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Prediction of physical performance through muscle enzymes activity

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Summary. Physical performance deteriorates during strenuous exercise as manifested by a decrease in maximal aerobic power and increased activity of serum muscle enzymes. The relationship between these parameters was investigated in 41 trained subjects during 24 h marches and the following recovery period. Peak O_2 uptake and serum activity of creatine phosphokinase (CPK) and glutamic oxalacetic transaminase (GOT) were measured. During the marches there was a simultaneous, significant elevation in serum CPK and GOT activity and a significant reduction in peak O_2 uptake. During the early recovery period (24 h) no significant changes occurred in muscle enzyme activity and peak O_2 uptake; thereafter (up to 72 h after the end of the march), a gradual decline in enzyme activity levels with a concomitant increase in peak O_2 uptake was observed, reaching pre-march values. A "mirror image" relationship between muscle enzyme activity and peak O_2 uptake was found during three clearly distinguished phases: a) 24 h march, b) early recovery stage and c) late recovery stage. These findings suggest that muscle enzyme leakage from muscle cells is closely related to the decline in muscular function and aerobic power. Thus, muscle enzyme activity might be a practical measure of physical performance capacity during the early and late stages of recovery from prolonged endurance exercise.

Key words: Endurance-training — Fatigue — Muscle membrane leakage — Physical performance — Muscle enzyme

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Introduction

Physical performance and fatigue both result from the ability or inability, respectively, to generate energy at a rate sufficient to meet muscle fuel requirements. In trained subjects a greater cardiac stroke volume (Ekblom 1969), a higher blood volume (Convertino et al. 1980), an increase in the number and size of mitochondria in skeletal muscle cells, and an increase in mitochondrial oxidative enzyme activity have been reported (Gleser and Vogel 1973; Salminen and Vihko 1984). These characteristics help to improve physical performance and delay fatigue. A comparison between trained and untrained subjects during exercise has revealed lower serum muscle enzyme activity in the former, and this has been related to the higher skeletal muscle cell ATP levels in trained subjects, which prevent membrane leakage and maintain membrane integrity (Saltin and Rowell 1980; Galun and Epstein 1984).

The ability to sustain physical exercise is determined by the ratio between oxygen uptake and the maximal aerobic power at a given work load (Gleser and Vogel 1973). Maximal aerobic power is well reflected by the prediction of peak oxygen uptake attained by ergometric testing (Astrand and Rodhal 1977). However, ergometric tests for real time physical performance after prolonged and heavy exercise are inconvenient for the subjects, sometimes not feasible due to minor injuries and are a logistic problem for research staff under field conditions. In search for an easily obtainable parameter to indicate changes in physical performance, we have investigated the relationship between muscle enzyme activity and changes in peak O_2 uptake during a prolonged march.

Material and methods

41 trained male subjects participated in 3 separate 24 h marches. Fifteen athletes took part in the first march (group A), 8 in the second (group B) and 18 in the third (group C). All participants were matched for weight, age, level of training, and peak O_2 uptake. The mean (±SEM) age, weight and pre-march peak O_2 uptake of all participants were 19.4±0.2 years, 73.4±2.1 kg and 53.2±1.2 ml · kg^{-1} · min^{-1} respectively. Prior to the march, all subjects were medically examined and found to be physically fit. All had performed endurance training for six months prior to the study, which included 6 h and 14 h marches. The subjects were asked to refrain from physical activity for two days preceding the march and gave their informed consent regarding participation in the study.

The three marches were carried out under the same protocol; the route walked was similar and so were climatic conditions. The marches started at 15:00—16:00 hours under comfort conditions of 18.5°±1.0° C dry bulb temperature, which declined during the night to a minimum of 7.0° C and rose during the following day to a maximum of 19.0° C. The subjects carried a backpack load of 27—32 kg and as a group maintained the same walking speed. Check points along the route were located after 9, 16 and 24 h walking (Table 1). The estimated work intensities at the different parts of the route were calculated according to Pandolf et al. (1977), to be 57%, 45%, and 33% of mean peak O_2 uptake. To minimize biochemical change which might occur with cessation of exercise, stops at check points did not exceed 10 min. Ad libitum water was consumed along the march. Light meals (sandwiches, beverages) were offered after blood was drawn.

The enzyme activity of creatine phosphokinase (CPK) and glutamic oxalacetic transaminase (GOT) were measured at intervals as shown in Table 1. Blood was withdrawn from the antecubital vein and collected in lithium-heparin vacutainers. CPK activity (normal range 10—140 u · l^{-1}) was estimated at room temperature by the acetylcystein method. GOT activity (normal range 10—40 u · l^{-1}) was measured according to Henry's method as modified by Kesseler et al. 1975.

Peak oxygen uptake was predicted before the march, and at various time points during the recovery period, as shown in Table 1, by using a bicycle ergometer and the Astrand standard tables (Astrand and Rodhal 1977).

Table 1. Test design of 3 independent groups; timing of blood sampling (CPK, GOT) and ergometric tests (ERG)

Group	Exercise				Recovery				
	pre	9 h	16 h	post	16 h	24 h	40 h	64 h	72 h
A									
CPK	+	+	+	+	+			+	
GOT	+	+	+	+	+			+	
Erg	+				+			+	
B									
CPK	+			+		+			+
GOT	+			+		+			+
Erg	+			+		+			+
C									
CPK									
GOT	+			+			+		
Erg	+			+			+		

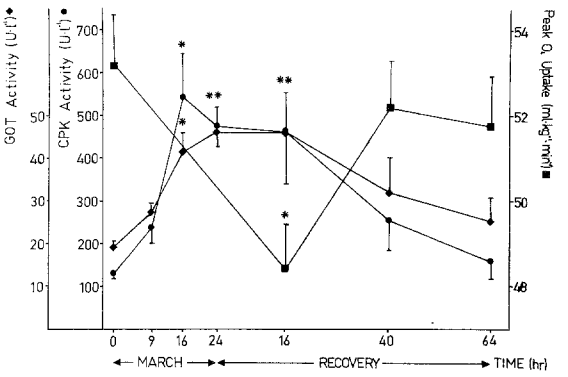

Fig. 1. Mean (±SEM) CPK (●), GOT (◆) activity levels, and peak O_2 uptake values (■) during exercise and recovery (group A)

Statistical analysis was performed using one way analysis of variance. If a significant F value was found ($p<0.05$) critical differences were analyzed by Tukey's procedure to locate the significant mean differences. A Pearson correlation test was used to search for relationships between variables. All values are presented as mean±SEM.

Results

Enzyme activity values in comparison to peak O_2 uptake for group A are presented in Fig. 1. CPK activity reached a maximal value (539.1±105.5 u · l^{-1}) after 16 h of exercise. GOT was also significantly elevated after 16 h and reached a maximal value after 24 h (46.2±3.8 u · l^{-1}) of marching. Enzyme activity values after the first 16 h of recovery did not differ from values at the end of the march nor did they differ from the peak values. After 40 h of recovery CPK and GOT decreased (256.6±71.2 u · l^{-1} and 31.9±7.7 u · l^{-1} respectively) and were not found to be significantly different from pre-exercise values, but still did not return to the normal range. After 64 h of recovery GOT values returned to the normal range (25.6±5.9 u · l^{-1}), while CPK values were still at the upper limit of normal (159.1±44.0 u · l^{-1}).

Peak O_2 uptake was significantly reduced 16 h after the march (48.4±1.1 ml · kg^{-1} · min^{-1}) and returned almost to the baseline value after 64 h of recovery (51.8±1.2 ml · kg^{-1} · min^{-1}). The statistical relationship between changes in peak O_2 uptake and enzyme activity at different time points of marching and recovery are presented in Table 2 (for group A only). A highly significant correlation was also found between CPK and GOT levels along the march ($r=0.896$; $p<0.0001$).

In Fig. 2, the GOT and CPK activity values and peak O_2 uptake of group B are summarized.

Table 2. Statistical correlations between changes in peak O_2 uptake and enzyme activity levels in group A; pre-exercise and during recovery

Time	Enzymes	Peak O_2 uptake	
		r.	*p*
Before march	CPK	−0.410	0.361
	GOT	0.230	0.551
End of march	CPK	−0.552	0.199
	GOT	−0.854	0.007*
16 hours recovery	CPK	−0.769	0.006*
	GOT	−0.606	0.048*
40 hours recovery	CPK	−0.826	0.003*
	GOT	−0.523	0.098
64 hours recovery	CPK	−0.743	0.009*
	GOT	−0.649	0.031*

Peak O_2 uptake indicates the difference between peak O_2 uptake values pre-exercise and after 16 hours recovery
* $p < 0.05$

Peak O_2 uptake (53.2 ± 1.2 ml · kg^{-1} · min^{-1}) which decreased significantly at the end of the march (45.2 ± 1.0 ml · kg^{-1} · min^{-1}), rose slightly 24 h later and then increased significantly at 72 h of recovery to a value similar to that of the pre-march value (54.8 ± 1.2 ml · kg^{-1} · min^{-1}). GOT activity was significantly elevated at the end of the march (98.4 ± 9.1 u · l^{-1}) and at 24 h of recovery, and returned to normal range 72 h after the end of the march (24.2 ± 4.7 u · l^{-1}). CPK activity was elevated at the end of the march (1692.8 ± 389.5 u · l^{-1}), decreased significantly after 24 h recovery (782.5 ± 76.9 u · l^{-1}) and then returned to normal 72 h after the march (185.6 ± 29.5 u · l^{-1}).

Figure 3 demonstrates the relationship between peak O_2 uptake and GOT values in group C. During exercise, both changed simultaneously in opposite directions: peak O_2 uptake decreased 1 h after the end of the march (50.0 ± 1.1 ml · kg^{-1} · min^{-1}) while GOT increased

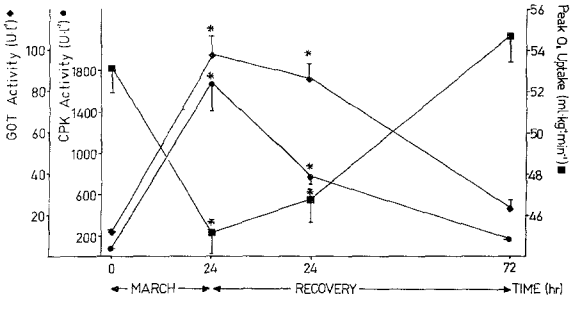

Fig. 2. Mean (±SEM) CPK (●), GOT (◆) activity levels, and peak O_2 uptake values (■) during exercise and recovery (group B)

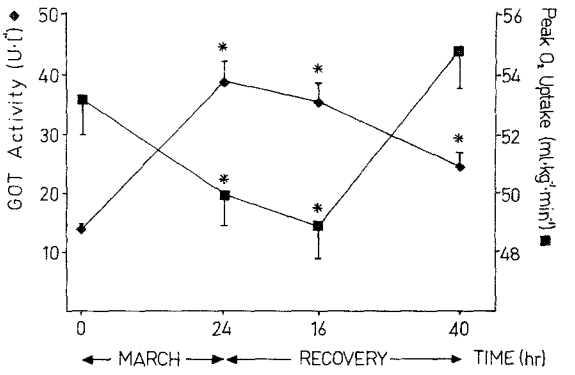

Fig. 3. Mean (±SEM) GOT (◆) activity levels and peak O_2 uptake values (■) during exercise and recovery (group C)

(39.4 ± 3.2 u · l^{-1}). After 40 h of recovery, peak O_2 uptake increased to its pre-exercise value (54.8 ± 1.2 ml · kg^{-1} · min^{-1}) and GOT decreased, but remained elevated as compared to the pre-march value (24.8 ± 1.9 u · l^{-1}).

Discussion

Our findings describe changes in peak O_2 uptake and serum enzymes activity, which in all 3 groups showed a similar "mirror image"-like pattern. During the marches enzyme activity rose while peak O_2 uptake decreased. After the long march, in the early stages of recovery (up to 24 h), enzymes activity decreased non-significantly while peak O_2 uptake increased slowly. During the later stages of recovery (40 h to 72 hours) both enzyme activity levels and peak O_2 uptake returned to pre-march values. This phenomenon occurred in the 3 marches, which were performed independently under similar conditions. However, the absolute values of enzyme activity differed markedly between the groups. Variability in the enzyme response to physical exercise has been previously reported. Within homogeneous groups and between groups of trained individuals, in spite of similar exercise conditions (Reinhart 1982; Apple et al. 1984; Apple et al. 1985).

During exercise, muscle enzyme activity is known to rise and subsequently decline in various patterns after cessation of exercise (Saltin and Rowell 1980; Ross et al. 1983). The changes in enzyme activity level probably reflect muscle membrane leakage (Magazanik et al. 1974) which is minimal until muscle ATP is markedly reduced (Shapiro et al. 1973; Thomson et al. 1975). Serum muscle enzyme activity is affected by the intensity and duration of the exercise performed and by the

fitness level of the subject (Forssell et al. 1975; Sanders and Bloor 1975; Berg and Haralambie 1978; Galun and Epstein 1984). With physical training, muscle ATP and creatine phosphate stores increase and thus assist in maintaining muscle membrane integrity (Karlsson and Saltin 1970). During prolonged exercise, muscle ATP is depleted (Hunter and Critz 1971), the muscle cell membrane leaks and thus enzymes efflux to the blood circulation (Ross et al. 1983). It is therefore suggested that the damage to muscle tissue during exercise causes the apparent decline in physical performance capacity, as indicated in this study by the changes in peak O_2 uptake and muscle enzyme activity (Fig. 1—3). Thus, the limiting factor in physical capacity after prolonged exercise may be the inability of peripheral tissue to utilize oxygen efficiently.

During the first 24 h of recovery there are minimal changes in the level of physical performance as well as in enzymes activity. This period may be referred to as the "fatigue period", where subjects are incapable of maintaining a particular, pre-exercise performance level over time (Edwards 1983). Later, when serum enzymes activity decreases, physical performance level rebuilds and reaches pre-exercise levels. Our results, together with previous findings (Saltin and Rowell 1980; Ross et al. 1983), indicate that the determination of changes in CPK and GOT serum levels at the end of a prolonged exercise may be a simple and reliable tool for predicting the relative reduction in the physiological function of human skeletal muscles, under similar conditions.

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