DYNAMICS OF SERUM POTASSIUM DURING EXERCISE IN HEREFORD CALVES: INFLUENCE OF PHYSICAL CONDITIONING

by

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B.S., Kansas State University, 1981

A MASTER'S THESIS

submitted in partial fulfillment of the

requirements for the degree

MASTER OF SCIENCE

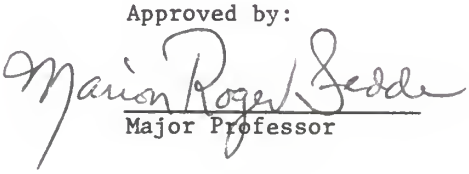
Department of Anatomy and Physiology

KANSAS STATE UNIVERSITY

Manhattan, Kansas

1987

Approved by:



Major Professor

ACKNOWLEDGMENTS

I wish to thank all those who have contributed effort and encouragement towards the completion of this project and thesis. To my major professor, Dr. M.R. Fedde, I am grateful and indebted for his patience, understanding, inspiration and guidance, throughout my coursework, and in instructing and encouraging me in the ways of a researcher. I thank the members of my graduate committee, Drs. Howard H. Erickson, Katharine K. Grunewald, and Robert D. Reeves for their valuable suggestions. For technical assistance, I thank Rick Pieschl, Pam Miller-Davis, Karen Eyer, Scott Van Pelt and Karen TerHune. Dr. George Milliken and Mr. Jeff Pontius provided assistance with the statistical analysis.

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ABSTRACT

We determined how rapidly serum potassium concentration ($[K^+]$) increased, its magnitude, and how quickly it decreased, during and following a 3.5 min exercise bout at maximal speed capability in 8 Hereford steers before and after physical conditioning. Physical conditioning was accomplished by a progressive increase in the duration of exercise from 5 to 30 $min \cdot day^{-1}$, 5 $days \cdot week^{-1}$, over a 6-week period, at an intensity of 60%-70% of each animal's maximum heart rate. Serum $[K^+]$ values rose rapidly after the start of exercise and declined rapidly to within 7% of preexercise values 5 min after exercise ceased. Before physical conditioning, serum $[K^+]$ increased from an average of 4.19 $mEq \cdot l^{-1}$ at rest to 6.71 $mEq \cdot l^{-1}$ at the highest treadmill speed the animals could sustain (1.8 to 2.4 $m \cdot s^{-1}$ at a 3° incline). Following physical conditioning, the serum $[K^+]$ increase at comparable treadmill speeds was about 5% lower than before conditioning (average of 6.37 $mEq \cdot l^{-1}$); however, the animals could now exercise from 0.6 to 0.8 $m \cdot s^{-1}$ faster than before conditioning and their maximal serum $[K^+]$ rose to an average of 7.47 $mEq \cdot l^{-1}$, a 10% increase over preconditioned maximal values. We conclude that serum $[K^+]$ changes rapidly at both onset and cessation of exercise with peak levels that are influenced by physical conditioning. The rise in serum $[K^+]$ appears to be linked to the speed of the exercise and, thereby, the number of contractions of the muscle fibers per unit time.

INTRODUCTION

Serum potassium concentration ($[K^+]$) increases during exercise and returns rapidly toward normal levels when exercise stops. This observation has been made in a wide variety of animal species: fish (26); cat (9, 11); rabbit (9); pig (6); dog (8, 16); calf (13); pony and horse (7, 18); and human (14, 21, 22). In most animals, normal values for serum $[K^+]$ range from 3.5 to 4.5 $mEq \cdot l^{-1}$ and hyperkalemia is considered to occur when the concentration exceeds 6 $mEq \cdot l^{-1}$ (23). It is generally assumed that K^+ moves out of skeletal muscle cells during contraction (8, 10).

Elevated extracellular $[K^+]$ has a marked influence on cardiac cells. Prominent ECG changes occur when plasma $[K^+]$ reaches values of 6-7 $mEq \cdot l^{-1}$. Rapid and prolonged depression of myocardial conductance occurs progressively as the plasma $[K^+]$ exceeds 8 $mEq \cdot l^{-1}$ and the ECG reveals "peaking" T-waves. As $[K^+]$ exceeds 10 $mEq \cdot l^{-1}$, asystole is a predominant mechanism for cardiac failure (23).

Beef cattle are sedentary animals that generally do not exercise voluntarily at heavy workloads; however, they are often forced into exertion during handling and shipping procedures. Kuhlmann et al. (13) measured serum $[K^+]$ 63% above resting values (7.0 $mEq \cdot l^{-1}$) during exercise in Hereford calves at a workload inducing maximal O_2 consumption. Because of the importance of hyperkalemia to cardiac function, we studied the dynamics of this ion with exertion in beef calves and the possible modulation of its increase with physical conditioning.

METHODS

Animals. Eight Hereford steer calves (Bos taurus) were obtained locally and housed in indoor stalls under a controlled environment (65-70°F; 40% R.H.). They were fed daily a mixture of alfalfa, prairie hay, dehydrated alfalfa pellets, and 0.5 kg of a vitamin-mineral mixture in cracked corn. A mineral salt block and water were provided ad libitum. Their average body weight was 169 ± 35 (SD) kg at the beginning of the study and 210 ± 21 (SD) kg at the end of the study with an average weight gain of 0.7 ± 0.3 (SD) $kg \cdot day^{-1}$.

Animal preparation. During the two weeks following purchase, the calves were trained to lead, accustomized to handling and being transported in a cart, and taught to walk on a treadmill. This initial training period did not include a physical conditioning program.

Following the training period, a 53 cm vinyl catheter (0.970 mm I.D., 0.143 mm O.D.) was inserted into the descending aorta under local anesthesia according to the technique of Will and Bisgard (24). The catheter remained in place during preconditioning measurements, but was removed during the 6 week conditioning period. The catheter was replaced again for postconditioning measurements.

Measurements. The dependent variables measured in this study included heart rate, body temperature, serum potassium and sodium concentrations, whole blood lactate concentration, hemoglobin, and hematocrit. The independent variables were the speed of the treadmill

(Anamill, Horsey, Inc.) set at an incline of 3° and the exercise capabilities of the animals.

Heart rate (HR) was determined from an electrocardiogram (ECG) with active electrodes, consisting of copper foil discs (4 cm dia.), placed in the middle of the forehead (-) and tail head (+), and a ground electrode attached to the skin of the rump. Although this electrode configuration is not a conventional lead system, it minimized movement artifact and allowed heart rate to be determined during exercise. Changes in the ECG waveform could only be observed when the animals were standing still on the treadmill.

Body temperature (T_b) was measured by inserting a calibrated thermistor (Yellow Springs Inst., Model 401) 15 cm into the lower intestinal tract and reading the value from a telethermometer (Yellow Springs Inst., Model 43TA).

Arterial blood samples were withdrawn from the indwelling aortic catheter into sterile evacuated tubes (10 ml volume). The samples were allowed to clot and immediately centrifuged to collect the serum. Serum [K^+] and [Na^+] were determined by ion selective electrodes (Nova 1 Sodium/Potassium Analyzer, Biomedical). Hemoglobin concentration [Hb] in arterial blood was measured with a hemoglobinometer (American Optical) and hematocrit (Hct) was determined on duplicate samples using the microhematocrit method. Lactate concentration [Lac^-] in whole arterial blood was determined by an enzymatic method (Technical Bulletin No. 826-UV, Sigma Chemical Co.).

Experimental protocol. On each day of the study, the animal was

weighed, transported to the laboratory, led onto the treadmill, and prepared for the experiment. Preparation included placement of the ECG electrodes and insertion of the thermistor into the lower intestinal tract. The animal was then allowed to stand quietly on the treadmill for a period of 20 to 30 min.

The study was divided into three phases: a. preconditioning measurement phase, (1 week); b. physical conditioning phase, (6 weeks); and c. postconditioning measurement phase, (1 week).

a. Preconditioning measurement phase. A few days after aortic catheterization, each animal was exercised for 3.5 min periods at progressively faster speeds (increments of 0.2 $m \cdot s^{-1}$) allowing sufficient time (15 min to 2 hours) between each exercise period for heart rate to return to near resting levels. Exercise started from a slow walk (0.6 $m \cdot s^{-1}$) and ended at the highest speed the animal could sustain for 3.5 min. Measurements were made and blood samples collected before (rest) and 3 min into each exercise bout. Previous studies have shown that O_2 consumption and other variables measured in this study reached a steady state by this time (13). The measurements were repeated twice on consecutive days. These data indicated the progression of change of the measured variables with increasing treadmill speed and demonstrated the magnitude of these variables at the animals highest attainable running speed for 3.5 min.

The calves were then exercised on two successive days at the highest speed they could sustain for 3.5 min. Heart rate was continuously monitored and blood samples were drawn at 30 sec intervals starting from one min before start of exercise to 5 min postexercise.

Additional measurements were made 10 min postexercise. These tests allowed determination of the dynamics of HR, $[K^+]$, and $[Na^+]$ during and following exercise.

b. Physical conditioning phase. After completing the preconditioning measurement phase, the animals entered a physical conditioning program. Each animal was exercised 5 days·week^{-1}, at a treadmill speed inducing 60-70% of its maximum heart rate, beginning with five min the first day and adding one min per day for the first two weeks. Thereafter, five min were added at the beginning of each subsequent week until each animal had exercised for 6 weeks at a final time of 30 min per day. Heart rate was monitored during each exercise period so the treadmill speed could be adjusted, if necessary, to maintain 60-70% of maximum.

c. Postconditioning measurement phase. Each animal was recatheterized and exercised on two consecutive days at the highest speed it could sustain in the preconditioning measurement phase; the time intervals of blood sampling and the measured variables were the same as before conditioning. These exercise bouts at similar workloads allowed comparision of the effects of physical conditioning.

The animals were then studied during incremental increases in treadmill speed, utilizing the same procedure as before conditioning, to attain their new maximum speed. At their new maximum speed (the highest treadmill speed the animals could sustain for 3.5 min), the animals were exercised twice on consecutive days with blood sampling at 30 sec intervals, as previously described, to compare the dynamics of the measured variables during and following exercise.

Data Analysis. Duplicate measurements for a given calf were averaged; then, the means of all calves were averaged for each conditioning phase. The pre- and postconditioning means were then compared and evaluated by a one-way analysis of variance or F-test for split-plot design with missing data (15) to determine if significant differences ($P < 0.05$) existed between the mean values.

RESULTS

Effect of physical conditioning on the measured variables at comparable treadmill speeds. Before conditioning, all animals were able to sustain speeds of 1.8 $m \cdot s^{-1}$ for 3.5 min, with two capable of sustaining 2.6 $m \cdot s^{-1}$. Following conditioning, all animals were able to attain peak speeds of 2.4 $m \cdot s^{-1}$ for this time interval, with one animal able to run at 3.2 $m \cdot s^{-1}$. Values of the measured dependent variables at the various speeds are shown in Table 1.

Following physical conditioning, maximal values for heart rate, blood lactate, hematocrit and $[Na^+]$ were delayed until higher treadmill speeds were reached. In addition, physical conditioning caused a small but significant decrease in serum $[K^+]$ at treadmill speeds where all eight animals could be compared.

Effect of physical conditioning on the dynamics of measured variables at maximal capabilities. Responses of the animals were compared at the maximal speed capability they could attain before conditioning, at that same speed following conditioning, and at the maximal speed capability after conditioning.

TABLE 1. Effect of physical conditioning on the measured variables at progressively increasing treadmill speeds

Speed $m \cdot s^{-1}$	n		Heart Rate $beats \cdot min^{-1}$		$[K^+]$ $mEq \cdot l^{-1}$		$[Na^+]$ $mEq \cdot l^{-1}$		$[Lac^-]$ $mg \cdot dl^{-1}$		T_b °C		Hct (%)		Hb $gm \cdot dl^{-1}$	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
0	8	8	70 ±3	71 ±2	4.4 ±0.1	4.2* ±0.1	142 ±1	143 ±1	14 ±2	10* ±1	39.0 ±0.1	38.7* ±0.1	30 ±1	29 ±1	10.3 ±0.2	10.3 ±0.3
0.6	8	8	116 ±5	117 ±5	5.0 ±0.1	4.6* ±0.1	143 ±1	143 ±1	14 ±2	8* ±1	39.2 ±0.1	38.9* ±0.1	32 ±1	30* ±1	11.3 ±0.3	10.8 ±0.4
1.0	8	8	148 ±6	135* ±7	5.4 ±0.1	4.8* ±0.1	143 ±1	143 ±1	17 ±2	8* ±1	39.4 ±0.1	39.0* ±0.1	33 ±2	31* ±1	11.7 ±0.4	11.4 ±0.3
1.4	8	8	190 ±7	163* ±7	5.9 ±0.2	5.2* ±0.1	145 ±1	144* ±1	41 ±7	11* ±2	39.7 ±0.1	39.2* ±0.1	37 ±1	33* ±1	12.9 ±0.3	12.2 ±0.4
1.6	8	8	212 ±6	182* ±9	6.3 ±0.2	5.6* ±0.1	147 ±1	144* ±1	60 ±8	21* ±5	39.9 ±0.1	39.3* ±0.1	38 ±1	35* ±1	13.3 ±0.3	13.0 ±0.4
1.8	8	8	216 ±4	203* ±9	6.3 ±0.1	5.9* ±0.2	148 ±1	146* ±1	68 ±6	38* ±9	39.9 ±0.1	39.5* ±0.1	38 ±1	37 ±1	13.4 ±0.3	13.8 ±0.4
2.0	3	8	220 ±3	214 ±8	6.7 ±0.3	6.2 ±0.2	149 ±2	147 ±1	71 ±9	50* ±10	39.9 ±0.1	39.5 ±0.1	38 ±1	38 ±1	13.5 ±0.2	14.5 ±0.4
2.2	2	8	229 ±14	219 ±7	7.0 ±0.7	6.4 ±0.1	149 ±2	148 ±1	81 ±3	60* ±10	39.9 ±0.1	39.5 ±0.1	39 ±1	39 ±1	13.7 ±0.2	14.7 ±0.4
2.4	2	8	225 ±12	224 ±6	6.9 ±0.8	6.5 ±0.2	149 ±2	149 ±1	84 ±1	68* ±8	39.9 ±0.0	39.6 ±0.1	38 ±1	39 ±1	13.8 ±0.2	14.9 ±0.4
2.6	2	4	230 ±15	223 ±3	7.4 ±1.1	6.6 ±0.3	151 ±1	149* ±1	93 ±6	67* ±14	39.9 ±0.0	39.6 ±0.2	38 ±0	39 ±1	13.8 ±0.0	15.1 ±1.0
2.8		2		224 ±3		6.6 ±0.2		149 ±2		73 ±0		39.6 ±0.4		39 ±1		14.9 ±0.1
3.0		2		230 ±1		6.7 ±0.3		149 ±1		78 ±0		39.6 ±0.4		39 ±1		15.1 ±1.0
3.2		1		228 ±0		7.0 ±0.0		151 ±0		82 ±0		39.6 ±0.0		39 ±1		14.9 ±0.0

*Denotes significant difference ($P < 0.05$) from preconditioning measurement. Values are means ±SE. n = no. of animals contributing to mean values at the respective speeds. Pre = preconditioning phase and Post = postconditioning phase of experiment.

Heart rate increased rapidly during the first minute of exercise reaching a peak value of 235 ± 4 $b \cdot min^{-1}$ at the maximal postconditioned speed capability (Fig. 1). With cessation of exercise, heart rate decreased most rapidly during the first two min of recovery, then decreased more slowly toward resting values. At comparable speeds, heart rate was not significantly influenced by physical conditioning, but it was significantly higher at the maximal postconditioned exercise capability and tended to return more slowly toward resting values when the treadmill was stopped.

Serum $[K^+]$ increased rapidly during the first min of exercise both before and after physical conditioning (Fig. 2). At the end of exercise, values were: 6.71 ± 0.11 $mEq \cdot l^{-1}$ at the animals' maximal preconditioned speed, 61% over rest values; 6.37 ± .11 $mEq \cdot^{-1}$ at the same speed after conditioning, 51% over rest values; and 7.47 ± .11 $mEq \cdot l^{-1}$ at the maximal postconditioned speed, 76% over rest values. After exercise ceased, serum $[K^+]$ decreased rapidly within the first two min, then more slowly toward the resting values. After physical conditioning, peak $[K^+]$ was significantly increased (10%) over maximal preconditioned values (solid circles of Fig.2) at the higher maximal speed capability but was significantly reduced (5%) when the animals ran at the same maximal preconditioned speeds.

Sodium concentration increased under all conditions during exercise. Physical conditioning had no significant influence on serum $[Na^+]$ when animals ran at the same treadmill speeds (Fig. 3). At maximal exercise speed following conditioning, peak serum $[Na^+]$ was significantly elevated. Serum $[Na^+]$ returned rapidly toward resting

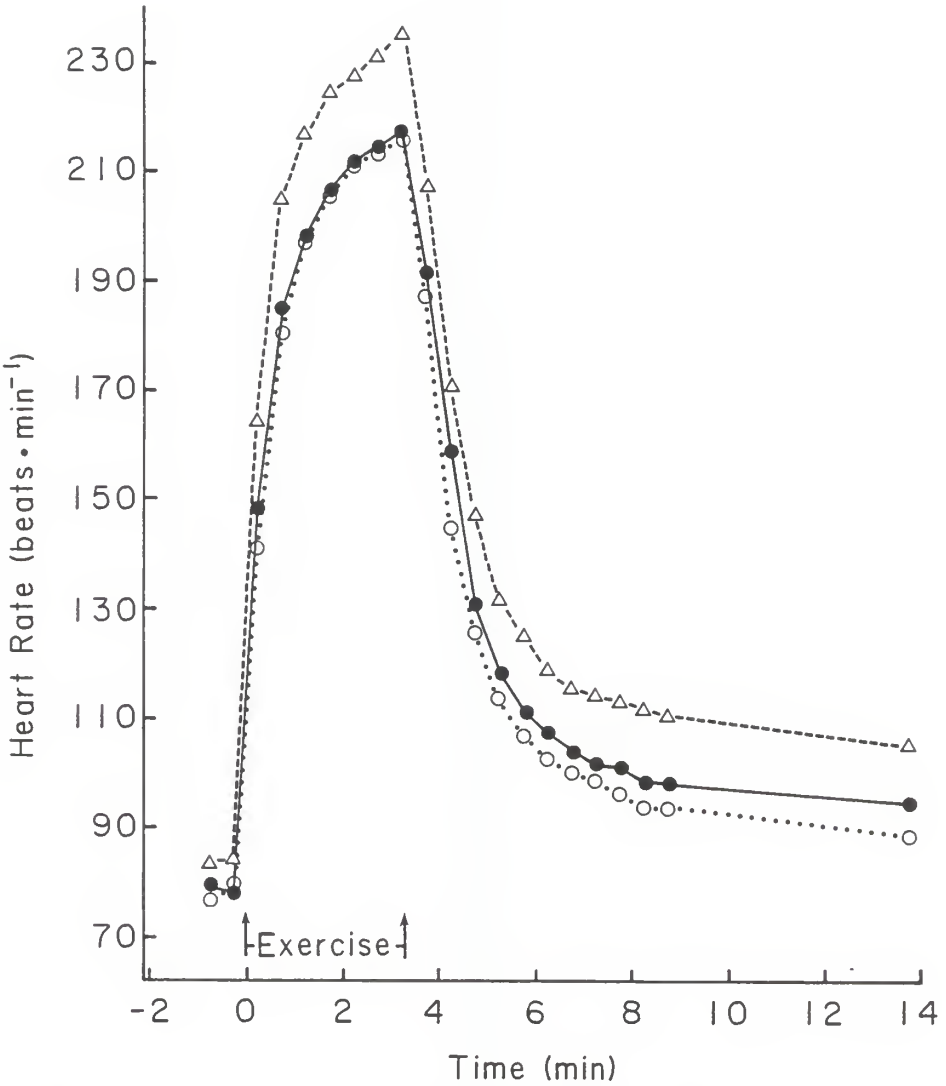

FIG. 1. Heart rates during rest and exercise before and after physical conditioning. Each point represents the mean of a 30 sec measurement from 8 calves. Closed circles represent preconditioned animals at their maximal exercise speed (range of 1.8 $m \cdot s^{-1}$ to 2.6 $m \cdot s^{-1}$); open circles represent postconditioned animals at their preconditioned maximal exercise speed; open triangles represent postconditioned animals at their maximal exercise speed (range of 2.4 $m \cdot s^{-1}$ to 3.2 $m \cdot s^{-1}$). The average SE for all points was $\pm$ 3.5.

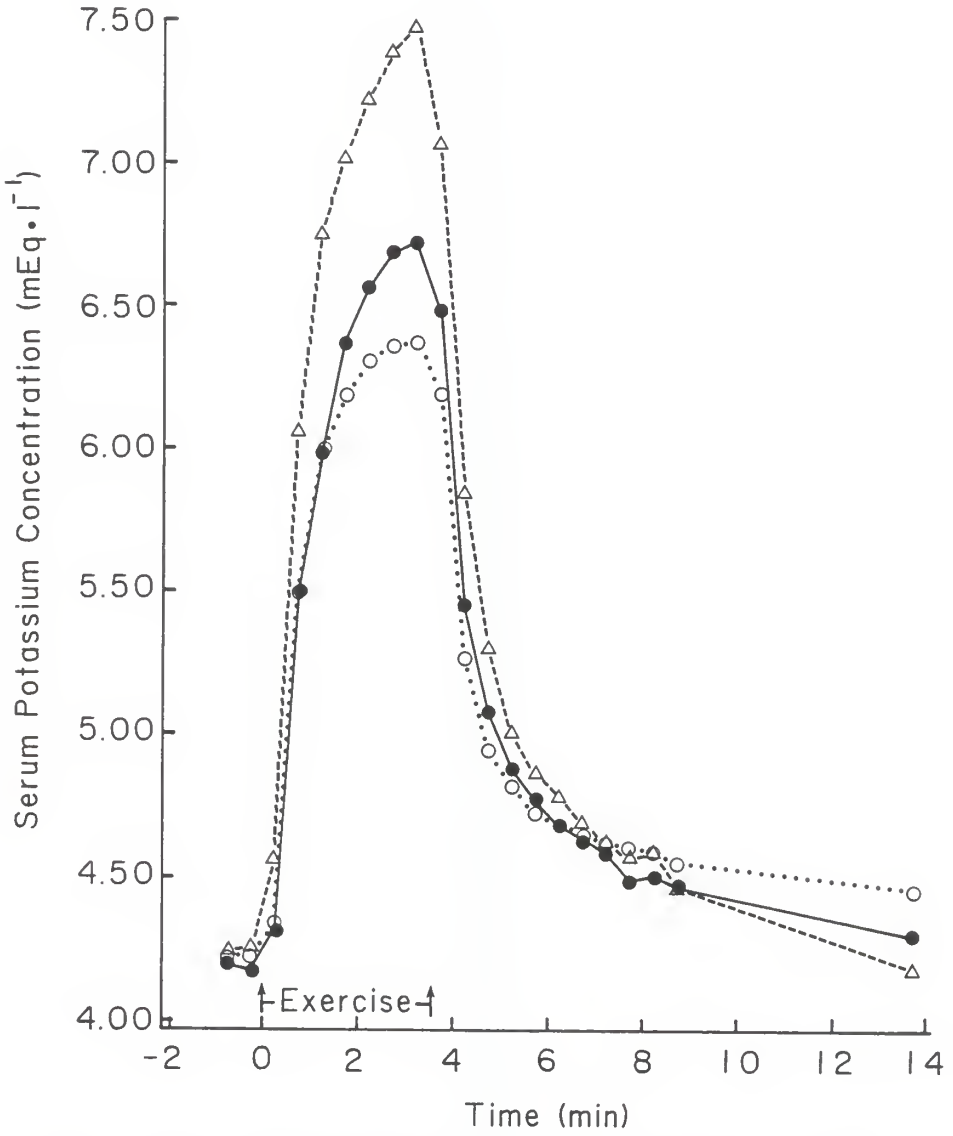

FIG. 2. Serum potassium concentrations during rest and exercise before and after physical conditioning. Each point represents the mean of a 30 sec measurement from 8 calves. Closed circles represent preconditioned animals at their maximal exercise speed (range of 1.8 $m \cdot s^{-1}$ to 2.6 $m \cdot s^{-1}$); open circles represent postconditioned animals at their preconditioned maximal exercise speed; open triangles represent postconditioned animals at their maximal exercise speed (range of 2.4 $m \cdot s^{-1}$ to 3.2 $m \cdot s^{-1}$). The average SE for all points was $\pm$ 0.11.

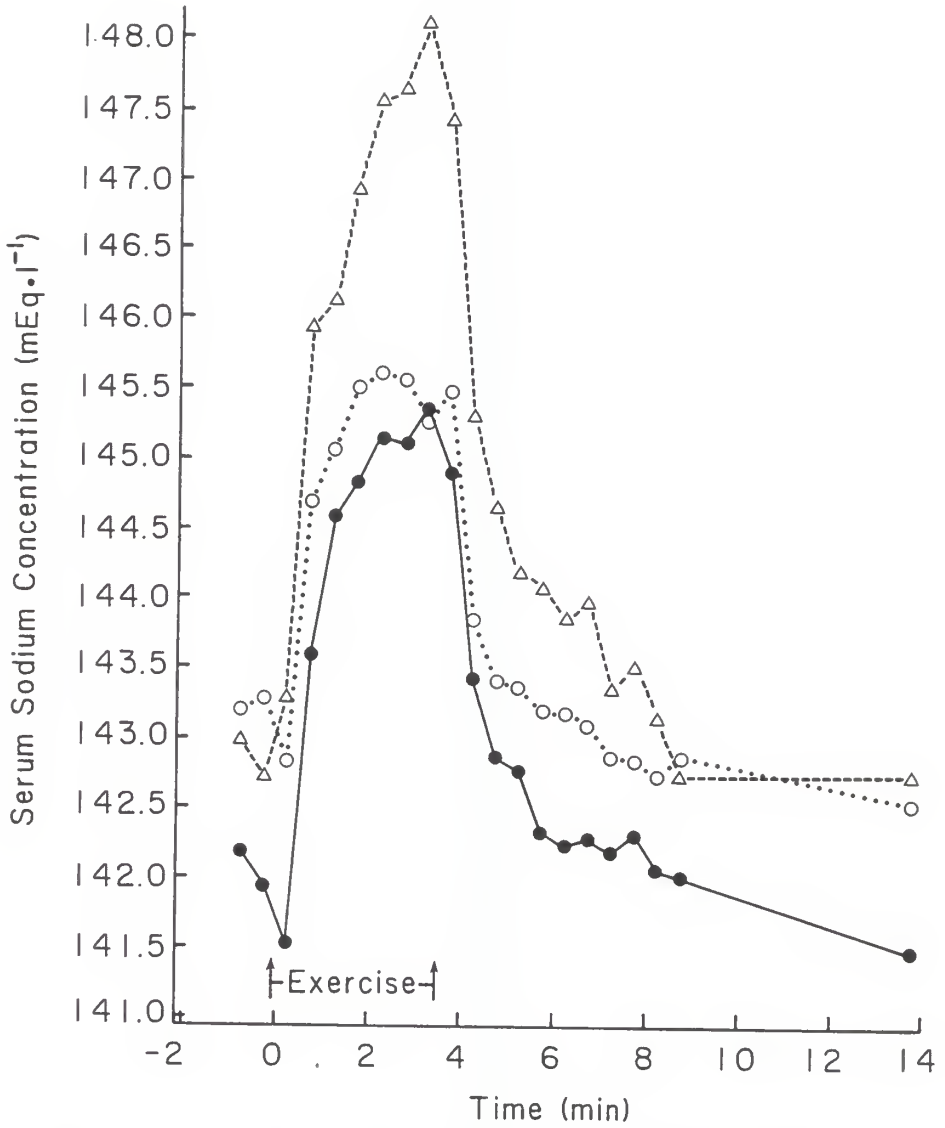

FIG. 3. Serum sodium concentrations during rest and exercise before and after physical conditioning. Each point represents the mean of a 30 sec measurement from 8 calves. Closed circles represent preconditioned animals at their maximal exercise speed (range of 1.8 $m \cdot s^{-1}$ to 2.6 $m \cdot s^{-1}$); open circles represent postconditioned animals at their preconditioned maximal exercise speed; open triangles represent post conditioned animals at their maximal exercise speed (range of 2.4 $m \cdot s^{-1}$ to 3.2 $m \cdot s^{-1}$). The average SE for all points was $\pm$ 0.5.

values after exercise ceased with approximately the same time course as for $[K^+]$.

The electrocardiogram was markedly altered by elevated serum $[K^+]$. The P-wave was undetectable in the ECG at maximal speeds both before and after physical conditioning (Fig. 4). After conditioning, P-waves were present at the maximal preconditioned speeds accompanied by a significantly lowered serum $[K^+]$. Elevation of the T-wave accompanied the increased serum $[K^+]$ during exercise in all cases.

DISCUSSION

Critique of methods. Although large volumes of blood (250 ml) were withdrawn from each calf each day of the experiment, only 1.8% of estimated total blood volume was taken and signs of depletion were not observed. Hematocrit remained similar during the resting periods within an experiment and from day to day as multiple experiments were conducted.

The arterial blood, drawn from the aorta via the indwelling catheter, provided a mixed sample representing the electrolyte and lactate concentrations that circulated throughout the body during exercise. The catheter placement did not interfere with the exercising animal.

Physical conditioning procedures have not been described for cattle; therefore, we conditioned our animals by endurance and aerobic exercise methods suggested for humans (3), i.e., at 60-70% of their maximum heart rate (speeds from 0.8 to 1.1 $m \cdot s^{-1}$). The conditioning

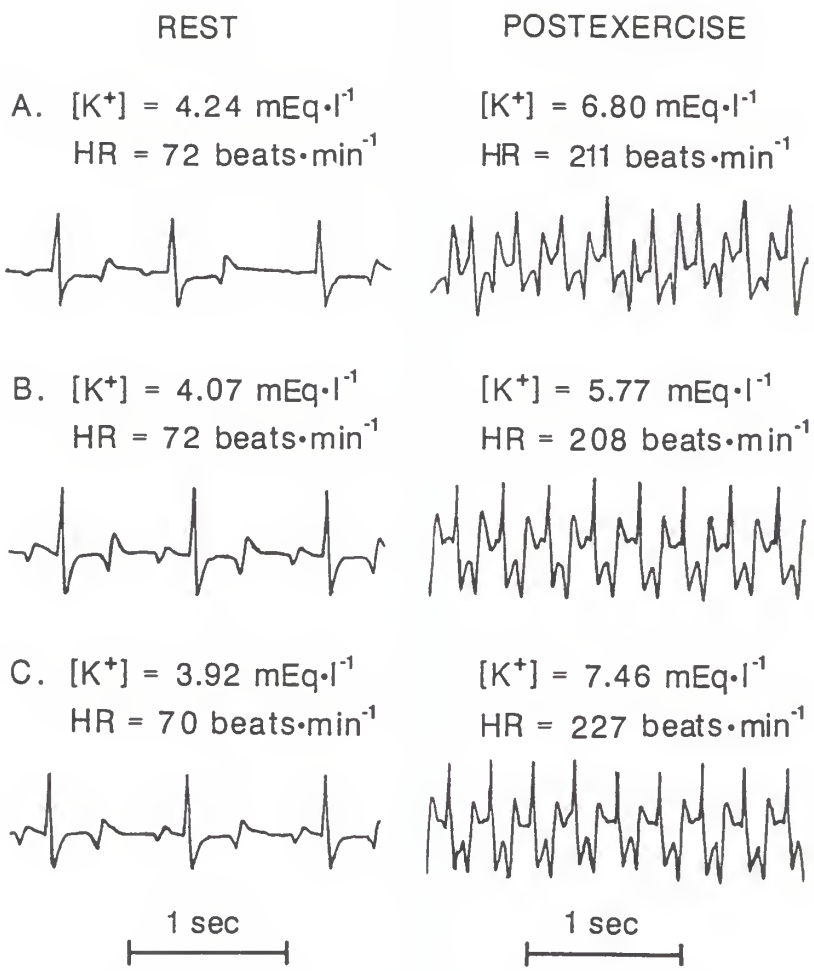

FIG. 4. ECG patterns during all three phases of conditioning from the same calf. Postexercise tracings were obtained within the first 5 sec after the treadmill was stopped. A. Rest and postexercise (1.8 $m \cdot s^{-1}$) at the preconditioned maximal exercise speed. B. Rest and postexercise (1.8 $m \cdot s^{-1}$) at the same speed as preconditioned maximal exercise speed following conditioning. C. Rest and postexercise (2.6 $m \cdot s^{-1}$) at the postconditioned maximal exercise speed.

speeds created a workload just below that producing a large increase in blood lactate (Table 1). Thus, this heart rate value appears to be useful in assessing the desired exercise conditioning regime in cattle.

Preliminary experiments had indicated that peak serum $[K^+]$ occurred within 3 min of a 5 min bout of exercise. We, therefore, chose to exercise the calves for 3.5 min and to make the last measurements during the last 30 sec. Furthermore, during the higher speeds, the animals could not run longer than 3.5 min. When more animals had been studied, it became apparent that serum $[K^+]$ had not plateaued at this time, especially when maximal speeds were accomplished (Fig. 2). However, the rate of rise in serum $[K^+]$ was markedly reduced after 3 min of exercise and it is probable that only a small additional increase would have occurred if the exercise period could have been extended.

Changes in serum $[K^+]$ with exercise. The serum $[K^+]$ increased rapidly (within 1-1 1/2 min) after the onset of exercise (Fig. 2). With increasing treadmill speeds, a progressive increase in serum $[K^+]$ occurred (Table 1); thus, the rise in $[K^+]$ seemed to be proportional to the number of muscular contractions per unit time.

Exercised induced hyperkalemia ($[K^+] > 6.0$ mEq$\cdot l^{-1}$) occurred at speeds above 1.4 m$\cdot s^{-1}$ in most of our unconditioned animals. Conditioning resulted in a significant decrease in serum $[K^+]$ at comparable treadmill speeds and hyperkalemia did not occur until speeds of about 1.8 m$\cdot s^{-1}$ were reached. Conditioning also resulted in an increased performance capability enabling the animals to run much faster and, as a result, serum $[K^+]$ rose to an even higher

concentration in the blood than had occurred in these animals at their maximal speed capabilities before conditioning (Fig. 2).

Many studies have tried to determine the causitive factor of the rise in serum $[K^+]$ during exercise. It is widely assumed that the contracting muscle cells release the K^+ ions. Increase in lactate production directly and increased PCO_2 indirectly may influence the extrusion of K^+ out of the exercising muscle cell (22). Alpha-adrenergic receptor stimulation, due to increased catecholamine activity during exercise, is also thought to be associated with stimulating increased release of K^+ from the muscle cells (25). An increase in the number of action potentials generated (9) and an incomplete reuptake of K^+ after each depolarization (19) could result in K^+ accumulation in the capillary blood. The increased muscle blood flow during exercise would then move the K^+ away from the muscle, causing the increase in the serum concentrations observed. An inhibition of the Na^+-K^+-pump, because of a deficit in energy rich phosphates (8), may inhibit the return of K^+ to the cell interior.

After exercise ceases, the exit of K^+ from muscle cells is greatly reduced and serum $[K^+]$ declines rapidly to near resting levels (Fig. 2). The muscle cells then participate in buffering the acute increase in serum $[K^+]$ resulting in the extrarenal reuptake of these ions (2). Beta-adrenergic receptor stimulation by circulating catecholamines, which are increasingly released during exercise, may stimulate cellular uptake of K^+ (25). Insulin may also play a role in augmenting cellular uptake of K^+ and maintenance of normal extrarenal K^+ metabolism (1). Even though serum $[K^+]$ and $[Lac^-]$ have been correlated during exercise,

there is a dissociation of serum [Lac^-] and [K^+] following exercise during the reuptake of K^+ (7).

Influence of physical conditioning on serum [K^+] changes during exercise. Our study showed that physical conditioning reduces the degree of hyperkalemia at a given level of exercise in the calf, in agreement with the results of Knochel et al. (12). Physical conditioning may evoke a number of physiological responses that lessen the release of potassium from the muscle cells and aid in returning the ion to the cell interior. Physical conditioning increases blood volume and cardiac output, O_2 carrying capacity, and mitochondria in the muscle. Conditioning is thought to have a greater effect on mitochondria in the subsarcolemmal location thereby improving the integrity of the cell membrane (3), perhaps resulting in a larger production of ATP to be used to improve pump activity. Physical conditioning increases Na^+-K^+ exchange across the sarcolemmal membrane because of an increase in Na^+-K^+ ATPase activity induced by an increased electronegativity of the pump and a net increase of pump units per unit mass of sarcolemma (12). Less lactate is produced with training and buffering of CO_2 is enhanced (3); thus, extrusion of K^+ out of the cell due to changes in [H^+] is reduced. After conditioning, exercise at comparable workloads is less of a stress and induces a reduced catecholamine response (3) which may result in a decreased release of K^+ from the muscle cells. Insulin does not fall as far during exercise following conditioning (3) and may result in increased regulation of K^+ homeostasis. Conditioning fosters recruitment of slow-twitch fibers (3) which have a lower [K^+] and higher [Na^+] (4, 20)

and results in improvement of the fibers oxidative capacity (3); thus, fewer fast- twitch fibers would be recruited to perform the same task.

After physical conditioning, our animals could run faster and serum $[K^+]$ rose higher than the values measured from the animals at their maximal speed capability in their unconditioned state. This represents a new exercise stress situation as the animals go beyond their previous conditioned capability. This increase in serum $[K^+]$ may reflect an increase in motor unit recruitment involving the action of additional muscle fibers. As the speed of running increases, there is increased recruitment of fast-twitch fibers (3), which contain more $[K^+]$ (4, 20) and contract more rapidly (3). These fibers may lose more K^+ to the extracellular fluid than the slow-twitch fibers and, hence, cause the increase in serum $[K^+]$.

Relationship of extracellular $[K^+]$ to excitability of nerve and muscle cells. The $[K^+]$ difference across the membrane of nerve and muscle cells is the principle determinant of the resting potential of a cell. Potassium is accumulated in cells by an energy dependent process so that intracellular potassium concentration far exceeds that in extracellular fluid. If the $[K^+]$ gradient across the cell membrane decreases, the resting membrane potential will decline. Furthermore, since the quantity of K^+ located outside the cells is so small, small shifts in K^+ into or out of cells produce large changes in extracellular $[K^+]$ and, thereby, a large change in the $[K^+]$ gradient across the membrane. A gain or loss of only 1% of the total body K^+ from the extracellular space can result in a 50% increase (or decrease) in plasma $[K^+]$ (5).

If $[K^+]$ in intra- and extracellular fluids are 155.0 $mEq \cdot l^{-1}$ and 4.0 $mEq \cdot l^{-1}$, respectively, the potassium equilibrium potential, E_{K^+}, calculated with the Nernst equation would be -95 mV. From the average change in serum $[K^+]$ at maximal preconditioned speed (6.71 $mEq \cdot l^{-1}$), a minimum change in E_{K^+} to -82 mV would be predicted. Thus, the resting membrane potential of nerve and muscle cells might be expected to decrease by 13 mV, resulting in increased excitability for those cells whose threshold has not been exceeded and much decreased excitability for those whose threshold had been surpassed. Following conditioning, the animals higher serum $[K^+]$, during maximal exercise, would result in an even lower resting membrane potential and accentuate the number of cells rendered inexcitable. Because the $[K^+]$ of the extracellular fluid is probably higher than that in the venous and arterial blood during exercise, an even larger depolarization of the cell membrane would be expected than is predicted from $[K^+]$ in the blood.

Cardiac cells are greatly affected by changes in the $[K^+]$ gradient across their membrane. Alterations of this gradient can alter the electrical stability and coordinated contraction. As extracellular $[K^+]$ increases, depolarization of the cell membrane and reduction in conduction velocity occurs. Eventually, re-entry ventricular tachycardia may degenerate into ventricular fibrillation and death (17).

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DYNAMICS OF SERUM POTASSIUM DURING EXERCISE IN HEREFORD CALVES: INFLUENCE OF PHYSICAL CONDITIONING

by

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B.S., Kansas State University, 1981

AN ABSTRACT OF A MASTER'S THESIS

submitted in partial fulfillment of the

requirements for the degree

MASTER OF SCIENCE

Department of Anatomy and Physiology

KANSAS STATE UNIVERSITY

Manhattan, Kansas

1987

We determined how rapidly serum potassium concentration ($[K^+]$) increased, its magnitude, and how quickly it decreased, during and following a 3.5 minute exercise bout at maximal speed capability in 8 Hereford steers before and after physical conditioning.

Each animal's maximal capability was determined by progressively increasing the treadmill speed to a value the animal could just sustain for a 3.5 minute exercise bout. Arterial blood samples were obtained from an indwelling aortic catheter. The measured variables obtained during this test included heart rate, serum potassium and sodium concentrations, whole blood lactate concentration, body temperature, hemoglobin and hemotocrit. Each animal was then physically conditioned by a progressive increase in duration of exercise from 5 to 30 $minutes \cdot day^{-1}$, 5 $days \cdot week^{-1}$, over a six week period, at an intensity of 60-70% of each animal's maximum heart rate. Responses of the animals were compared at the maximal speed capability they could attain before conditioning, at that same speed following conditioning, and at the maximal speed capability after conditioning. The measured variables obtained during these tests included serum $[K^+]$ and $[Na^+]$ concentrations and heart rate.

Serum $[K^+]$ values rose rapidly after the start of exercise and declined rapidly to within 7% of preexercise values 5 minutes after exercise ceased. Before physical conditioning, serum $[K^+]$ increased from an average of 4.19 $mEq \cdot l^{-1}$ at rest to 6.71 $mEq \cdot l^{-1}$ at the highest treadmill speed the animals could sustain for the desired time period (1.8 to 2.4 $m \cdot s^{-1}$ at a 3° incline). Following physical conditioning,

the serum $[K^+]$ increase at comparable treadmill speeds was 5% lower than before conditioning (average of 6.37 $mEq \cdot l^{-1}$); however, the animals could now exercise from 0.6 to 0.8 $m \cdot s^{-1}$ faster than before conditioning and their maximal serum $[K^+]$ rose to an average of 7.47 $mEq \cdot l^{-1}$, a 10% increase over precondition maximal values.

Serum $[K^+]$ changes rapidly at both the onset and the cessation of exercise with peak levels that are influenced by physical conditioning. The rise in $[K^+]$ appears to be linked to the speed of the exercise and, thereby, the number of contractions of the muscle fibers per unit time.