increased. Some centrally mediated thermoregulatory mechanisms are suggested to be responsible for this response. Senay (1979) suggested that antidiuretic hormone (ADH) may have a role in the thermoregulatory response due to its sensitivity to plasma osmolality. Sawka et al. (1985) suggested that increased osmolality may mediate the reduced sweat rates that occur with hyperthermia. It has been suggested that this may be due to a direct effect of the osmolality on the thermoregulatory centre in the hypothalamus. Current data suggest that fluid replacement during prolonged exercise may serve to prevent changes in osmolality rather than blood volume (Montain, & Coyle, 1992) A second compensation in the hypohydrated response is that maximal cutaneous blood flow is much lower with hypohydration compared to euhydration.

Nadel et al. (1980) found that hyperhydration had no effect on blood volume, internal temperature, or cutaneous blood flow but did cause a reduction in heart rate seen with CD.

The importance of body water loss and its effects on the magnitude of CD was recognized by Hamilton et al. (1991). Water restriction during prolonged exercise in warm environments resulted in a continuous increase in core temperature. The increase was attributed to failure of adequate peripheral circulatory and/or sweating responses to

the increased thermal load. They found that fluid replacement prevented stroke volume and cardiac output from declining during prolonged exercise. Although a decline in stroke volume was still observed in this study it was associated with a decline in venous return to the heart due to excessive water losses from sweating. Rowell and Brenglemann (1971) suggested that fluid replacement may not serve to replace body water lost during prolonged exercise. It did prevent the decline in stroke volume because it alleviated the increases in body temperature. Therefore would decrease sweating rate. Although Hamilton et al. (1991) found that fluid replacement prevented stroke volume from decreasing and heart rate from increasing, it did not prevent the rise in oxygen consumption.

Several aspects concerning the influence of hypohydration on the cardiovascular response to prolonged exercise must be considered. The lower stroke volume may be related to the deficit in vascular volume, and cutaneous circulation (heat transfer) may be reduced when stroke volume is reduced.

The excessive hyperthermia that occurs in dehydrated individuals during exercise in the heat is due, in part, to, 1) modifications in the control of skin blood flow, 2) a higher threshold for cutaneous vasodilation reduces the core-to-skin heat transfer during the initial heat storage phase of exercise, and, 3) a reduction in maximal skin blood

flow.

Neilson et al.(1984) observed an increase in heart rate even with prolonged swimming in which core temperature remained stable. It is unlikely that the observed drift in heart rate can be due to a reduction in plasma volume due to dehydration from sweating or to increases core temperature since these factors remain fairly stable during swimming (Neilson et al., 1984). Water immersion tends to induce a fall in haematocrit resulting from a shift in extravascular fluid into the vascular beds. The reduction in plasma volume with swimming is though to be the result of the movement of water into the working muscles (Khosla and Dubois, 1981). Although sweat loss does not appear to play an important role in swimming, urine production is much larger during water immersion than out of water activity. The greater urine production serves to equilibrate to a greater extent the absolute body water losses of the two types of activity.

INFLUENCE OF CENTRAL COMMAND

It has been proposed that there is a centrally mediated descending neural stimulus that automatically adjusts skeletal muscle force generation to accomplish fatiguing work. In this model the increasing $\dot{V}O_2$ as seen with prolonged exercise is the result of the increased neural drive to maintain muscle force with an increased sympathetic

stimulus that increases heart rate as seen during CD (Hartley et al., 1972).

Davies and Thompson (1986) observed a marked increase in $\dot{V}O_2$ during a four hour run at a constant treadmill speed. They suggested four possible explanations for this increase. The first was the decrease in body weight due to sweating. This accentuated an increase in $\dot{V}O_2$ expressed in relative terms ($ml \cdot kg \cdot min^{-1}$). The second explanation was the shift in energy substrate utilized by the subjects. There was a gradual shift from carbohydrates to fats to fuel the exercise which would increase the oxygen requirement for the exercise, as reflected by the respiratory exchange ratio (RER). Thirdly, $\dot{V}O_2$ may have risen due to the increased work of the respiratory muscles. The fourth explanation for the gradual rise in $\dot{V}O_2$ was a decrease in the mechanical efficiency that may have occured near exhaustion. Davies and Thompson (1986) suggested that this could explain some increases in $\dot{V}O_2$ near the end of prolonged exercise but could not explain earlier increases.

In addition, the intramuscular acidosis resulting from the dissociation of lactic acid seems to be directly related to the loss of force generated by the muscle fibers. As a result of the loss of force, some fast-twitch motor units are recruited in order to compensate. The recruitment of new muscle fibers would cause cardiac output and $\dot{V}O_2$ to increase continuously (Camus et al., 1988).

Camus, Atchou, Buckner, Giezendanner and Di Prampero (1988) suggested that the rise in $\dot{V}O_2$ with prolonged exercise may be due to the removal of lactate which appears in the blood during exercise. An alternative explanation, according to Hagberg, Mullen and Nagle (1978) is that the slow rise in $\dot{V}O_2$ was due to the increase in temperature and ventilation. Neilson et al. (1990) observed that the progressive rise in oxygen consumption was not due to increases in working muscle but by increases in oxygen consumption for other organs and tissues.

## MYOCARDIAL FATIGUE

With the increasing popularity of endurance sport there has been an increasing concern that its prolonged nature could cause numerous cardiovascular events that may induce failure of the myocardium.

Myocardial contractility has been thought to be a factor underlying CD. Cardiac output is the product of stroke volume and heart rate. In the adaptation to prolonged exercise cardiac output may increase by more than 20% which is attributed mostly to an increase in stroke volume. The implication is that myocardial contractility may contribute to changes in stroke volume during prolonged exercise (Johnson, 1987). Stroke volume is determined by the contractile force of the left ventricle and the afterload (systolic pressure) that the left ventricle must

overcome. The myocardium, unlike skeletal muscle, is not able to recruit different numbers of muscle fibers in order to vary the contractile force. Instead, the contractile force of the heart is determined by the end-diastolic volume and contractility (Tibbits & Hamman, 1991).

Cardiac muscle increases its strength of contraction when it is stretched. An increased diastolic volume stretches the ventricular muscle fibers and causes them to contract more forcefully (Starlings Law of the Heart). End-diastolic volume, therefore, becomes a crucial determinant of cardiac output.

As a result of prolonged exercise, there is evidence that systolic function of the heart may be adversely effected. Upton et al. (1980) found that during maximal exercise, which followed a session of prolonged exercise, subjects were unable to match the same stroke volume that they achieved when maximal exercise was performed alone. With respect to diastolic function, Douglas et al. (1987) found an increased velocity of atrial or late diastolic inflow. Although these changes are quickly reversed during recovery, similar patterns for inflow have occurred with the aging and ischemic heart.

It appears that there are definite alterations to the myocardium during the late phases of prolonged exercise. However, it is undetermined whether these changes contribute to the magnitude of CD.

## ATHLETES AND HEART RATE MONITORS

The heart rate monitor is one of the most popular training aids utilized by endurance athletes. As race distances in sports such as running, triathlon and duathlon, grow from endurance to ultra-endurance the volume of training increases as does the time required for training. The heart rate monitor allows the athlete to monitor intensity so that they train and race at the highest intensity without the onset of metabolic acidosis. This is the intensity associated with anaerobic threshold.

The use of heart rate monitors and the occurrence of CD during endurance training or racing could have major implications to the athlete. O'Toole et al. (1987) reported a 7% increased in heart rate over a 3 hour treadmill run at 50% $\dot{V}O_2$max. The increase expressed in percent only seems small but if the starting heart rate was 130 bpm then the final heart rate could have been approximately 10 beats higher. Similarly, Basset (1987) reported increased heart rates from 142 to 156 from minutes 30-120 of treadmill running at 60% $\dot{V}O_2$max (O'Toole et al., 1987).

## OTHER CONSIDERATIONS

### HYDRATION

Although reduction in blood volume appears to contribute to CD, (plasma volume is well maintained even when there is considerable loss of body water), dehydration

appears to increase the severity of the drift that occurs. Consequently, an athlete that suffers from even a small degree of dehydration during training or racing may increase the magnitude of the CD that occurs. In addition to an increased CD, dehydration will also reduce thermoregulatory and cellular function which can hinder muscular performance.

POSTURE

In general, circulating blood volume is greater during supine than upright exercise. The reduction in blood volume from supine to upright exercise is the result of a reduction in plasma volume. Consequently in sports such as the triathlon and duathlon where a number of body positions are required these fluid shifts may effect the magnitude of CD at a given body position.

Wells, Stern, Kohert and Campbell (1987) examined fluid shifts during successive running and cycling. Their results show that more severe weight loss occurred during the running phase regardless of whether or not it was performed as the first or second event. In the upright position, the hydrostatic pressure results in maximum volume loss and this is exacerbated by exercise in this position (Senay and Pivarnik, 1985). It seems that static forces determine plasma volume before exercise begins and the osmotic forces take over once exercise has begun (Lundvall et al., 1972). Total fluid shifts were less severe during

the second phase of exercise regardless of the mode. It was suggested that during the second phase the blood volume was maintained by shifting red cell water, (water inside red blood cell), into the plasma. When exercise of more than one mode is performed it should be noted that when posture changes, for example from cycling to running, plasma volume may decrease as much as 10-15% within the first 10-20 minutes of exercise. This is due to the hydrostatic forces. This decrease should not be confused with any reduction that takes place after 30 minutes or longer which could be the result of dehydration and thus be a contributor the CD (O'Toole et al., 1987).

## CONCLUSION

It appears that the CD that takes place with prolonged exercise is precipitated by the thermoregulatory response to whole body heating. The central and peripheral adjustments that take place appear to be closely linked to each other. Thermoregulatory reflexes cause a rise in cutaneous vasodilation which is then forced to compete with the working muscle for the available cardiac output. Cutaneous venodilation and vasodilation appear to lower central blood volume diverting most of the blood to the periphery.

The impact of CD on the athlete that utilizes the heart rate monitor can be significant. Further research is necessary in order to determine the magnitude of the heart

rate change for athletes that compete and train at various distances so that the usefulness of this training aid can be fully realized.

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# APPENDIX B

Informed Consent

CARDIOVASCULAR DRIFT STUDY - INFORMED CONSENT

The purpose of this study is to examine the cardiovascular responses to prolonged exercise performed in standardized conditions at an intensity equivalent to 60-70 percent of $\dot{V}O_2max$. As well, the effect of prior cycling exercise (70 minutes) on the cardiovascular responses to running (40 minutes) when the two are performed consecutively will be examined.

I, _____, do hereby acknowledge:

* I consent to perform two continuous $\dot{V}O_2max$ tests
  - treadmill
  - cycle ergometer

* I consent to perform two control exercise sessions comprising of:
  - 110 minutes of continuous cycling
  - 110 minutes of continuous running

* I consent to perform one exercise session consisting of 70 minutes of continuous cycling and 40 minutes of continuous running.

* I consent to perform the above tests during a one month period at specified times under controlled conditions.
  a) I will refrain from my regular training routine for at least 48 hours prior to each test
  b) I will consume a mixed diet in which approximately 60-70% of my total caloric intake will be complex carbohydrates during the entire course of the study
  c) I will consume no caffeine two hours prior to each test
  d) I will consume no alcohol six hours prior to each test

* I understand a heart rate monitor will be attached across my mid-sternum region to record my heart rate every minute during exercise; a mouthpiece will be worn and expired gases will be analyzed by a Horizon cardiorespiratory system for respiratory and metabolic factors

* I understand that prior to and during all control sessions and the cycle/run session blood samples will be obtained by finger prick

* I understand that lean body mass and sum of skin folds will be assessed by anthropometric measures

* Even though I will be undergoing exercise to the point of exhaustion, I understand that there is very little risk involved if I am a healthy, active individual and that emergency equipment and trained personnel are available to deal with unusual situations that may arise

* I understand that I may temporarily experience local muscle fatigue, discomfort, nausea, and lightheadedness when the cardiac output rebreathing maneuver is performed

* I understand that the tests will be administered by qualified personnel under the direct supervision of the investigator

* I understand that while it is unlikely that I may be injured or ill during a test, lab personnel are trained in emergency procedures and emergency equipment is on-site at all times

* I understand that I may ask questions or request further explanations or information about the procedures at any time before, during or after testing

* I understand that I am in control at all times, that I am able to withdraw from, reduce or modify my involvement in the study at any time and that the test may be terminated by the investigators upon observation of any symptoms of distress or abnormal responses

* I understand that all my results are strictly confidential, that they may be cited by ID label or used to calculate a group mean and that all data will be securely locked in a cabinet at all times

* I understand that whether I participate in the study or choose not to participate my instructors/employers will not have access to any information collected in this study

* I do hereby release, _____, and its employees

name of institution

from any liability with respect to any injury or damage that I may suffer during participation. I acknowledge that I have read, understood, and agree to the contests of this informed consent agreement in its entirety.

| | |
|---|---|
| _____ | _____ |
| Signature | date |
| _____ | _____ |
| Witness | date |

## APPENDIX C

Dill and Costill's Equation for Estimation of Plasma Volume from Haemoglobin and Hematocrit

BVa BVb (Hbb/Hba)

$$CVa = BVa(Hcta)$$
$$PVa = BVa - CVa$$

$$BV\% = 100\ (BVa - BVb)/BVb$$
$$CV\% = 100\ (CVa - CVb)/CVb$$
$$PV\% = 100\ (PVa - PVb)/PVb$$

where, BV = blood volume
CV = red cell volume
PV = plasma volume

and, b = before treatment
a = after treatment

From: Dill, D.B., & Costill, D.L. (1974). Calculation of percentage changes in volumes of blood, plasma and red cells in dehydration. Journal of Applied Physiology, 37, 247-248.

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**THE EFFECT OF PROLONGED CYCLING AND RUNNING ON SELECTED CARDIOVASCULAR RESPONSES**

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Date: May 19, 1994