EFFECT OF SINGLE AND REPEATED EXERCISE ON THE ANAEROBIC THRESHOLD

V. L. Utkin, V. V. Zaitseva,
and N. A. Koganova

UDC 612.015.3-06:766.1

KEY WORDS: exercise; anaerobic threshold.

The anaerobic threshold (AT) corresponds to the load at which products of anaerobic metabolism, especially lactic acid, begin to accumulate in the tissues. It has been shown that systematic exercise raises the AT [2-4]. Changes in AT under the influence of a single physical exertion have not been studied, apart from isolated reports that continuous work more than 2 h in duration by adults lowers AT by 8-10% [13, 15].

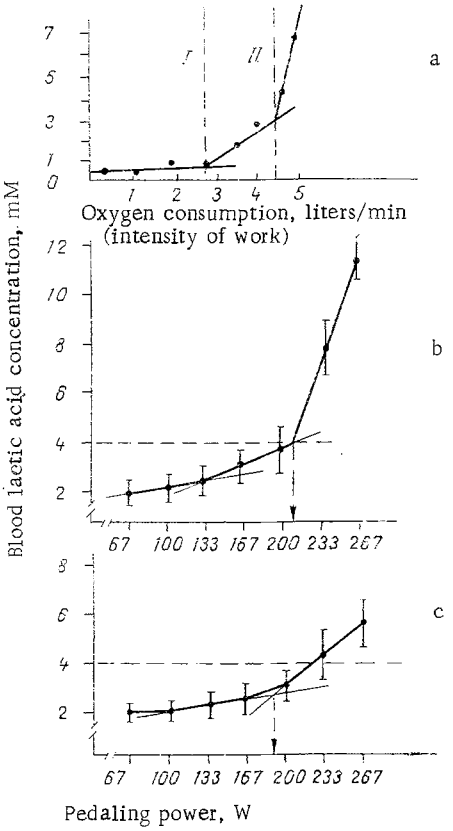

Fig. 1. Blood lactic acid concentration in healthy adult men with an increase in intensity of physical work. a) Scheme of determination of AT: I) aerobic threshold, II) anaerobic threshold; b) before exertion; c) after prolonged, continuous exertion of threshold intensity.

Department of Biomechanics, Central Institute of Physical Culture. Department of Clinical and Experimental Physiology, A. V. Vishnevskii Institute of Surgery, Academy of Medical Sciences of the USSR, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR M. I. Kuzin.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 96, No. 9, pp. 11-14, September, 1983. Original article submitted January 31, 1983.

0007-4888/83/9609-1199$07.50

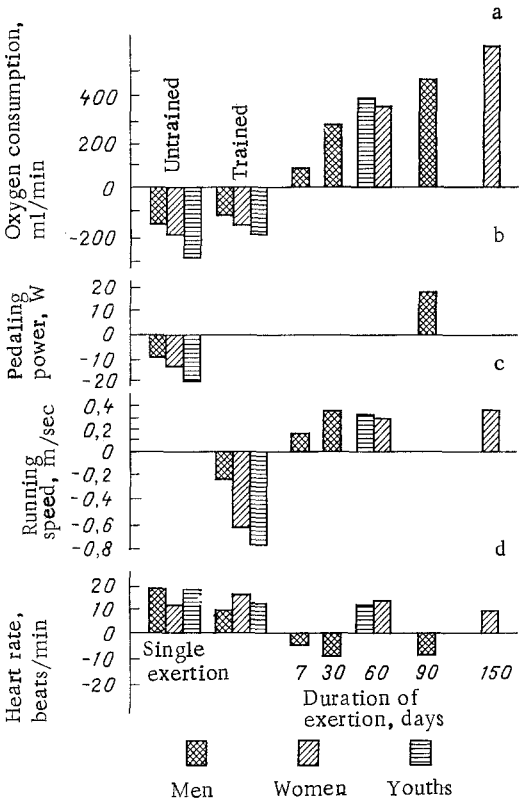

Fig. 2. Changes in parameters corresponding to AT in persons of different ages and sexes during single and repeated exercises. a) Oxygen consumption, b) pedaling power on bicycle ergometer, c) running speed, d) heart rate.

The object of this investigation was to study the effect of two types of exercise on AT: single exertion until fatigue, and systematically repeated exertion with an intensity close to the AT level.

EXPERIMENTAL METHODS

Changes in AT under the influence of the different types of exercise were studied in 37 clinically healthy persons: 12 men aged 18-35 years, 13 youths aged 15-16 years, and 12 girls aged 15-16 years. To determine AT the subjects performed physical exercise of gradually increasing power, graded in the laboratory relative to pedaling on a "Monark" bicycle ergometer, and during running under natural conditions, when it was graded according to the level of tachycardia, programmed by means of a cardiac pacemaker [1, 7]. At each degree of exertion the respiratory minute volume and oxygen consumption were recorded by a gas analyzer (Beckman, USA), and the heart rate was monitored. The running speed was measured under natural conditions and air for analysis was collected in a Douglas bag. Blood for analysis was taken from the terminal phalanx of a finger before and at each level of exertion. The lactic acid concentration was determined by the method in [12]. The AT point [8, 11] was determined graphically (Fig. 1a). The widely used method of determining AT at the 4 mM level [3, 8] was rejected in favor of the graphic method in order to avoid mistakes arising during determination of the AT level after physical exertion. The error is due to the fact that prolonged exercise (to fatigue) leads to a decrease in the absolute blood lactate levels (Fig. 1b, c). This fact, in turn, is connected with exhaustion of glycogen reserves in the muscle fibers [6, 10]; the initial reserves are not restored before 24 h after the end of work [9].

Single exercise lasted 1.5-2 h before signs of uncompensated fatigue appeared. AT was determined before and after exertion; the rest after the second AT determination was 15 min in duration.

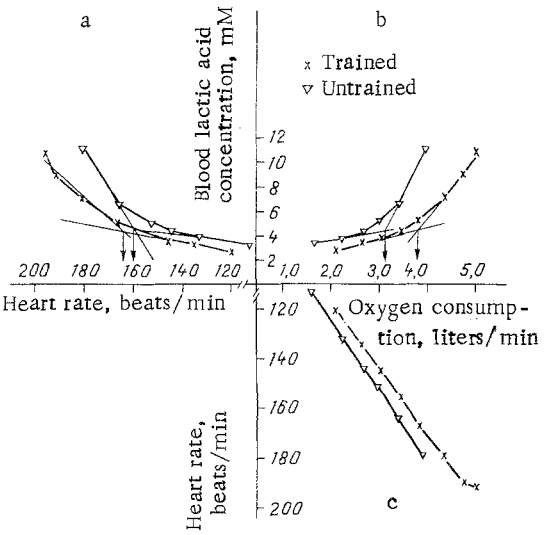

Fig. 3. Correlation between oxygen consumption, heart rate, and blood lactic acid concentration in persons with different levels of physical training during exercise.

The duration of systematic exercise was 5 months. The AT level was determined before the beginning of the investigation, 7 days later, and thereafter regularly at intervals of 30 days.

EXPERIMENTAL RESULTS

Data showing changes in AT relative to the initial level under the influence of exercise under various conditions are given in Fig. 2. Single physical exercise caused a statistically significant fall in AT ($P < 0.01$) in all subjects. Values of the oxygen consumption, pedaling power, and running speed fell in both adults and youths to correspond to the AT level. Some differences were observed between groups of subjects of the same sex and age, but differing in their level of physical training: in better trained subjects significantly smaller changes were observed in AT.

Systematic exercises for a period of 7 days did not cause a statistically significant fall in the AT level (Fig. 2). Under the influence of systematic exercises for a period of 30 days or more, an increase in AT relative to the initial level was observed in all subjects according to values of the oxygen consumption, pedaling power, and running speed. This result agrees with data in the literature [2-5]. Changes in the same direction were observed by Wasserman et al. [14], who consider that AT is the first increase in lactic acid concentration compared with its resting level. During systematic exercises both limits of the aerobic-anaerobic transition [11] are evidently displaced toward higher loads.

Under the influence of systematic exercises the threshold level of tachycardia, i.e., the heart rate corresponding to AT, changed. However, the character of these changes differed to some extent from the dynamics of the threshold level of oxygen consumption (Fig. 2a, d). Systematic exercises by untrained people led only to a small increase in the threshold level of tachycardia. The reason is that simultaneously with a change in dependence of the blood lactate concentration on oxygen consumption, dependence of the heart rate on oxygen consumption also changed (Fig. 3b, c).

The threshold level of tachycardia becomes an informative parameter of physical working capacity only in physically trained subjects, when continued exercise affects the dependence of the blood lactate concentration on oxygen consumption, but virtually no longer causes any change in the dependence of heart rate on oxygen consumption. According to Eckblom [4], this dependence begins to be manifested not later than 32 months after the beginning of regular exercises.

The results described above shed some light on the character of relations between oxidative phosphorylation and glycolysis in the energy supply for muscular work. They enable AT to be regarded as the level of work at which synergism between the above mechanisms of energy provision begins to be manifested. Immediately after a single physical exertion up to the fatigue level, the aerobic capacity falls [2] because the glycolytic source of energy is activated by work of lower intensity than in the unfatigued state. It must be pointed out that early activation of the lactate mechanism takes place despite the fall in the muscle glycogen level, and it is manifested as a decrease in the absolute lactate concentration in the peripheral blood (Fig. 1c).

Systematic exercises with an intensity close to AT, on the other hand, contribute to the development of aerobic capacity [24]. It can be tentatively suggested that this is why the level of loading up to which the energy demand for exercise can be satisfied mainly aerobically is raised.

Training for 7 days had no appreciable training effect, and therefore did not change the ratio between the contributions of oxidative phosphorylation and glycolysis to the supplying of energy for muscular work.

Prolonged (until fatigue) exercise thus causes AT to fall below its initial level. Systematic exercise for 7 days does not cause any statistically significant change in AT. Systematic exercises for 30 days or more significantly increase AT. The relationships discovered for the change in AT are evidence that under the influence of physical exertion the contributions of oxidative phosphorylation and glycolysis to the supplying of energy for muscular work are changed: the higher the aerobic capacity of the body, the higher the absolute and relative (as a percentage of maximal oxygen consumption) levels of loading at which products of anerobic metabolism begin to accumulate.

These results can be used to monitor physical working capacity and also to model motor activity. Under these circumstances the use of the threshold level of tachycardia to monitor physical working capacity is valid only for physically trained subjects.

LITERATURE CITED

1. V. D. Chepik and V. L. Utkin, Teor. Prakt. Fiz. Kul't., No. 11, 8 (1969).
2. P. O. Astrand and K. Rodahl, Textbook of Work Physiology, New York (1977).
3. J. A. Davis, N. H. Frank, B. J. Whipp, et al., J. Appl. Physiol., 46, 1039 (1979).
4. B. Eckblom, Acta Physiol. Scand., Suppl. No. 328, 9 (1969).
5. G. W. Heath, J. M. Hagberg, A. A. Ehsani, et al., J. Appl. Physiol., 52, 634 (1981).
6. I. Jacobs, P. Kaiser, and P. Tesch, Europ. J. Appl. Physiol., 46, 47 (1981).
7. V. L. Karpman, A. F. Sinyakow, B. G. Lyubina, et al., Med. Sport, No. 12, 374 (1972).
8. W. Kindermann, G. Simon, and J. Keul, Europ. J. Appl. Physiol., 42, 25 (1979).
9. J. D. MacDougall, G. R. Ward, D. G. Sale, et al., J. Appl. Physiol., 42, 129 (1977).
10. S. S. Segal and G. A. Brooks, J. Appl. Physiol., 47, 514 (1979).
11. J. S. Skinner and T. H. McLellan, Res. Quart. Exercise Sport, 51, 234.
12. G. Strom, Acta Physiol. Scand., 17, 440 (1949).
13. A. A. Sucec, W. H. Phillips, and D. H. Selder, Med. Sci. Sports, 12, 127 (1980).
14. K. Wasserman, J. Whipp, S. N. Koyal, et al., J. Appl. Physiol., 35, 236 (1973).
15. R. A. Wiswell, R. H. Girandola, R. Bulbian, et al., Med. Sci. Sports, 12, 86 (1980).