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Analysis of Changes in the Pulse Rate during Exercise and Recovery after Sixty Hours of Complete Fasting

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Summary. In a group of seven men the increase of the pulse rate at the onset of standard exercise on a bicycle ergometer and the drop during recovery were assessed, during the control period and after 60 h fasting. Analysis into a rapid (vagus, central) component of the pulse increment and a slow (peripheral, sympathetic) component was made and their velocity constants were obtained. Fasting lead to a rise of the pulse increment and an increase of the sympathetic component at the expense of the component of vagus inhibition. These results confirm that acute fasting leads to a drop of physical performance, as under normal conditions analogous changes are caused by more intense exercise.

Acute fasting is one of the factors which exert a marked effect on a number of metabolic processes in the organism. This must be reflected also in changes of the physical performance of man. As one of the indicators of physical performance changes in the pulse rate are frequently used, either in conjunction with the simple step test or as part of the evaluation of results of spiroergometric examinations, etc. [1, 2]. In particular the close linear correlation between oxygen consumption and pulse rate is important [2], as it renders the investigation of the pulse rate more valuable though the latter represents only one aspect of the complex circulatory response to the increased metabolic demands of the working muscles [1, 3].

The biphasic course of changes in the pulse rate at the beginning of exercise and during recovery with a rapid component followed by a second slow one [3] renders mathematical analysis into two exponential processes with markedly different half-lives possible. The rapid component is apparently due to vagus inhibition and the slow component is controlled by the sympathetic nerve. This analysis renders it at the same time possible to evaluate some vegetative regulations associated with exercise [4].

Acute fasting has a marked effect on circulation already at rest. Therefore such changes can be expected even more during physical exercise. An account of their investigation as regards the pulse rate and its regulation is the subject of the present communication.

Material and Methods

The trial was made in seven healthy normally nourished men (average age 23.83 ± 3.12 years, weight index according to VERDANCK [12] 102.82 ± 3.40). Six volunteers were students, one was a doctor.

Complete fasting lasted 60 h. The experimental subjects received during this period only water and unsweetened tea. The fluid intake was not limited.

Each experimental subject was examined after previous practical instruction twice on an Elema bicycle ergometer, in the morning on fasting at the onset of the experiment (i.e. after 12 h fasting) and for the second time at the same time of the day after 60 h fasting. The load, 1.5 watt per kg body-weight, was applied for 6 min. The pulse rate was recorded by means of a cardiotachometer Prema and transmitted to a kymograph 5 min prior to the experiment, during exercise and during the first 10 min of recovery. Several other parameters which were investigated are not the subject of the present communication. In view of the medium grade load, in all instances not later than in the last 2 min of the experiment a steady state was attained.

The analysis of the pulse rate curves is based on expressing their course at the onset of exercise and during recovery as the sum of two exponentials with a short and longer half-life [4].

For the period of exercise we can express it as follows

$$\alpha(t) = \alpha_1 (1 - e^{-k_1 t}) + \alpha_2 (1 - e^{-k_2 t}) \text{ for } t\varepsilon <t_0, t_S> \qquad (1)$$

where $\alpha(t)$ = the increment of pulse rate as compared with the value at rest at time t,

$\alpha_{1,2}$ = the rapid [1] and slow [2] component of the pulse increment during exercise,

$k_{1,2}$ = the velocity constants of the rapid and slow component ($k_1 > k_2$),

t_0 = the onset of exercise,

t_S = the moment when the steady state was reached or the end of exercise, if the steady state is not reached.

During recovery

$$\alpha(t) = \alpha_1' e^{-k_1' t} + \alpha_2' e^{-k_2' t} \text{ for } t\varepsilon <t_R, \infty) \qquad (2)$$

$\alpha'_{1,2}$ = the rapid and slow component of the pulse increment during recovery,

$k'_{1,2}$ = the velocity constant of the rapid and slow component ($k'_1 > k'_2$),

t_R = the end of exercise and onset of recovery.

The following relations also apply

$$\alpha_0 \leqq \alpha_1 + \alpha_2$$
$$\alpha_0 = \alpha_1' + \alpha_2' \qquad (3)$$

where α_0 = the increment of the pulse rate as compared with the initial value in the steady state or at the end of exercise if the steady state is not attained. In this case, of course, in equation three only the unequality applies and the accuracy of the parameters *for the time of exercise* by means of simple graphic analysis declines considerably.

Analogously as in oxygen consumption, we described the area below the curve of pulse increments during recovery as the pulse debt (pd) which can be divided into the area beneath both exponentials, i.e. the rapid component of the pulse debt (pd_1) and the slow component (pd_2). The pulse debt and its components are calculated by integration of equation 2 within the range of t_R to ∞ and thus the following relationship apply

$$pd_1 = \frac{\alpha_1'}{k_1'}, \quad pd_2 = \frac{\alpha_2'}{k_2'}, \quad pd = pd_1 + pd_2. \qquad (4)$$

The illustration of all above relationships and the method how the parameters are obtained by the graphic method are given in Fig. 1.

Results

60 h fasting led in the group of seven men to a rise of the pulse rate at rest from 74.6 ± 10.5 to 86.8 ± 19.6 p/min. This difference in the paired t-test is statistically significant ($t = 3.2261$, $p < 0.02$).

Changes in the pulse rate as compared with the value at rest in the control period and during fasting expressed as average values for the whole group are given in Fig. 2. The results of the analysis of pulse rate increments during exercise and at the onset of recovery are recorded in the Table.

The maximum increment of pulse rate (α_0) during fasting rises on an average only by 5.2 p/min, the difference is, however, significant.

Analysis into exponential components at the onset of exercise indicates that while during the control period the rapid component of the pulse increment (α_1) predominates markedly over the slow component (α_2), during fasting this relationship shifts in favour of the slow component. This is particularly marked when expressed in per cent (Table). These relations for average values of the pulse increment and its components are illustrated in Fig. 3.

The mutual ratio of the two components of the pulse increment during recovery (α_1', α_2') and their changes are analogous.

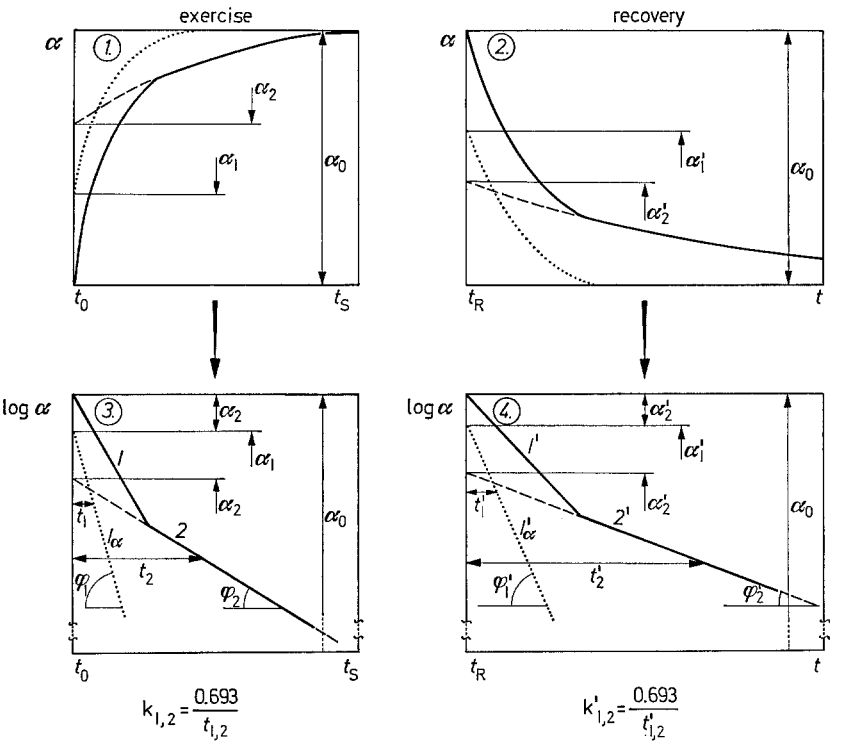

Fig. 1. Shape of curve of pulse increment at onset of exercise [1] and during recovery [2] and their graphic analysis in semilogarithmic coordinates [3, 4]. t = time, t_0 = onset of exercise, t_S = moment when steady state is attained, t_R = onset of recovery. α = pulse increment, α_0 = pulse increment during steady state. $\alpha_{1,2}$ $\alpha'_{1,2}$ = rapid and slow component of pulse increment at onset of exercise and during recovery. k_1, k_2, k'_1, k'_2 = appropriate velocity constants obtained by calculation from half-lives $t_{1,2}$ and $t'_{1,2}$. *1* = steep part of curve. *2* = slow part of curve corresponding to slow exponential. *1a* = rapid component obtained by subtracting extrapolated part 2 from 1, $\varphi_{1,2}$, $\varphi'_{1,2}$ = corresponding angles with time axis

Analysis of the velocity constants of the two exponentials revealed in particular the following facts:

a) the velocity constants of the rapid component are many times greater than the constants of the slow component [4];

b) the velocity constants of the rapid and slow component are markedly higher at the beginning of exercise than during recovery [4];

c) fasting leads in general to a rise of all velocity constants.

The difference in the rapid component is relatively small (in k_1 it is insignificant), in the slow components (k_2, k_2') the increase is much more marked.

Table. *Parameters of curves of pulse increment — average values ($\bar{x}$) and standard deviations (s)*

n = 7		Exercise					Recovery						
		α_0 (p/min)	α_1 (p/min)	α_2 (p/min)	k_1 (min^{-1})	k_2 (min^{-1})	α'_1 (p/min)	α'_2 (p/min)	k'_1 (min^{-1})	k'_2 (min^{-1})	pd_1 (p)	pd_2 (p)	pd (p)
Control period	$\bar{x}$	61.2	45.1	16.1	5.92	0.35	48.5	12.7	0.62	0.04	81.3	235.4	316.7
	s	4.9	3.5	2.9	0.39	0.05	5.7	5.4	0.34	0.02	18.7	123.0	109.9
	(100%)		(73.69% ±5.72)	(26.31% ±4.72)			(79.24% ±9.31)	(20.76% ±8.82)					
Fasting period	$\bar{x}$	66.7	29.2	37.5	6.72	0.64	31.4	35.3	0.71	0.14	45.8	274.4	320.1
	s	5.5	7.8	8.3	0.42	0.08	7.6	10.1	0.10	0.03	16.0	119.6	112.3
	(100%)		(43.77% ±11.69)	(56.23% ±12.44)			(47.07% ±11.39)	(52.93% ±15.14)					
Statistical evaluation of differences (paired t-test)	t	3.3150	7.3791	5.9382	6.5733	7.6853	6.7986	5.8771		3.9886	4.7703		
	p	<0.05	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	—	<0.01	<0.01	—	—

t = tested criterium, p = level of significance, α_0 = total pulse increment in the steady stade as compared with initial value, α_1, α_2 = rapid and slow component of pulse increment during exercise, k_1, k_2 = velocity constants of the rapid and slow component during exercise, α'_1, α'_2 = rapid and slow component of pulse increment during recovery, k'_1, k'_2 = velocity constant of rapid and slow component during recovery, p = pulse, pd = pulse debt, $pd_{1,2}$ = rapid and slow component of pulse debt.

The pulse debt (pd) increases during fasting only insignificantly, mainly in its slow component (pd_2). The rapid component of the pulse debt (pd_1), on the other hand, declines significantly during fasting.

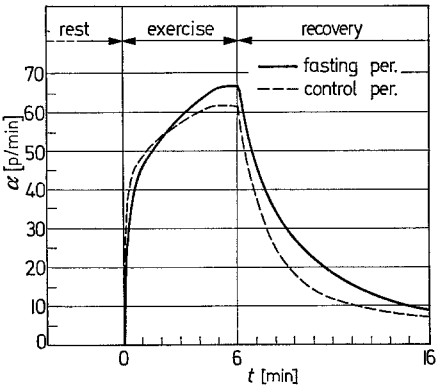

Fig. 2. Course of average curves of pulse increment for entire group during exercise and recovery in control period and during fasting. α = pulse increment as compared with value at rest

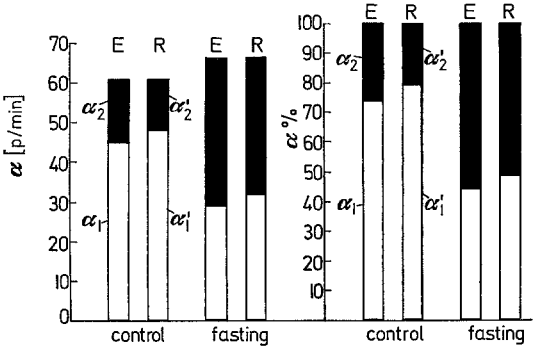

Fig. 3. Analysis of average pulse increment (α) during control period and during fasting. During exercise (E) rapid component = α_1, slow component = α_2. During recovery (R) rapid component = α'_1, the slow component = α'_2

Discussion

The biphasic course of the rising pulse rate at the onset of exercise and the return to the value at rest during recovery is a fact which has been known for a long time [3, 7]. This type of adaptation to conditions of exercise and recovery seem to be a more general biological mechanism, as in an analogous manner also changes in ventilation [10] and oxygen consumption [6, 11] can be analyzed.

The rapid rise of the pulse rate at the onset of exercise is probably due to vagus inhibition which originates in the motor area of the cerebral cortex. That a vagus reflex is involved is suggested in particular by:

a) the short period of latency of the reflex typical for vagus reflexes [3];

b) the shortening of the cardiac cycle at the onset of exercise at the expense of the diastole which is typical for a reduction of the vagual tonus [7];

c) after removal of the ganglion stellatum the initial increase in rate is maintained, but it disappears after vagotomy [3], after atropine and during a high temperature [9].

In the second stage of acceleration the systole also becomes shorter [3]. Accelerators are a safety factor which starts to function only during more intense exercise when also humoral factors participate.

The quantitative participation of both components and their mutual relationship can be ascertained by means of graphic analysis [4] or by processing on a computer.

The thus obtained rapid component of the increment and its velocity constant can thus be described as the central vagus component and the slow component as the peripheral or sympathetic one. In our previous work [4] we demonstrated that the participation of the slow component increases with the rising load and that, the load being equal, it is greater in women, i.e. a physically less efficient group than in men of similar age. It seems thus the greater the pulse increment during the steady state or at the end of exercise, and the greater the ratio of the slow component (peripheral, sympathetic) in an individual or group on an equal load the smaller the physical efficiency and thus the peripheral safety regulation in the adaptation of circulation must play a greater role.

Acute fasting for 60 h lead to marked physical and mental fatigue of the experimental subjects. They displayed orthostatic changes and an acceleration of the pulse rate at rest. In addition to the increased catabolism of fats and body proteins fasting leads also to considerable losses of electrolytes and body fluids and the plasma volume declines [8]. This all leads to a deterioration of the tolerance of physical exercise [5].

As regards the pulse rate, the pulse increment rises in the steady state though the basal pulse rate is already increased. The analysis proper of the course of the pulse rate curves indicates that the participation of the slow components of the pulse increment rises absolutely and relatively at the onset of exercise and during recovery. These changes are statistically significant.

This shift from vagus inhibition towards sympathetic regulation confirms the reduction of physical fitness as a result of fasting and is in keeping with the stress situation which acts on the organism.

The velocity constants of the rapid vagus component are not very much influenced by fasting, contrary to the marked rise of both constants of the slow component. This rise involves a more rapid return to values at rest during recovery. This could be erroneously interpreted as increased performance. It is, however, a fairly generally valid rule that considerably altered values of a certain parameter have a tendency to change more rapidly under the influence of a stimulus (so-called law of initial values [13]). This applies probably also to constants of the considerably greater slow, sympathetic component during fasting. The more rapid rise of the slow component at the onset of exercise suggests at the same time also an increased sympathetic tonus.

The average magnitude of the pulse debt increases only slightly during fasting. This small increment is, however, solely due to the rise of the slow sympathetic component of the debt, the participation of which during the control period amounts to 74.33% of the total pulse debt and 85.69% during fasting. This distribution can therefore be also considered as evidence of the reduced efficiency of circulation during fasting.

The results thus indicate that graphic analysis or analysis by computers of changes in the pulse rate can also under conditions of stress serve as a further parameter for evaluating changes of physical performance of man.

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