MECHANISMS OF ADAPTATION TO PHYSICAL EXERTION AND EFFECT OF CO_2 EXCESS ON THEIR FORMATION

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Over a moderate range of concentrations carbon dioxide (CO_2) contributes to the formation of emergency [5] and long-term adaptive reactions to hypoxia [8]. In the latter case CO_2 quickens the appearance of positive changes in mechanisms of regulation of the circulation [8]. The hypoxid factor plays an important role in adaptation of the body to extremal physical exertion [1].

The aim of this investigation was to study the mechanisms of action of CO_2 on the development of adaptation to physical exertion and the possibility of using this gas to optimize the adaptation process.

METHODS

Male albino rats weighing 180 g were compelled to swim in aquaria carrying a load equal to 7.5% of their body weight, in water at a temperature of 32°C, daily 6 times a week for 3 months. During the introductory week the animals swam without a load, and under these circumstances the duration of their stay in the water was increased daily from 5 to 30 min. After the introductory week and every month of training, the animals' maximal working capacity was determined from the duration of swimming with the load until they could no longer stay on the water surface. During the first month the duration of swimming was increased every week from 10 to 40% of the working capacity determined previously, during the second month it was increased from 30 to 60%, and during the 3rd month it was maintained at the level of 50-60%. Animals of group 1 were trained in a normocapnic atmosphere (0.7 ± 0.04% CO_2), those of group 2 in a hypercapnic atmosphere (3.9 ± 0.08% CO_2). Rats of group 3 were kept in the same hypercapnic atmosphere without physical exertion for 30 min, 6 times a week for 3 months, and group 4 consisted of intact animals. Carbon dioxide in the necessary concentration was supplied beneath the Plexiglas lids of the aquaria, and the gaseous composition with respect to O_2 and CO_2 was monitored continuously during each session (Beckman OM-11 gas analyzer, USA). The animals were investigated after 1, 2, and 3 months of training, 20-24 h after the final swim. Under urethane anesthesia (1.5 g/kg) the systolic pressure (SP) in the left ventricle (LV), the degree of its maximal rise and fall ($\pm SP_{max}$) during occlusion of the aorta for 30 sec, and the heart rate (HR) were determined. To assess sympathetic and parasympathetic influences, the adrenergic and cholinergic reactivity of the myocardium was determined from the change in SP after injection of noradrenalin (NA) in a dose of 5 μg/kg and the change in HR following injection of acetylcholine (ACh) in a dose of 20 μg/kg into the left external jugular vein. The NA concentration in the tissue of LV of the heart [6] and the ACh level in the atrial tissue [10] were recorded. The ability of the heart to maintain the reserves of these mediators during arduous swimming under conditions of maximal working capacity, found during the introductory period of training, was determined.

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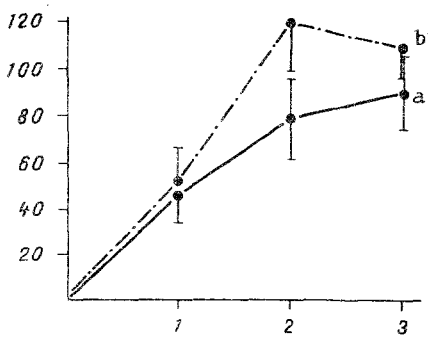

Fig. 1. Changes in maximal working capacity of rats during training (M ± mt). Abscissa, duration of training (in months); ordinate, increase in working capacity (+ΔT, per cent compared with introductory period). a) Training in ordinary atmosphere, b) training in atmosphere with excess of CO_2.

RESULTS

Addition of CO_2 to the normoxic atmosphere was reflected significnatly in the working capacity of the rats during the period of training to swim. It will be clear from Fig. 1 that the increase in maximal working capacity during two months of training under hypercapnic conditions was significnatly greater than in normocapnia. With an increase in the duration of training under hypercapnic conditions to three months, the rats' working capacity no longer increased, as it did during normocapnia, but decreased somewhat compared with the previous period of investigation. Under hypercapnic conditions, the active factors were probably molecular CO_2 and HCO_3^-, for pCO_2 and the HCO_3^- level in plasma from mixed (arteriovenous) blood, taken from the caudal vessels of the rats immediately after a training session were higher than the corresponding values under normocapnic conditions with no change in pH. Meanwhile, the hypercapnic conditions, the degree of increase in pCO_2 and the HCO_3^- concentration in the blood plasma fell progressively from the first until the third month of training) this may have been the result of activation of the mechanisms of their binding and utilization. As the intracellular pCO_2 rises, carboxylation of pyruvate is intensified and acid cycle (TAC) is increased, with the result that utilization of lactate is increased and the lactacidemia is abolished [4, 11]. In fact, in the present experiments the degree of lowering of BE became steadily less marked from the first to the third months of training under the influence of hypercapnia.

After training under normocapnic conditions the ability of the myocardium of LV to maintain SP_{max} during isometric contraction was increased only after 3 months, but under hypercapnic conditions, it was increased as early as after 2 months (Table 1). After 3 months this parameter remained at the same raised level. The increase in myocardial contracility could be due to its more rapid hypertrophy [2]. During training under normocapnic conditions it was 3 months before the weight of LV became statistically significantly increased, whereas under hypercapnic conditions this occurred after only 2 months, and the increase continued until the 3rd month.

After 2 months, before the increase in myocardial contractility and its hypertropy, training with physical exertion under normocapnic conditions lowered HR and increased the sensitivity of the cardiac pacemaker to ACh. By the 3rd month the degree of these changes increased, the ACh concentration in the rats' atria rose, and the ability of the heart to maintain the ACh reserves in its tissues during arduous physical exertion also was enhanced. After 2 months of training under hypercapnic conditions the bradycardia became more marked, the sensitivity of the cardiac pacemaker to ACh increased even more, the ACh level in the atrial tissue rose, and the stability of its reserves in the atrial tissue did not increase progressively, as during normocapnia, but fell below levels observed at the previous time of investigation.

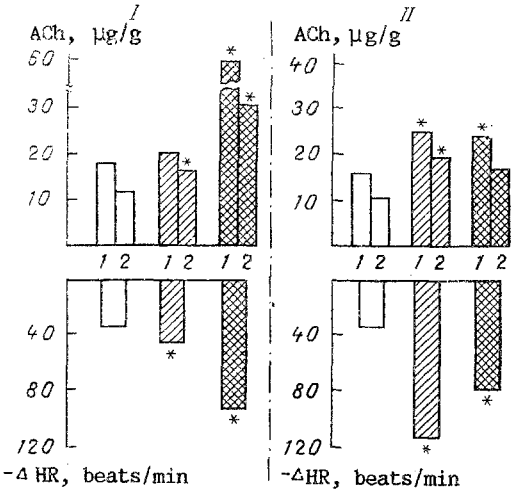

Fig. 2

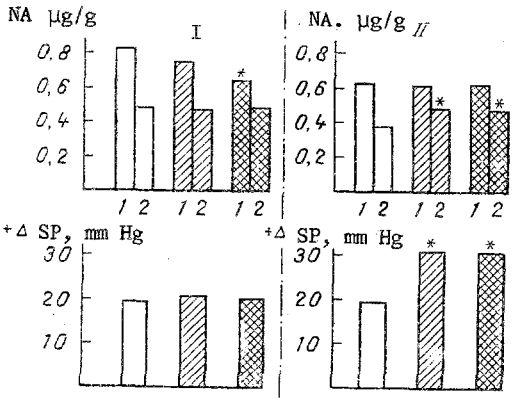

Fig. 3

Fig. 2. ACh concentration (in μg/g) in atrial tissue and degree of fall of HR (−ΔHR, beats/min) under the influence of ACh, in untrained rats and rats trained for 2 (I) and 3 (II) months. 1) ACh concentration before physical work, 2) the same, after physical work. Unshaded columns, untrained animals; obliquely shaded, rats trained in ordinary atmosphere; cross-hatched, rats trained in excess of CO_2. *P < 0.05 compared with untrained animals.

Fig. 3. NA concentration (in μg/g) in tissue of LV and degree of increase in SP (+ΔSP, mm Hg) under the influence of NA in untrained rats and in rats trained for 2 (I) and 3 (II) months. 1) NA concentration before physical work, 2) the same, after physical work. Remainder of legend as to Fig. 2.

TABLE 1. Effect of Excess CO_2 in Atmosphere on Some Parameters of Cardiac Activity of Rats during Training to Swim

CO_2 concentration, %	Duration of training, months	HR at rest, beats/min	Initial SP in LV, mm Hg	SP_{max} on occlusion of aorta	SP_{max} after occlusion of aorta for 30 sec	Degree of fall of SP_{max} by 30th second of occlusion of aorta
Control	—	363,1±16,8 (27)	88,8±2,2 (29)	+82,6±6,7 (29)	+64,0±2,7 (29)	−18,6±2,7 (29)
0,7±0,04 (ordinary atmosphere)	1	370,4±20,5 (10)	89,4±3,8 (8)	+81,8±5,7 (8)	+67,6±3,6 (8)	−14,2±3,6 (8)
	2	357,6±12,3 (11)	82,9±3,7 (10)	+88,0±5,4 (10)	+74,4±3,2 (10)	−13,6±3,3 (10)
	3	308,5±13,0* (8)	85,2±3,2 (6)	+83,3±8,1 (6)	+73,7±3,4 (6)	−9,6±3,4* (6)
3,9±0,08 (excess CO_2 in atmosphere)	1	369,6±19,6 (11)	93,5±4,3 (8)	+79,7±9,7 (8)	+63,2±3,8 (8)	−16,5±3,8 (8)
	2	324,7±14,8* (11)	91,0±2,0 (10)	+83,0±3,6 (10)	+75,1±1,9 (10)	−7,9±1,8* (10)
	3	300,8±28,9* (8)	83,0±3,9 (6)	+85,8±10,1 (6)	+74,6±2,9 (6)	−11,2±2,8* (6)

Legend. *P ± 0.05 compared with control. Number of observations given in parentheses.

Under normocapnic conditions, after 3 months of training the stability of the NA reserves in the tissues of LV increased during arduous physical exertion, with no change in the background level, and its positive intotropic effect was potentiated. Under hypercapnic conditions the background NA level in the tissue of LV fell after 2 months, but the stability of the NA reserves increased during arduous physical work. After 3 months the changes in the NA concentration indicated above remained the same, but an increase in sensitivity of the myocardium of LV to NA was recorded (Figs. 2 and 3).

Addition of CO_2 in a concentration of 4% to a normoxic atmosphere thus leads to a more rapid and considerable increase in the animals' working capacity and also to an increase in functional capacity of the myocardium. This may be because CO_2 and HCO_3^- potentiate

the excitatory effects of physical work on the circulation and also, perhaps, optimize the concentration between the respiratory and hemodynamic systems at the level of the lungs [5, 7]. Hypercapnia helps to maintain the reserves of mediators in the heart and to increase the efficiency of their action on myocardial function, thereby ensuring greater ease of its control by excitatory and inhibitory influences during and after work. With an increase in the duration of training the positive action of hypercapnia on the adaptation process is lost. Positive changes in working capacity and in the mechanisms of regulation of theheart begin to disappear. A similar phenomenon was found by the writers during training of rats in the presence of O_2 deficiency, during hypercapnia of the same degree [8]. We know that during training the ventilation and circulatory responses to CO_2 are weakened because of a decrease in sensitivity of the specialized chemoreceptors [3] and diminution of the degree of rise of pCO_2 and of the HCO_3^- level, as a result of their increased utilization by the tissues. Weakening of the warning action and strengthening of the metabolic action of hypercapnia are accompanied by a fall in the ACh concentration and a decrease in the stability of its reserves in the heart, and also in the sensitivity of the myocardium to ACh. An increase in carboxylation of pyruvate in cholinergic neurons leads to a decrease in acetyl-CoA formation from it and to weakening of ACh biosynthesis [9]. HCO_3^- anions inhibit the pentose cycle, as a result of which the passage of K^+ into the intracellular space is reduced and the hyperpolarizing effect of ACh on cells of the cardiac pacemaker is weakened [4]. On the whole conditions for reducing parasympathetic influences are created, although the properties of the sympathetic mechanism of regulation of the heart and the functional capacity and mass of the myocardium are preserved just as at the height of training. These regulatory changes probably lead to inappropriately prolonged preservation of cardioexcitatory influences after the end of the training period, and this may cause restriction of working capacity. In moderate concentrations the carbon dioxide may help to quicken the development of adaptation to physical exertion, but only in the early stages of training.

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