Eur J Appl Physiol (1984) 53:63–70

European Journal of
Applied
Physiology
and Occupational Physiology

Cardiovascular, hormonal and body fluid changes during prolonged exercise

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Summary. During prolonged heavy exercise a gradual upward drift in heart rate (HR) is seen after the first 10 min of exercise. This "secondary rise" might be caused by a reduction in stroke volume due to reduced filling of the heart, which is dependent upon both hemodynamic pressure and blood volume. Swimming and bicycling differ with respect to hydrostatic pressure and to water loss, due to sweating. Five subjects were studied during 90 min of bicycle exercise, and swimming the leg kick of free style. The horizontal position during swimming resulted in a larger cardiac output and stroke volume. After the initial rise in heart rate the "secondary rise" followed parallel courses in the two situations. The rises were positively related to the measured increments in plasma catecholamine concentrations, which continued to increase as exercise progresssed. The secondary rise in HR could not be explained by changes in plasma volume or in water balance, nor by changes in plasma [K]. The plasma volume decreased 5–6% (225–250 ml) within the first 5 to 10 min of exercise both in bicycling and swimming, but thereafter remained virtually unchanged. The sweat loss during bicycling was four times greater than during swimming; but during swimming the hydrostatic conditions induced a diuresis, so that the total water loss was only 25% less than during bicycling.

Key words: Heart rate – Cardiac output – Water balance – Plasma volume – Plasma [K]-[Na] – Plasma catecholamines – Muscle water – Muscle [K]-[Na] – Swimming – Bicycling

Introduction

Hydrostatic and/or osmotic pressure gradients cause water shifts between the body fluid compartments. Swimming and upright bicycling differ with respect to hydrostatic conditions. During swimming the hydrostatic pressure of the surrounding water counteracts or abolishes that part of the vascular transmural pressure which is due to gravitational forces. During upright bicycle exercise, on the other hand, the transmural vascular pressure is increased below heart level and part of the blood volume is shifted to vessels in the legs and abdominal cavity. Further, the water loss due to thermoregulatory sweating is negligible when swimming in cool water because of the high heat capacity and heat conductivity of water, while the heat loss is regulated through sweating when bicycling in air. The loss of water from the vascular bed would, therefore, presumably be greater during prolonged bicycling than during prolonged swimming. A decrease in circulating blood volume would reduce the diastolic filling of the heart, which would be more reduced during bicycle exercise than during swimming. The gradual, secondary rise in heart rate which is seen during prolonged exercise, like bicycling and running, might therefore be abolished or reduced during swimming if this increment in heart rate were caused by a decreased central venous pressure. If the secondary rise, however, is independent of central venous filling pressure, then other mechanisms, such as afferent nervous activity from the exercising muscles, or efferent sympathetic nervous activity might be responsible.

The aim of the present study was to compare total body- and muscle water balance with changes in heart rate and cardiac output during prolonged swimming and bicycling. Further, the loss of potassium from the exercising muscles to the plasma was studied because extracellular potassium changes have been shown to evoke central cardiovascular responses. Changes in plasma catecholamines and antidiuretic hormone concentrations were also measured as possible mediators for the secondary rise in heart rate and changes in water balance, respectively.

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Subjects and procedure

Five men (age 20–30 years, weight 71–80 kg, height 173–196 cm) volunteered to participate after being fully informed about the procedures and the risks involved. They swam the leg kick of freestyle (arms resting on a floating polystyren board), in a swimming flume (Åstrand and Englesson 1974) at 29° C water temperature. The speed was set to elicit an initial heart rate (HR) of 120–130 b. p. m. The subjects were able to maintain this speed for 90 min. The bicycling exercise was performed in a climatic chamber sitting upright in a chair bicycle ergometer behind the crank with the legs in a horizontal position. The air temperature was adjusted (in preliminary experiments) so that during exercise an average skin temperature of 29° C was obtained, i.e., the same as during swimming (29° C water). The bicycling intensity was adjusted so that oxygen uptake was similar to that measured during swimming. This amounted to approximately 60% of their $\dot{V}_{O2max}$.

Each subject was studied on three separate experimental days (3–6 days interval) performing 20 min swimming, 90 min swimming, and 90 min bicycling, respectively. The subjects were instructed to drink 1 l water in the evening before an experimental day, to ensure a positive water balance. When the subjects arrived in the morning after a light breakfast, a urine sample was taken and its osmolality measured to assess water balance. They then rested in the supine position for at least 30 min while a long venous catheter was introduced into an antecubital vein to about the level of the subclavian vein. An injection of 131 I-RIHSA (< 8 μCi) was given i.v. in the opposite arm; 10 and 20 min later bloodsamples were drawn from the catheter for later analysis in order to measure plasma volume (PV). Then a resting muscle sample was obtained by the needle biopsy technique (Bergström 1962) from M. vastus lateralis. The subjects emptied their bladder, were weighed and within 5 min they started to exercise, swimming or bicycling.

During exercise, blood samples were drawn (a total of 120 ml during the 90 min experiments), heart rate (HR) was monitored throughout and cardiac output ($\dot{Q}$) and rate of oxygen uptake ($\dot{V}_{O_2}$) were measured at approximately 10, 20, 35, 55, and 80 min of exercise. A muscle biopsy was taken after 20 min of bicycling, causing an interruption in the exercise of less than 30 s, and again at the end of the 90 min bicycling. The samples were taken less than 10 s after the subject stopped pedalling and while still sitting in the chair. In the swimming experiments the subject had quickly to get out of the water and lie down on a bridge across the swimming flume to have the muscle sample taken. Less than 30 s elapsed from the time swimming was stopped until the biopsy was taken. However, this interruption before the subject could return to the water was considered too long. Therefore two swimming experiments were performed, one lasting 20 min and one 90 min, with biopsies taken before and at the end of the exercise. Immediately after the biopsy the subject quickly dried himself. He was weighed and then emptied his bladder.

Methods

Oxygen uptake rate ($\dot{V}_{O_2}$) was measured by the open circuit method. Douglas bags were filled with expired air, the volume of which was measured in a Tissot wet spirometer. The oxygen and carbon dioxide concentrations were analyzed on a Servomex (OA 180) paramagnetic O_2 analyzer and a Beckman LB2 infrared CO_2 analyzer, respectively. Heart rate (HR) was monitored by electrocardiography. The signals were transmitted by a radio telemetry system (Danica). HR was counted every 5 min over 1 min. In the bicycle experiments skin temperature was measured with a hand-held thermocouple at 15 sites and weighted according to area (Nielsen and Nielsen 1965).

Cardiac output ($\dot{Q}$) was measured immediately after each $\dot{V}_{O_2}$ measurement by the acetylen-argon-oxygen rebreathing method, using a mass spectrometer (Bonde-Petersen et al. 1980). During the swimming experiments a modified respiratory valve was used, which permitted respiration through a snorkel until the rebreathing bag was switched on.

The water loss during exercise, was determined by weighing the subject immediately before and after exercise on a balance which could be read to ± 5 g (Krogh and Trolle 1936). The calculated metabolic weight loss due to the O_2-CO_2 shift, and an estimated respiratory water loss of 0.03 g/l pulmonary ventilation was subtracted from total loss to give sweat loss. Urine production during the exercise period was measured to the nearest 5 ml, and initial, pre- and postexercise urine samples were analysed for osmolality by the freezing point depression method (Advanced L3 osmometer). A total water balance for the exercise period, and oxidative water gains were calculated (Consolazio et al. 1963). The ratio $\dot{V}_{CO_2}/\dot{V}_{O_2}$ was used to estimate the ratio of fat to carbohydrate combustion. Sweat loss, respiratory water loss, urine production, and blood sampling were accounted for.

Blood samples were analyzed for: Hematocrit (Hct) corrected for trapped plasma (factor 0.96) and venous-to-total body Hct-ratio (factor 0.93). Hemoglobin concentration (Hb) was determined by the Drabkin cyanmethemoglobin method using a Zeiss spectrophotometer. Plasma protein concentration was determined by the biuret reaction (Boehringer, ®). Plasma [Na] and [K] were analyzed on an atomic absorption spectrophotometer (Perkin Elmer 372).

Pre-exercise plasma volume (PV) was determined from the resting blood samples by counting the RIHSA I-131 γ activity (Selektronic-counter) and extrapolating to zero from the 10- and 20-min values corrected for background. Further changes in plasma volume occuring during exercise were calculated relative to the measured plasma volume from Hct and Hb according to Dill and Costill (1977).

Plasma concentrations of antidiuretic hormone (ADH) were determined in the resting subject before and immediately after exercise by a radioimmuno method. Epinephrine (E) and norepinephrine (NE) concentrations in plasma were measured by high pressure liquid chromatography (Bioanalytical Systems).

The biopsies were immediately frozen in isopentane cooled in liquid nitrogen, stored at −80° C, and later analyzed for total water

content by weighing before and after freeze drying, for potassium, sodium, and magnesium content by atomic absorption spectrophotometry after extraction in HNO_3 (Sjøgaard 1983), and for glycogen and lactate content by fluorometric methods (Karlsson 1971). These results were expressed per kilogram dry muscle weight (kg dw). Further the biopsies were processed for histochemical analysis and stained for determination of glycogen depletion and fiber types.

Statistical differences were tested with Student's *t*-test for paired samples or by non-parametric two-way analysis of variance (Sachs 1976). Significance was set at the 0.05 level of confidence.

Results

The mean values at 10 min exercise for oxygen consumption were 2.19 and 2.42 $l \times min^{-1}$ and for heart rate 121 and 132 beats $\times$ min^{-1} in bicycling and swimming, respectively. These differences were not statistically significant (Fig. 1, Table 1). Mean skin temperatures were within the same range, 27–30° C in the two exercise conditions. Oxygen consumption and skin temperature remained unchanged throughout the two types of experiments, while a significant secondary rise occurred in HR (Fig. 1). The mean increments in HR from 10th to 90th min of exercise (ΔHR), were the same during the two exercise conditions: + 13 (range 9–20) beats $\times$ min^{-1} during swimming and + 13 (range 7–21) beats $\times$ min^{-1} during bicycling.

Cardiac output was higher during swimming than during bicycling ($P < 0.05$) from the first measurement at the 10th min and remained stable. Therefore, all measurements during exercise were averaged. The difference was only partly explained by the slightly higher $\dot{V}_{O_2}$ during swimming, because the (a-v) O_2 difference was significantly lower during swimming (cf. Table 1). The calculated stroke volumes were significantly higher during swimming [e.g., after 20 min 147 (131–174) ml in swimming compared to 130 (109–156) ml in bicycling ($P < 0.05$)]. Stroke volume decreased with time as HR increased, similarly in both swimming and bicycle exercise.

Water fluxes. The average water loss due to sweating was 691 (range 265–975) ml in the bicycle experiments and only 180 (range −82–422) ml during swimming. The water loss in the urine on the other hand was greater in the swimming experiment. The average diuresis over the 90-min exercise was 1.9 (1.0–4.5) ml $\times$ min^{-1} (total mean 237 ml) during bicycling and 4.7 (2.0–6.5) ml $\times$ min^{-1} (total mean 487 ml) during swimming. The total water balances for the two situations were −1,025 ml in bicycling and −782 ml in the swimming experiment (Fig. 2). The urine osmolalities did not change during bicycling but had decreased by about 50% after swimming (Table 2).

Plasma volume (PV) decreased by about 5% ($P < 0.05$) within the first 5–10 min of exercise: 258 (94–390) ml during bicycling and 229 (29–449) ml during swimming. Plasma volume then remained virtually unchanged till the end of 90 min exercise (Fig. 3).

The muscle samples taken after 20 min of bicycling and swimming, respectively, showed signif-

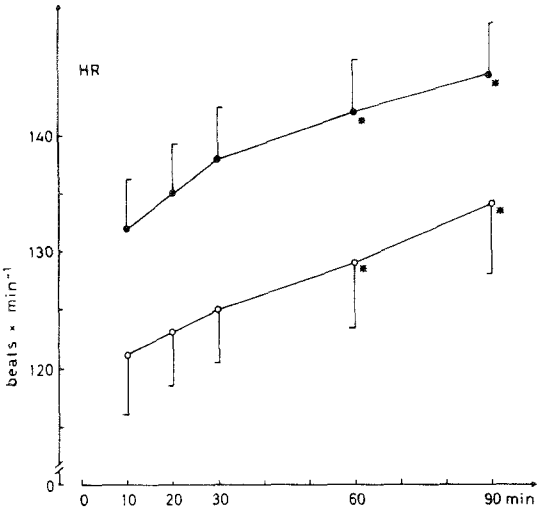

Fig. 1. Heart rate during 90 min of swimming ● and bicycling ○. Average values and SE for five subjects are shown. ✶ indicates the values which are significantly different from the 10-min value

Table 1. Averages of all measurements taken during swimming and bicycling between 10 and 80 min of mean skin temperature ($\bar{T}_{sk}$), oxygen consumption rate ($\dot{V}_{O_2}$) cardiac output ($\dot{Q}$) and arterio-venous oxygen difference [(a−v) O_2 diff.]. (5 subjects)

	Bicycling		Swimming		Sign of diff
	Mean	(Range)	Mean	(Range)	
$\bar{T}$sk °C	29.0	(27.8–29.8)	28.4	(27.6–29.9)	NS
$\dot{V}_{O_2}$ $l \times min^{-1}$	2.19	(1.73–2.47)	2.42	(1.83–2.99)	NS
$\dot{Q} \times min^{-1}$	16.9	(13.5–19.2)	19.8	(17.2–24.2)	$P < 0.025$
(a−v) O_2 diff. ml $\times$ l^{-1}	131	(120–153)	122	(113–136)	$P < 0.05$

icant increases in water content from the resting values, with an overall mean increase of 0.22 l × (kg dw)$^{-1}$ (Table 3). A further increase occurred during the following 70 min in four of the subjects during bicycling and in three subjects while swimming. The overall increase in muscle water during 90 min exercise averaged 0.49 l × (kg dw)$^{-1}$.

Electrolytes. Plasma [K] increased significantly from a resting value of 4.1 (3.5–4.6) mmol × l^{-1} within 10 min of bicycling or swimming (Fig. 4). After 30 min of swimming plasma [K] had increased to 4.6 (4.3–5.2) mmol × l^{-1}, while during bicycling a somewhat higher value of 5.0 (4.6–5.5) mmol × l^{-1} was attained. In both types of exercise plasma [K] remained at this level throughout the rest of the exercise. Plasma [Na] and [Mg] remained virtually unchanged during both types of exercise, although a tendency to increase was observed for both electrolytes at the end of bicycling (plasma [Na] = 134 (121–141) mmol × l^{-1} swimming and 135 (129–138) mmol × l^{-1} bicycling and plasma [Mg] = 0.73 (0.65–0.78) mmol × l^{-1} in both conditions).

Muscle [K] (Table 3) was only significantly decreased at the end of the bicycling experiments (approximately 10%). During bicycling muscle [Na] increased in all subjects within 20 min and in four of them a further increase occurred during the following

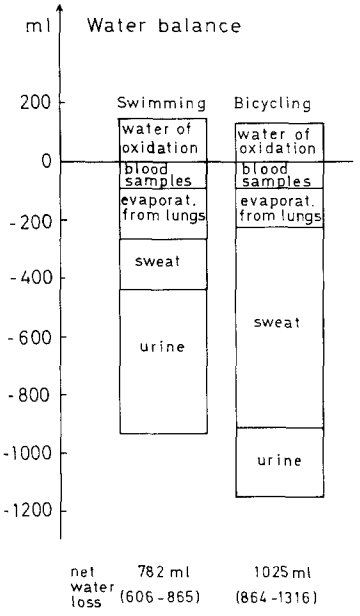

Fig. 2. Graphical presentation of the water balance after 90 min of swimming or bicycling. Average values of five subjects

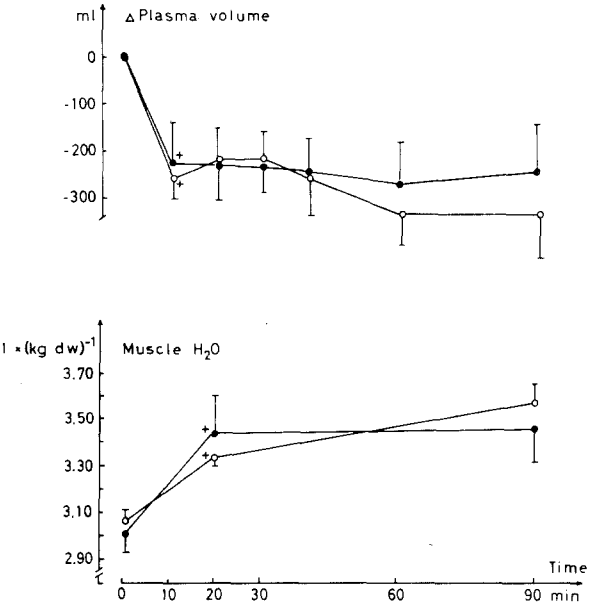

Fig. 3. Changes in plasma volume (above) and in muscle water contents (below) during 90 min exercise, swimming ● and bicycling ○. Average values and SE for five subjects. + denotes the earliest values which were significantly different from resting values

Table 2. Urine flows, urine osmolality, and plasma ADH during swimming and bicycling

	Urine			Plasma	
	Flow (ml × min^{-1})	Osmolality (mOsm × kg H_2O^{-1})		[ADH] (pg × 100 ml^{-1})	
	During	Before	After	Before	After
Swimming					
Mean	4.7	793	297	3.4	2.5
Range	(2.0–6.5)	(621–1148)	(156–496)	(0.9–6.7)	(1.2–3.8)
Bicycling					
Mean	1.9	862	625	2.4	4.2
Range	(1.0–4.3)	(639–1042)	(313–873)	(0.8–5.6)	(1.0–7.1)

70 min bicycling. The same tendency was seen during swimming. No systematic changes were observed in muscle [Mg].

Glycogen/lactate. There was a continuous breakdown of glycogen in vastus lateralis which was somewhat larger during bicycling [286 mmol × (kg dw)$^{-1}$] than during swimming [182 mmol × (kg dw)$^{-1}$]. The glycogen depletion pattern demonstrated that during bicycling as well as swimming the slow fibres were depleted earlier than the fast twitch fibres. Lactate

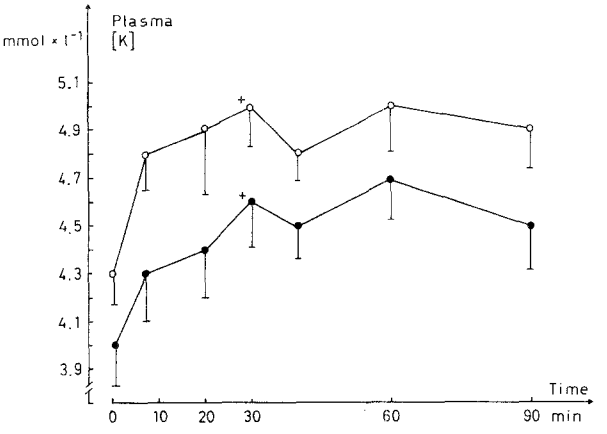

Fig. 4. Plasma potassium concentrations [K] measured during 90 min of swimming ● and bicycling ○. Average values and SE from five subjects are shown. + denotes the earliest values which were significantly different from resting values

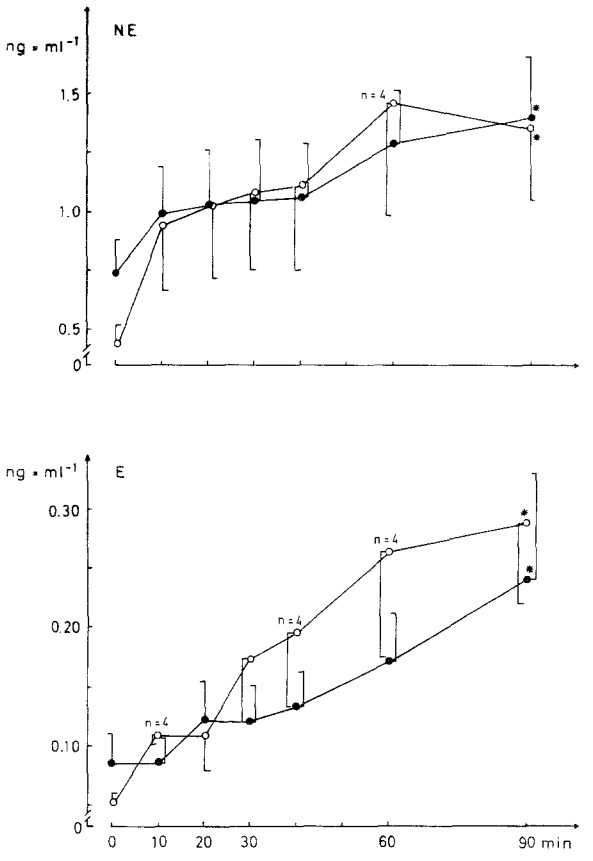

Fig. 5. Changes in plasma concentrations of norepinephrine (above) and epinephrine (below) during 90 min of swimming ● or bicycling ○. Average values from five subjects and SE. ✱ indicates significant differences from early exercise values (see text)

Table 3. Mean values and (range) of muscle H_2O, Na, K, Mg, lactate, and glycogen concentrations per kilogram dry weight analysed from muscle biopsies during swimming and bicycling in five subjects. $n = 4$ indicated by *

		Rest before	Exercise	
			20 min	90 min
H_2O	Swimming	3.02 (2.88–3.08)	3.17* (3.12–3.39)	
(1 × kg dw^{-1})	Swimming	3.00 (2.79–3.31)		3.46 (3.27–3.85)
	Bicycling	3.07 (2.95–3.18)	3.32 (3.29–3.39)	3.57 (3.29–3.74)
Na	Swimming	9.6* (3.3–21.1)	7.5 (4.6–10.0)	
(mmol × kg dw^{-1})	Swimming	6.6* (5.4–7.4)		12.2 (6.5–21.2)
	Bicycling	6.5 (3.6–7.4)	11.2 (7.8–13.7)	15.4 (9.4–20.5)
K	Swimming	396* (36.6–41.9)	403* (37.9–42.0)	
(mmol × kg dw^{-1})	Swimming	407* (39.9–42.5)		400* (36.7–42.9)
	Bicycling	410 (39.8–42.7)	390 (36.7–42.5)	371 (35.1–41.6)
Mg	Swimming	4.0* (3.7–4.4)	4.0* (3.7–4.4)	
(mmol × kg dw^{-1})	Swimming	3.9 (3.7–4.1)		3.7 (3.3–4.0)
	Bicycling	4.1 (3.7–4.8)	3.9 (3.7–4.0)	3.9 (3.4–4.2)
Lactate	Swimming	8.2* (6.1–10.4)	8.5 (7.2–9.3)	
(mmol × kg dw^{-1})	Swimming	6.6* (4.7–10.5)		6.8* (4.3–8.8)
	Bicycling	6.0 (4.1–7.7)	9.1 (5.8–17.7)	11.5 (5.9–15.2)
Glycogen	Swimming	359* (289–585)	380* (255–476)	
(mmol × kg dw^{-1})	Swimming	471* (379–564)		298* (114–408)
	Bicycling	493 (259–526)	344 (274–389)	207 (88–291)

accumulation in the muscle was insignificant after 20 min exercise. Only at the end of the bicycling a small significant increase had occurred (Table 3).

Hormonal changes. The plasma catecholamine concentrations increased during the entire exercise period both during swimming and bicycling (Fig. 5). Norepinephrine (NE) increased from about 0.6 to 1.4 ng $\times$ ml^{-1} both in swimming and bicycling (non significant in bicycling); and epinephrine (E) in plasma increased both in swimming and bicycling from approximately 0.07 to 0.26 ng $\times$ ml^{-1}. The plasma concentrations of E increased significantly from 10 to 90 min of swimming, and from 20 to 90 min in bicycling. The plasma NE was significantly higher at 90 min than at 20 min both in swimming and bicycling (two-way analysis of variance).

Plasma antidiuretic hormone (ADH) values were significantly increased after 90 min of bicycling, while mean values of ADH after swimming were unchanged (Table 2).

Discussion

The present experiments showed a secondary rise in HR both in swimming and bicycling. After the initial increase a gradual secondary rise in HR occurred during the prolonged, moderate exercise. Both during swimming and bicycling the rise in HR was positively related to the measured increases in plasma catecholamines. This suggests a possible effect of these substances, or of changes in sympathetic nervous activity (SNA) as a cause for the secondary rise in heart rate (Fig. 1). Plasma NE concentrations were increasing similarly in the two situations, while plasma E in bicycling followed a parallel but slightly higher curve than in swimming (Fig. 5).

During bicycle exercise with β-blockade using pindolol Cornet et al. (1977) found that the secondary rise in HR was smaller than in the control situation. They concluded that β-receptors were involved. This concurs with our findings. Cornet et al. also concluded that increasing core temperature contributed through a direct action on the heart muscle, decreasing its contractility at increasing temperature. We did not measure core temperature in the present experiments. However, during prolonged swimming we found in another series of experiments a secondary rise in HR without a change in core temperature between 30 and 90 min of swimming (Nielsen 1984). This speaks against a temperature effect on HR.

The upward drift in heart rate has previously been attributed to a fall in central blood volume, which may cause a decreased filling pressure of the heart, and thereby a decreased stroke volume. A decrease in central blood volume may be caused either by a redistribution of blood to the periphery or by a decrease in PV. The gradual thermoregulatory increase in skin blood flow has thus been mentioned as cause for the secondary rise in HR, and also the reduction in PV due to sweating during prolonged exercise (Saltin and Steenberg 1964; Ekelund 1966; Sawka et al. 1979; Cornet et al. 1977; Rowell et al. 1966, 1969). However, it seems unlikely that a continuous increase in skin blood volume takes place during 90 min swimming, where the core temperature remained stable from the 30th min as mentioned above. Neither can the observed drift in HR be due to a reduction in PV caused by a gradual dehydration due to sweating, since PV fell by approximately 250 ml within the first 10 min of exercise, but thereafter remained unchanged, both during swimming and bicycling. Thus, the observed drift in HR cannot be caused by low pressure baroreceptor reflexes from the atria or pulmonary circulation responding to reductions in filling pressure.

In our experiments, both swimming and bicycling, the cardiac output was maintained unchanged throughout. But it was significantly higher during swimming, and the (a$-$v) O_2 difference thus smaller for a similar oxygen uptake than during bicycling. Stroke volume was also higher during swimming, as observed by others (Keener and Sinning 1980; McArdle et al. 1976), but in both types of exercise we found a secondary rise in HR. At the end of exercise HR had increased 13 bpm on the average in both conditons (Fig. 1) without any change in $\dot{Q}$. The stroke volume, therefore, had decreased. These changes with time were not related to changes in plasma volume.

Water immersion of resting subjects is reported to induce a fall in Hct indicating a shift of extravascular fluid into the vascular bed (Graveline et al. 1963; McCally 1964; Khosla and DuBois 1979, 1981; Greenleaf et al. 1980; Bonde-Petersen et al. 1983). In contrast to this, we observed a fall in PV of 250 ml within the first 5$-$10 min during swimming exercise. This net decrease may be caused by the simultaneous shift of water into the exercising muscles. Assuming a 10-kg active muscle mass, it can be calculated from the muscle biopsies that about $^1/_2$ l of H_2O had diffused into the exercising muscles during 20 min of exercise, and more than 1 l after 90 min of exercise.

Although the sweat loss was almost four times greater ($P < 0.05$) during bicycling, the net water loss from the body was only about 25% higher after bicycling than after swimming (Fig. 2). This was due to the much larger urine production during swimming ($P < 0.05$) (Table 2). It appears that in an exercise

condition the "immersion-effects" on the filling of the heart results in ADH inhibition and significantly increased diuresis, as found for resting subject (Epstein et al. 1981; Khosla et al. 1979; Greenleaf et al. 1980).

Possible direct influences on HR reactions could have been brought about by an increase in muscle interstitial K concentration, which has been shown to affect cardiovascular responses (Hirche et al. 1980; Wildenthal et al. 1968). Plasma [K] which reflects interstitial [K] increased (most in bicycling), but remained unchanged during the last 60 min of exercise (Fig. 3).

Plasma K concentration was elevated above the resting value throughout both types of exercise, indicating a loss of K from the active muscles. Only after bicycling did muscle K content decrease significantly in M. vastus lateralis. But different muscle groups may well have been involved to different extents during swimming and bicycling. The lactate values were higher and the glycogen values lower in M. vastus lateralis at the end of the bicycle exercise than after swimming (Table 3), suggesting that this muscle performed relatively more of the total work during bicycling than during swimming. A continuous net loss of K from exercising muscles may occur only above a certain relatively high work load. The fact that plasma [K] attained the highest plateau values during bicycling further indicates that some muscles worked harder and therefore lost more K during bicycling than any muscle during swimming.

Conclusions

In spite of the differences in sweating and peripheral "pooling" due to hydrostatic forces, the gradual "secondary rise" in HR was the same during prolonged bicycling and swimming exercise. The rise was positively related to the increase in plasma catecholamines which occurred as exercise progressed. The stimulus for this increased sympathetic nervous activity/plasma catecholamine level remains obscure.

We could not substantiate previous hypotheses that the secondary rise in HR would be related to plasma volume changes, nor to the dehydration due to sweating. Neither was it related to changes in muscle or plasma K concentration.

Acknowledgements. We wish to express our gratitude to Drs. Jan Krogh Hansen, University of Odense, Institute of biochemistry, and Jørgen Warberg, University of Copenhagen, Medical Physiology Lab. C., who made the analyses on plasma catecholamines and ADH, respectively. The experiments were financed through grants from the Danish Sports Research Counsil, the Danish Space Board and the Danish Medical Research Foundation.

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Accepted May 1, 1984