Development of Ischemic Contracture in Isolated Skeletal Muscles: Relation to the Capacity for Anaerobic Glycolysis

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Abstract In order to evaluate the dependence of the rate of ischemic contracture development in isolated skeletal muscles on the capacity for anaerobic glycolysis, factors affecting ischemic contracture development were examined using rat soleus and quadriceps muscles. The contracture developed faster in slow-twitch oxidative (SO) fibers than in fast-twitch glycolytic (FG) fibers. In both types of fibers, the rate of contracture development was inversely related to the medium pH. Addition of lactate increased the rate of contracture development. In FG fibers, the lactate level during contracture was directly, and the glycogen level inversely, proportional to the medium pH. An increase in muscle lactate level by 4-h swimming accelerated the contracture development, and reduced further lactate accumulation during contracture. Types of exercise training known to influence the capacity for anaerobic glycolysis (interval training and repeated fast climbing and swimming) decreased the velocity of contracture development. Isoprenaline administration *in vivo* accelerated contracture development in association with reduced lactate level during contracture. Swimming training counterbalanced the action of isoprenaline. Adrenalectomy accelerated contracture development in SO fibers, and decreased lactate accumulation during contracture in FG fibers. In adrenalectomized rats, the effects of exercise were more pronounced in SO fibers than in FG fibers. These results indicate that the rate of contracture development in isolated skeletal muscle is inversely related to the capacity for anaerobic glycolysis.

Key words: anaerobic glycolysis, contracture, exercise, skeletal muscle.

It has been known that a muscle exhibits contracture when its blood supply is blocked (ischemic contracture). The ischemic contracture has been studied mostly on the myocardium [1–6], and it has been found that the magnitude of ischemic contracture in the myocardium depends on increased myoplasmic Ca^{2+} contraction associated with decreased level of ATP [3, 7]. In turn, the above situations are dependent on the capacity for anaerobic glycolysis in the tissue [3, 6, 7]. Thus, it

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has been suggested that the magnitude of myocardial ischemic contracture is a gross indicator of early irreversible ischemic injury [5]. On the other hand, the development of ischemic contracture in skeletal muscle has been used for studies of energy metabolism in ischemic conditions [8, 9]. The present study was undertaken to study the influence of various factors on the development of ischemic contracture in skeletal muscle with special reference to the capacity for anaerobic glycolysis. The factors studied include changes in the medium pH, addition of lactate to the medium, acute exercise, exercise training, administration of catecholamines, and adrenocortical insufficiency.

MATERIALS AND METHODS

Male Wistar rats (body weight, 200–250 g) were used. All animal experiments were carried out in accordance with generally accepted rules for the care and use of laboratory animals. The rats were kept in standard conditions of vivarium and standard laboratory diet. They were allowed free access to drinking water and food. The lighting schedule was 12 h light + 12 h dark. In group of rats, bilateral adrenalectomy was performed by the dorsal route. Adrenalectomized rats were maintained with 1% of NaCl in the drinking water.

In exercise experiments, swimming in water at $32 \pm 1^\circ\text{C}$ (4 or 8 h for control rats and 4 h for adrenalectomized rats) was used. In the swimming tank, the mean water surface for each rat was 80 cm^2 . The limited water surface area enhanced the swimming activity of the rats. Rats were sacrificed immediately or 4 h after exercise.

Five types of training experiments were performed. Rats of all training groups exercised 5 days a week. Three groups were trained with running on a motor-driven treadmill with an incline of 8° . Models of sprint training (running velocity $80\text{ m}\cdot\text{min}^{-1}$; initially two 30-s bouts, from the 6th week 12 bouts; rest interval between bouts, 2.5 min), interval training (running velocity $40\text{ m}\cdot\text{min}^{-1}$, duration of running bouts increased from 2.5 to 4.5 min and the number of bouts from 2 to 6, rest intervals 2.5 min), and continuous aerobic running (running velocity $20\text{ m}\cdot\text{min}^{-1}$, daily running duration increased from 8 to 45 min) were used. In one group, a swimming training program was used (initially 60 min of exercise, duration increased by 10 min daily up to 3 h). In the fifth group, fast climbing was used with the aid of a special vertical treadmill, stimulating climbing up to 1.5 m on a slope of 80° (duration of each bout 5 s, additional load increasing from the initial of 100 to 350 g on the 6th week; number of exercise bouts increased from 2 to 5; rest intervals between bouts, 1.5 min). The duration of training was 10 weeks for running, and 6 weeks for swimming and fast climbing; 48 h after the last training session the rats were sacrificed by cervical dislocation. Both untrained and swimming-trained rats were injected with $1\text{ mg}\cdot\text{ng}^{-1}$ of isoprenaline 10 min prior to sacrifice.

Both the soleus and the quadriceps femoris muscle were excised from the animals. The quadriceps muscles were divided into red and white parts, and only

the white parts containing mainly fast-twitch glycolytic (FG) fibers were used. The soleus muscles were used as samples containing slow-twitch oxidative (SO) fibers. Samples of the right side muscles were immediately frozen in liquid nitrogen and stored at -20°C for determination of glycogen and lactate. Muscles of the left side were used for recording the development of ischemic contracture tension, and were then frozen and stored for glycogen and lactate determination. All preparative and analytical procedures were performed at 4°C . Glycogen was determined by the method of Lo *et al.* [10] and lactate by the standard enzymatic procedure after extracting the powered tissue with perchloric acid. For lactate determination, commercial kits of Lachema (Czechoslovakia) were used. All analyses were made in duplicate.

The development of ischemic contracture in muscles was recorded on a chart recorder using the method of Kôrge and Männik [5]. The experimental solution contained 137 mM NaCl, 4 mM KCl, 1 mM MgCl_2 , 1 mmol KH_2PO_4 , 12 mM NaHCO_3 and 2 mM CaCl_2 , and was kept at 37°C . The results were expressed as the mean $\pm$ SEM. Statistical significance between subgroups was evaluated by the Student-Fischer *t*-test as well as paired *t*-test. The probability level of 0.05 was designated as significant. Correlation analysis was also performed between individual indices.

RESULTS

Effects of medium compositions on the development of contractures

Within the pH range from 5.5 to 8.5, the rate of contracture development was inversely related to the medium pH (Fig. 1). After steady contracture tension had developed, the lactate content in muscles containing fast-twitch glycolytic (FG)

Fig. 1. Development of contracture in isolated soleum muscle at various medium pH levels. Ordinate-arbitrary units indicating muscle shortening.

fibers was proportional to the medium pH. During the contracture, the glycogen content appeared to be inversely proportional to the medium pH, but the differences were insignificant. In muscles containing slow-twitch oxidative (SO) fibers, the lactate and glycogen content during contracture was not related to the medium pH. A significant change in the rate of contracture tension development was found at pH 8.5 but not at pH 5.5 in comparison with that at pH 7.4 (Table 1). At pH 7.4 and 8.5, the contracture developed faster in SO than in FG fibers, while lactate and glycogen levels were higher during contracture in FG than in SO muscle. At pH 5.5 these differences were not significant (Table 1).

Addition of 120 mM lactate to the medium caused a significant increase in the rate of contracture development in SO fibers (from 0.73 ± 0.021 to 1.08 ± 0.029 mm · min⁻¹; in FG fibers, from 0.61 ± 0.016 to 0.97 ± 0.02 mm · min⁻¹).

Effects of acute exercise

Swimming for both 4 and 8 h caused an increase in the rate contracture development. The effect of 8-h swimming was more pronounced. During the post-exercise recovery, the rate of contracture development declined. Four hours after the end of 4-h swimming but not after 8-h swimming, the contracture developed was close to that in control rats. The lactate levels were lower after 4-h swimming in both types of muscle fibers and after 8-h swimming only in FG muscle fibers. The glycogen content was lower in both types of muscle after 4- and 8-h swimming (Table 2).

An increased lactate content and decreased glycogen content was caused by swimming in both types of muscle fibers. Significant correlations were found between the lactate and glycogen levels just after the exercise, and the rate of contracture development. In SO fibers, the dependence on the lactate level at the beginning of contracture was higher ($r=0.78$) than in FG fibers ($r=0.54$). In FG fibers, a very high negative correlation was found between glycogen level at the beginning of contracture and the rate of contracture development ($r=-0.91$). In SO fibers, this correlation was lower ($r=-0.67$).

The dependence of the rate of contracture development on the medium pH was also confirmed in muscles excised after swimming for 4 or 8 h (Table 3).

Effects of exercise training

Three variants of training (interval training, prolonged swimming, and fast climbing) resulted in a reduction in the rate of contracture development in both types of muscle fibers (Table 4). However, these changes were not related to the training effect on the muscle glycogen store. Fast running (sprint training) increased the rate of contracture development in both types of muscle fibers. The same was the effect of endurance running on FG fibers. In all these three cases, there were no significant increases in the muscle glycogen content. In SO fibers, interval training caused an elevated lactate level during contracture. In FG fibers, prolonged running caused a decrease in the lactate level during contracture.

Table 1. Dependence of contracture development and glycolysis in isolated muscle on medium pH (mean $\pm$ SEM, $n=7$).

	Soleus muscle (oxidative fibers)			Quadriceps muscle (glycolytic fibers)		
	pH 5.5	pH 7.4	pH 8.5	pH 5.5	pH 7.4	pH 8.5
V_c ($\text{mm} \cdot \text{min}^{-1}$)	0.79 ± 0.02	0.71 ± 0.03	$0.65 \pm 0.02^*$	$0.77 \pm 0.02^*$	0.58 ± 0.03	$0.43 \pm 0.02^*$
Lactate during contracture ($\mu\text{g} \cdot \text{g}^{-1}$)	39.9 ± 2.8	41.3 ± 4.3	41.3 ± 3.4	$45.7 \pm 3.0^*$	58.8 ± 5.4	$66.4 \pm 4.8^*$
Glycogen during contracture ($\mu\text{g} \cdot \text{g}^{-1}$)	8.48 ± 0.32	7.94 ± 0.32	7.38 ± 0.38	9.95 ± 0.51	8.84 ± 0.78	$8.03 \pm 0.49^*$

V_c , rate of contracture development. Asterisks denote statistically significant difference ($p < 0.05$) from values obtained at pH 7.4.

Table 2. Effects of swimming for 4 or 8 h on the rate of contracture development and glycogenolysis in isolated muscles (mean $\pm$ SEM, $n=7$).

	Oxidative fibers			Glycolytic fibers		
	V_c ($\text{mm} \cdot \text{min}^{-1}$)	Lactate at contracture ($\mu\text{g} \cdot \text{g}^{-1}$)	Glycogen at contracture ($\mu\text{g} \cdot \text{g}^{-1}$)	V_c ($\text{mm} \cdot \text{min}^{-1}$)	Lactate at contracture ($\mu\text{g} \cdot \text{g}^{-1}$)	Glycogen at contracture ($\mu\text{g} \cdot \text{g}^{-1}$)
Sedentary controls	0.71 ± 0.02	41.3 ± 2.6	7.95 ± 0.61	0.61 ± 0.02	58.8 ± 2.4	8.85 ± 0.54
Immediately after 4-h swimming	$1.03 \pm 0.05^*$	$24.2 \pm 1.4^*$	$2.91 \pm 0.09^*$	$0.89 \pm 0.04^*$	$26.5 \pm 1.0^*$	$6.83 \pm 0.31^*$
1.5 h after swimming	$0.89 \pm 0.03^*$	35.2 ± 3.2	5.72 ± 0.69	0.63 ± 0.02	$36.4 \pm 2.0^*$	$7.02 \pm 0.35^*$
4 h after swimming	0.72 ± 0.03	44.9 ± 3.1	9.00 ± 0.71	$0.54 \pm 0.02^*$	59.7 ± 4.9	$7.01 \pm 0.34^*$
Sedentary controls	0.64 ± 0.01	41.2 ± 2.9	7.90 ± 0.98	0.58 ± 0.02	56.7 ± 3.9	10.18 ± 0.79
Immediately after 8-h swimming	$1.38 \pm 0.06^*$	45.5 ± 3.0	$1.10 \pm 0.07^*$	$0.99 \pm 0.03^*$	$42.1 \pm 2.1^*$	$3.02 \pm 0.28^*$
4 h after swimming	$0.93 \pm 0.04^*$	$28.2 \pm 2.0^*$	$3.81 \pm 0.18^*$	$0.73 \pm 0.03^*$	$31.0 \pm 2.2^*$	$5.91 \pm 0.48^*$

Asterisk denotes a statistically significant difference ($p < 0.05$) from values in sedentary controls.

Table 3. Dependence of the contracture development in muscles excised after swimming for 4 or 8 h (mean $\pm$ SEM, $n=7$).

	Oxidative muscle			Glycolytic muscle		
	pH 5.5	pH 7.4	pH 8.5	pH 5.5	pH 7.4	pH 8.5
After 4-h swimming						
V_c ($\text{mm} \cdot \text{min}^{-1}$)	1.19 $\pm$ 0.05	1.03 $\pm$ 0.05	0.88 $\pm$ 0.03*	1.03 $\pm$ 0.05*	0.89 $\pm$ 0.03	0.62 $\pm$ 0.01*
Lactate at contracture ($\mu\text{g} \cdot \text{g}^{-1}$)	21.7 $\pm$ 1.7	24.2 $\pm$ 1.9	38.0 $\pm$ 3.1*	24.7 $\pm$ 1.5	26.5 $\pm$ 3.0	54.57 $\pm$ 2.8*
Glycogen at contracture ($\mu\text{g} \cdot \text{g}^{-1}$)	3.27 $\pm$ 0.14	2.91 $\pm$ 0.09	3.03 $\pm$ 0.12	7.98 $\pm$ 0.61	6.83 $\pm$ 0.42	6.74 $\pm$ 0.54
After 8-h swimming						
V_c ($\text{mm} \cdot \text{min}^{-1}$)	1.47 $\pm$ 0.07*	1.20 $\pm$ 0.06	0.88 $\pm$ 0.04*	1.20 $\pm$ 0.05*	0.99 $\pm$ 0.04	0.81 $\pm$ 0.05*
Lactate at contracture ($\mu\text{g} \cdot \text{g}^{-1}$)	43.9 $\pm$ 2.8	45.5 $\pm$ 3.7	50.3 $\pm$ 3.8	51.1 $\pm$ 4.1	52.1 $\pm$ 5.9	58.0 $\pm$ 2.9
Glycogen at contracture ($\mu\text{g} \cdot \text{g}^{-1}$)	1.97 $\pm$ 0.05*	1.10 $\pm$ 0.08	0.88 $\pm$ 0.03*	3.02 $\pm$ 0.37	3.02 $\pm$ 0.13	4.11 $\pm$ 0.22*

Asterisk denotes a significant difference ($p < 0.05$) from values at pH 7.4.

Table 4. Effects of various training on contracture development in isolated muscles (mean $\pm$ SEM).

	n	Soleus muscle (oxidative fibers)			Quadriceps muscle (glycolytic fibers)		
		Glycogen ($\mu\text{g} \cdot \text{g}^{-1}$)	V_c ($\text{mm} \cdot \text{min}^{-1}$)	Lactate at contracture ($\mu\text{g} \cdot \text{g}^{-1}$)	Glycogen ($\mu\text{g} \cdot \text{g}^{-1}$)	V_c ($\text{mm} \cdot \text{min}^{-1}$)	Lactate at contracture ($\mu\text{g} \cdot \text{g}^{-1}$)
Sedentary untrained rats	7	7.81 $\pm$ 0.38	0.72 $\pm$ 0.17	43.2 $\pm$ 3.1	9.97 $\pm$ 0.97	0.57 $\pm$ 0.01	56.4 $\pm$ 2.0
Rats trained by a model of sprint training	6	9.74 $\pm$ 1.21	0.80 $\pm$ 0.03*	38.9 $\pm$ 2.1	10.87 $\pm$ 1.42	0.71 $\pm$ 0.03*	45.7 $\pm$ 3.3*
Rats trained by a model of interval training	6	12.49 $\pm$ 0.08*	0.58 $\pm$ 0.01*	56.1 $\pm$ 3.8*	16.50 $\pm$ 0.51*	0.48 $\pm$ 0.02*	59.1 $\pm$ 4.5
Rats trained by endurance training	6	20.98 $\pm$ 1.07*	0.70 $\pm$ 0.02	47.5 $\pm$ 1.2	13.88 $\pm$ 2.01	0.65 $\pm$ 0.02*	48.7 $\pm$ 2.1*
Rats trained by prolonged swimming	7	23.45 $\pm$ 0.94*	0.60 $\pm$ 0.03*	50.9 $\pm$ 4.7	15.62 $\pm$ 1.07*	0.51 $\pm$ 0.01*	52.8 $\pm$ 2.7
Rats trained by fast clambering (strength training)	6	10.05 $\pm$ 0.68*	0.65 $\pm$ 0.02*	49.9 $\pm$ 5.8	14.78 $\pm$ 1.07*	0.40 $\pm$ 0.01*	68.9 $\pm$ 6.7

Asterisk denotes significant difference ($p < 0.05$) from values in sedentary untrained rats.

Table 5. Action of isoprenaline (subcutaneous injection of $1 \text{ mg} \cdot \text{kg}^{-1} \cdot \text{b.w.}$) on the contracture development in muscles isolated from untrained and swimming-trained rats (mean $\pm$ SEM, $n=7$).

	Oxidative fibers		Glycolytic fibers	
	V_c ($\text{mm} \cdot \text{min}^{-1}$)	Lactate at contracture ($\mu\text{g} \cdot \text{g}^{-1}$)	V_c ($\text{mm} \cdot \text{min}^{-1}$)	Lactate at contracture ($\mu\text{g} \cdot \text{g}^{-1}$)
Untrained rats	0.55 ± 0.01	59.0 ± 4.7	0.60 ± 0.03	54.7 ± 3.7
Untrained rats + isoprenaline	$0.93 \pm 0.04^*$	$25.7 \pm 1.1^*$	$0.95 \pm 0.05^*$	$26.7 \pm 0.9^*$
Trained rats	0.60 ± 0.03	50.9 ± 4.7	0.51 ± 0.01	52.8 ± 2.7
Trained rats + isoprenaline	0.64 ± 0.02	49.2 ± 3.4	0.51 ± 0.02	58.5 ± 4.1

Asterisk denotes a statistically significant difference between injected and noninjected rats ($p < 0.05$).

Table 6. Effect of adrenalectomy on contracture development in muscles excised from sedentary rats or after 4-h swimming (mean $\pm$ SEM, $n=6$).

	Oxidative fibers		Glycolytic fibers	
	V_c ($\text{mm} \cdot \text{min}^{-1}$)	Lactate ($\mu\text{mol} \cdot \text{g}^{-1}$)	V_c ($\text{mm} \cdot \text{min}^{-1}$)	Lactate ($\mu\text{mol} \cdot \text{g}^{-1}$)
Adrenalectomized rats				
Sedentary	0.80 ± 0.04	36.2 ± 3.8	0.60 ± 0.03	43.7 ± 3.8
Immediately after swimming	1.52 ± 0.08	16.1 ± 1.8	0.93 ± 0.04	20.2 ± 1.1
4 h after swimming	1.05 ± 0.03	30.9 ± 1.4	0.77 ± 0.03	33.6 ± 3.1
Normal rats				
Sedentary	0.71 ± 0.02	41.3 ± 2.6	0.61 ± 0.02	58.8 ± 2.4
Immediately after swimming	1.03 ± 0.05	24.2 ± 1.4	0.89 ± 0.04	26.5 ± 1.0
4 h after swimming	0.72 ± 0.03	44.9 ± 3.1	0.63 ± 0.02	59.7 ± 4.9

Hormonal influences

A large dose of isoprenaline administered to untrained rats caused a decrease in the rate of contracture development in association with a low lactate level during contracture in both types of muscle fibers. Trained rats failed to show such responses to isoprenaline injection (Table 5).

Adrenalectomy produced no significant changes in the rate of contracture development in sedentary rats. During contracture, however, a decreased glycogen level in SO fibers and reduced lactate level in FG fibers were noticed (Table 6). In adrenalectomized rats, the effect of 4-h swimming on the rate of contracture development was pronounced in SO fibers but not in FG fibers. After swimming, the velocity of contracture development in SO fibers was 1.5 times higher than in FG fibers (1.52 ± 0.08 compared to $1.03 \pm 0.05 \text{ mm} \cdot \text{min}^{-1}$ in normal animals). In exercised adrenalectomized rats, the lactate level during contracture were significantly lower and glycogen levels higher compared with exercised normal rats. Results obtained 4 h after swimming indicated that the recovery process proceeded in adrenalectomized rats more slowly than in normal rats.

DISCUSSION

The present results suggest the dependence of the rate of ischemic contracture development in isolated muscles on the capacity for anaerobic glycolysis. The inverse relationship between the magnitude of contracture development and capacity for anaerobic glycolysis is indicated by the faster development of contracture in SO fibers than in FG fibers. FG fibers are known to have a higher capacity for anaerobic glycolysis than SO fibers (for review see [11]). Anaerobic glycolysis is inhibited by the decreased pH and accumulated lactate [12–15]. On the other hand, alkalosis promotes anaerobic glycolysis [16], in accordance with the present result that the rate of contracture development was inversely related to the medium pH. Addition of lactate increased the rate of contracture development. In FG fibers, the dependence of the rate of glycolysis is supported by the fact that, during contracture, the lactate level was directly proportional and glycogen level inversely proportional to the medium pH. In FG fibers, the lactate levels were higher than in SO fibers during contracture.

Four-hour swimming caused an increase in the muscle lactate content (in SO fiber, from 3.2 ± 0.07 to $12.4 \pm 0.68 \mu\text{g} \cdot \text{g}^{-1}$; in FG fibers, from 3.7 ± 0.09 to $14.7 \pm 1.75 \mu\text{g} \cdot \text{g}^{-1}$). Consequently, a part of the capacity for anaerobic glycolysis may be utilized during exercise to result in acceleration of contracture development. Taking into account the lactate level in muscle when the contracture was started, it appeared that, in SO fibers, the contracture caused a rise in the muscle lactate content by $3.8 \pm 2.6 \mu\text{g} \cdot \text{g}^{-1}$ in sedentary rats and only by $5.3 \pm 2.3 \mu\text{g} \cdot \text{g}^{-1}$ in exercised rats. On FG fibers, the rises in lactate content were 55.1 ± 2.4 and $46.0 \pm 1.2 \mu\text{g} \cdot \text{g}^{-1}$, respectively. During contracture, glycogen was depleted almost in the same amount during contracture in sedentary and exercised rats (SO fibers, $2.3 \pm$

0.9 and $3.2 \pm 0.4 \mu\text{g} \cdot \text{g}^{-1}$ in sedentary and exercised rats respectively; FG fibers, 1.6 ± 1.0 and $2.1 \pm 0.7 \mu\text{g} \cdot \text{g}^{-1}$ respectively). The dependence of contracture development on the initial lactate and glycogen levels was confirmed by their statistically significant correlations.

The picture was almost the same after swimming for 8 h. The rate of contracture development increased in association with a decrease in the amount of lactate produced during contracture. The lactate concentration increased in SO fibers by $26.6 \pm 3.2 \mu\text{g} \cdot \text{g}^{-1}$ instead of $38.1 \pm 2.9 \mu\text{g} \cdot \text{g}^{-1}$ in controls, in FG fibers by $25.3 \pm 2.3 \mu\text{g} \cdot \text{g}^{-1}$ instead of $54.3 \pm 3.9 \mu\text{g} \cdot \text{g}^{-1}$ in controls. After 8-h swimming the contracture was started at a low glycogen level. Further glycogen depletion was significantly reduced.

Exercise training exerts a strong influence on the muscle glycogen store and oxidative metabolism (for review see [11]). Special training is necessary for improving the capacity for anaerobic glycolysis. The most effective tool for this is interval training, i.e., repetition of highly intensive exercise bouts over short rest intervals [17, 18]. The training regimes used caused increases in the glycogen stores of the skeletal muscle, except for sprint training. Sprint training resulted in an increase in the rate of contracture development in SO fibers. In FG fibers, the rate of contracture development increased as a result of sprint training and endurance running. The increased rate of contracture development was associated with reduced accumulation of lactate during contracture. These results may be interpreted on the basis of the specific nature of adaptation to chronic exercises. In sprint exercises, the main pathway for ATP resynthesis consists of breakdown of phosphocreatine [18, 19], while in aerobic endurance exercises—oxidative phosphorylation [11, 18]. Therefore, the adaptation processes concern in the first place these metabolic pathways. Instead of improvement of anaerobic glycolysis, the present results suggested that the capacity for this metabolic pathway was even reduced.

In both types of skeletal muscle fibers, the rate of contracture development decreased as a result of interval training as well as prolonged swimming and fast climbing. The lactate content during contracture tended to be lower in these cases except for the effect of prolonged swimming on FG fibers. The stimulatory effect of interval training on anaerobic working capacity and glycolytic enzymes is rather well documented [20–22]. One may assume the same result from repeated fast climbing. In prolonged swimming the action on aerobic processes may be connected with repeated diving of rats during swimming.

Administration of isoprenaline in large doses causes pronounced metabolic disturbances. An expression of those was an increased rate of contracture development in association with reduced lactate level during contracture. Obviously, the metabolic disorders induced by isoprenaline aggravated the capacity for anaerobic glycolysis. Swimming training counterbalanced the action of isoprenaline.

Adrenalectomy accelerated the contracture development in SO fibers and decreased the lactate accumulation during contracture in FG fibers. In normal

sedentary rats, the lactate level increased by $11.8 \pm 1.6 \mu\text{mol} \cdot \text{g}^{-1}$ during the contracture, while in sedentary adrenalectomized rats only by $5.0 \pm 1.9 \mu\text{mol} \cdot \text{g}^{-1}$ in SO fibers. In FG fibers, lactate increased during contracture in normal rats by 55.1 ± 2.8 and in adrenalectomized rats by $41.6 \pm 3.8 \mu\text{mol} \cdot \text{g}^{-1}$. In oxidative fibers the effects of exercise were more pronounced in adrenalectomized rats. In SO fibers, the rate of contracture development increased 1.5 times. The lactate production during contracture in exercised adrenalectomized rats was $5.0 \pm 1.9 \mu\text{mol} \cdot \text{g}^{-1}$ compared to $11.8 \pm 1.6 \mu\text{mol} \cdot \text{g}^{-1}$ in normal rats, indicating that adrenal insufficiency aggravates anaerobic glycolysis in skeletal muscles during exercise [23, 24]. In adrenalectomized rats, glycogen utilization decreased in the soleus muscle containing mainly SO fibers [23–26].

In conclusion, the obtained results gave evidence that the magnitude of contracture development in isolated skeletal muscle is inversely related to the capacity for anaerobic glycogenolysis and may be used as an integral index of this capacity.

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