N O T I C E

THIS DOCUMENT HAS BEEN REPRODUCED FROM
MICROFICHE. ALTHOUGH IT IS RECOGNIZED THAT
CERTAIN PORTIONS ARE ILLEGIBLE, IT IS BEING RELEASED
IN THE INTEREST OF MAKING AVAILABLE AS MUCH
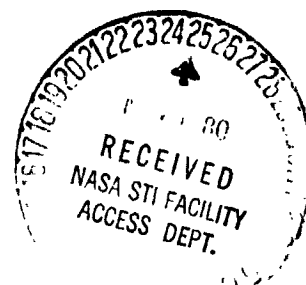INFORMATION AS POSSIBLE

EFFECT OF HYPOKINESIA ON CARDIAC CONTRACTILE FUNCTION AND NERVOUS REGULATION OF THE HEART

Meyerson, F. Z., V. I. Kapel'ko, M. S. Gorina,
A. N. Shchegol'kov and N. P. Larionov

EFFECT OF HYPOKINESIA ON CARDIAC CONTRACTILE FUNCTION
AND NERVOUS REGULATION OF THE HEART

Meyerson, F. Z., V. I. Kapel'ko, M. S. Gorina,
A. N. Shchegol'kov and N. P. Larionov*

It is well known, that adaptation of the heart to prolonged change in the lev- /1138**
el of load as a function of the size of the load may lead to three different condi-
tions: deadaptation of the heart in hypokinesia, adaptation of the heart with train-
ing and finally compensatory hypertrophy in cases of circulatory illnesses, the last
mentioned having been designated as cardiac transadaptation [4].

The theory that is currently developing in regard to the mechanism of these
three conditions [14] calls for a detailed comparison of the contractile function of
the myocardium in adaptation, transadaptation and deadaptation of the heart. Mean-
while, in contrast to adaptation and transadaptation, the contractile function of the
heart in deadaptation that has been induced by hypokinesia was the subject of only i-
solated research projects [11] and was not subjected to quantitative evaluation by
modern methods.

The purpose of the present research is to assess the heart's pumping function as
well as the processes of contraction and relaxation of the cardiac muscle in longterm
hypokinesia and on such a basis to compare the function of the organ in the three ba-
sic conditions that characterize its longterm adaptation to a changed load level.

Methodology. In the morphology laboratory of the Kiev Research Institute for Medical
Problems in Physical Education hypokinesia was produced in young rabbits by placing
each animal in a separate narrow cage. The hypokinetic period lasted 3 months. Dur-
ing that time the animals' weight increased slowly or even decreased. 3 animals /1139

---

out of 9 after 1.5-2 months presented paralysis of the hindlimbs. In these animals after 3 months, regardless of weight loss, the weight of the left ventricle had increased 34% and the relative weight 63%. Relative weight of the right ventricle had increased 40%. Experimental results for these animals are not included in the results. For the remaining 6 animals there was no difference in the weight of the ventricles as compared with control rabbits of the same weight. These animals and 9 controls were subjected to acute experiments under urethane anesthesia (1.6 g/kg) with open thorax and artificial respiration. For measuring pressure the cavity of the left ventricle was catheterized through the apex and electromagnetic fluorometric Nihon Kohden sensors were placed on the ascending aorta and left carotid artery. A Mingograf-34 was used to record blood flow in these vessels and the pressure in the left ventricle together with its differential curve.

These processes were recorded during the stabilization stage 10 minutes after completion of the preparation and likewise when there were two functional loads: an increase in contraction rate and 30 second clamping of the ascending aorta. Contraction rate was imposed by electrostimulation via an electrode attached to the right atrium. The rate was increased, starting from the initial level, 0.5 Hz at a time until a pronounced alternation appeared.

In order to determine adrenoreactivity there was a single administration of norepinephrine (0.5 micrograms/kg) and for the assessment of cholinoreactivity excitation of the peripheral segment of the vagus nerve following bilateral vagotomy. The excitation parameters were: frequency 20 Hz, length 5 milliseconds, initial amplitude 2 volts with subsequent lowering to threshold value. Sequence of experimental steps was as follows: increase in frequency, administration of norepinephrine, resection of vagus nerves with excitation of the right nerve, aorta clamping.

The following indices were calculated: index of the intensity of functional structures - multiplying the pressure being built up in the left ventricle by the contraction rate and then dividing into the weight of the ventricle; contractile index [18] - dividing maximal speed of pressure development into the value for the pressure being developed at the moment of maximum speed; relaxation index [5] - dividing the maximal speed of pressure drop into the value of the systolic pressure being developed; stroke volume - by the area under the blood flow curve; time of isometric contraction - from the beginning of the rise in pressure to the beginning

2

of ejection; time of relaxation - from the end of ejection until the ventricular pressure drops to the diastolic level; diastolic pressure in the aorta - by the amount of systolic pressure in the ventricle at the moment when ejection begins; negative chronotropic effect at excitation of the vagus nerve - by the degree of lengthening of the interval between contractions. Following the experiment there was a determination of the weight of the left ventricle and the conventional method was used [12] to determine actomyocin ATPase activity in a tissue specimen.

Results of the Study. Prior to thoracotomy the contraction rate in the control experiments was 303+10/min and did not differ reliably from the corresponding value in experiments on the animals subjected to hypokinesia, 282+22.

Table 1 presents data characterizing the pumping function of the left ventricle and the contractile function of the myocardium. Contraction rate, minute volume and stroke volume showed no reliable difference from control values and this agrees with observations on humans [10].

At the same time there was an essential difference in the indices for the contractile function of the myocardium. The difference lay in the fact, that when there was no change in systolic pressure and the structural functioning index characterizing functional load exerted on a unit of muscular mass the cardiac muscle of animals in hypokinesia was marked by a significant drop in the contractile index and relaxation index and likewise by a much slower buildup and letdown of pressure in the ventricle. The slowing down of contraction and relaxation processes in the heart muscle of animals in hypokinesia was not accompanied by reliable changes in the phasic structure of systole and diastole.

There was also no change in the indices for maximal volume rate and linear rate of ejection into the aorta. The concurrence of a 42% drop in the speed of pressure buildup and a practically unaltered ejection rate may be conditioned, as the data in Table 1 show, by the drop in diastolic pressure and resistance in the aorta.

The reduction in overall vascular resistance under the influence of hypokinesia is a rather well known fact [3] and in our experiments this reduction was accompanied by a sharp rise in the flow of blood through the carotid artery of hypokinetic an- /1140 imals: maximal flow rate and overall amount of flow per min rose 1.6-2 times (Tab.1).

TABLE 1. INDICES FOR CONTRACTILE FUNCTION OF LEFT VENTRICLE AND HEMODYNAMICS
IN CONTROLS AND HYPOKINETIC ANIMALS WITH SPONTANEOUS CONTRACTION RATE

A Показатели		B Контроль (9)	C Гипокинезия (6)	D % изменения	P <
Минутный объем (мл)	a	217±4	202±23	—7	
Минутный объем (мл/кг)	b	108±6	100±12	—8	
Ударный объем (мл)	c	0.79±0.04	0.82±0.12	+3.7	
Частота сокращений за 1 мин	d	274±7	251±17	—8	
Давление, развиваемое левым желудочком (мм)	e	85±3	78±5	—9	
» (Pa × 10³)	f	11.33±0.40	10.4±0.67		
Показатель ИФС (мм/мг/мин)	g	7.7±0.3	6.55±0.75	—15	
» » (Pa/мг/мин)	h	1028±40	873±100		
Скорость развития давления (мм/с)	i	5070±300	2950±300	—42	0.001
» (Pa × 10³/c)	j	676±40	393±40		
Скорость падения давления (мм/с)	k	2480±200	1730±80	—30	0.001
» (Pa×10³/c)	l	331±27	231±11		
Индекс сократимости (с⁻¹)	m	93±6	68±4	—27	0.01
Индекс расслабления (с⁻¹)	n	29±2	22±1	—24	0.01
Время изометрического сокращения (мс)	o	20±1.5	21±2	+5	
Время изгнания (мс)	p	97±4	118±8	+14	
Время расслабления (мс)	q	51±4	55±3	+8	
Диастолическая пауза (мс)	r	53±4	53±7	0	
Максимальная объемная скорость изгнания (мл/мин)	s	976±58	870±69	—11	
Диаметр аорты (мм)	t	5.7±0.4	5.6±0.3	—2	
Максимальная линейная скорость изгнания (см/с)	u	73±7	65±7	—11	
Диастолическое давление в аорте (мм)	v	54±3	43±4	—20	0.05
» (Pa × 10³)	w	7.20±0.40	5.73±0.53		0.05
Максимальная скорость протока в сонной артерии (мл/мин)	x	32±3	51±7	+60	0.05
Проток в сонной артерии за мин (мл)	y	5.5±1.0	11.0±1.7	+100	0.02

z Примечание. Давление выражено в мм рт. ст. и паскалях (1Pa = 133.32 мм рт. ст.).

Key: A. Indices   B. Control (9)   C. Hypokinesia (6)   D. % change

a. Minute volume (ml)
b. Minute volume (ml/kg)
c. Stroke volume (ml)
d. Contraction rate/min
e. Pressure built up in left ventricle (mm)
f. same (Pa x 10³)
g. Structural function index (mm/mg/min)
h. same (Pa/mg/min)
i. Rate of pressure development (mm/sec)
j. same (Pa x 10³/sec)
k. Rate of pressure drop (mm/sec)
l. same (Pa x 10³/sec)
m. Contractile index (s^{-1})
n. Relaxation index (s^{-1})
o. Time of isometric contraction (msec)
p. Time of ejection (msec)
q. Time of relaxation (msec)
r. Diastolic pause (msec)
s. Maximal volume rate of ejection (ml/min)
t. Diameter of aorta (mm)
u. Maximal linear ejection rate (cm/sec)
v. Diastolic pressure in aorta (mm)
w. same (Pa x 10³)
x. Maximal flow rate in carotid artery (ml/min)
y. Flow in carotid artery per minute (ml)
z. Remark: Pressure expressed in mm Hg and in Paschals (1 Pa = 133.32 mm Hg)

4

TABLE 2. CONTRACTILE FUNCTION OF THE HEART AND HEMODYNAMICS OF CONTROL AND HYPOKINETIC ANIMALS AT A RATE OF 330/MIN

A Показатели		B Контроль	C Гипокинезия	D % изменения	p<
Давление в левом желудочке (мм)	a	80±4.5	68±8.5	—15	
» (Pa × 10³)	b	10.67±0.60	9.07±1.13		
Показатель ПОС (мм мг/мин)	c	7.6±0.33	6.8±1.15	—11	
» (Pa мг мин)	d	1013±44	907±153		
Скорость развития давления (мм/c)	e	4160±420	2800±290	—32	0.05
» (Pa × 10³/c)	f	547±56	373±39		
Скорость падения давления (мм/c)	g	2800±310	1500±290	—45	0.02
» (Pa × 10³ c)	h	373±41	200±39		
Индекс сократимости (c⁻¹)	i	78±5	69±5	—12	
Индекс расслабления (r⁻¹)	j	34±2.1	23±2.0	—32	0.01
Время расслабления (мс)	k	43±6	53±6	+23	
Диастолическая пауза (мс)	l	33±9	23±5	—31	
Время изгнания (мс)	m	85±3	83±4	—3	
Объемная скорость выброса (мл мин)	n	750±92	618±80	—18	
Ударный объем (мл)	o	0.60±0.09	0.48±0.06	—20	
Минутный объем (мл)	p	197±30	157±23	—21	
Диастолическое артериальное давление (мм)	q	52±10	40±5	—19	
» (Pa × 10³)	r	6.93±1.33	5.33±0.67		

Key: A, B, C, D as in Table 1.

a. Pressure in left ventricle (mm)
b. same (Pa x 10³)
c. Structural function index (mm/mg/min)
d. same (Pa/mg/min)
e. Rate of pressure buildup (mm/sec)
f. same (Pa x 10³/sec)
g. Rate of pressure drop (mm/sec)
h. same (Pa x 10³/sec)
i. Contractility index (s^{-1})
j. Relaxation index (s^{-1})
k. Time of relaxation (msec)
l. Diastolic pause (msec)
m. Time of ejection (msec)
n. Volume rate of ejection (ml/min)
o. Stroke volume (ml)
p. Minute volume (ml)
q. Diastolic arterial pressure (mm)
r. same (Pa x 10³)

Thus in animals kept in hypokinesia a long time the reduction in the rate of contraction and relaxation processes is compensated by lowered resistance of the vascular bed. As a result the pumping function of the ventricle is maintained at nearly the normal level. However such compensation may prove inadequate under conditions of high contraction rate, when adaptive changes in the rate of contraction and relaxation play the leading role. Results obtained in comparing cardiac functions in the controls and hypokinetic animals at an elevated rate agree with this hypothesis.

Table 2 presents parameters of cardiac function at the maximal rate, which was used in all experiments: 330/min. The Table shows a clearer retardation in myocardial relaxation for hypokinetic animals — relaxation rate was 46% lower and relaxation index 32% lower, whereas at the initial rate the speed was lower by 30 and 24% respectively (Table 1). As a result myocardial relaxation time for these animals

5

TABLE 3. FUNCTIONAL INDICES OF LEFT VENTRICLE AT SHORT TERM AORTA CLAMPING

A Показатели на 3-5 с	B Контроль	C Гипокинезия	D % изменения
Частота сокращений (в мин) **a**	239±14	226±8	—6
Развиваемое давление (мм) **b**	156±8.5	172±14.8	+10
» (Pa × 10³) **c**	20.8±1.1	22.8±2.0	
Показатель ПФС (мм/мг/мин) **d**	11.6±0.36	12.3±0.85	+5
» (Pa/мг/мин) **e**	1546±48	1640±113	
Скорость развития давления (мм/с) **f**	3510±420	4300±670	+22
» (Pa × 10³/с) **g**	468±56	573±89	
Скорость падения давления (мм/с) **h**	2320±210	2600±480	+12
» (Pa × 10³/с) **i**	309±28	347±64	
Индекс сократимости (c⁻¹) **j**	67±9	61±3	—10
Индекс расслабления (c⁻¹) **k**	14.8±0.6	15.0±1.6	+1

Key: A'. Indices for 3-5 sec   B,C,D as in Table 1

a. Contraction rate (in min)   f. Pressure development rate (mm/sec)
b. Pressure buildup (mm)   g. same (Pa x 10^3/sec)
c. same (Pa x 10^3)   h. Pressure reduction rate (mm/sec)
d. Structural function index   i. same (Pa x 10^3/sec)
   (mm/mg/min)   j. Contractility index (sec^{-1})
e. same (Pa/mg/min)   k. Relaxation index (sec^{-1})

increased and there was a decrease in the diatolic pause in comparison with the controls. These changes went hand in hand with notable diminution in minute volume at a high imposed contraction rate. The value for minute volume in hypokinetic ani- /1142 mals went down 22% as compared with the initial level (Table 1), whereas in the controls the imposed frequency produced no significant reduction — 9% altogether.

In connection with another type of cardiac load — 30 sec aorta clamping — all functional parameters for the left ventricle of hypokinetic animals showed practically no difference from control values (Table 3). Thus there was no encroachment on the ability of the myocardium to generate power under the influence of hypokinesia.

Definite differences between control and hypokinetic animals were also noted in an evaluation of the sensitivity of the circulatory system to neural influences. It follows from Table 4, that in control experiments systolic pressure as well as the speed of myocardial contraction and relaxation rose by 44-70% under the influence of norepinephrine. At peak effect there was likewise an increase in the maximal speed of ejection amounting to 36% and stroke volume increased 23%. At the same time there was a rise in diastolic pressure in the aorta, expressive of the vascular component

A Показатели	B Контроль (6)	C Гипокинезия (6)	E Разница (в %)	p<
a Развиваемое давление	144±15	112±3	−32	0.05
b Частота сокращений	97±1	101±0.5	+4	
c Показатель НФС	140±15	113±3	−27	
d Минутный объем	122±6	115±5	−7	
e Ударный объем	123±6	113±5	−10	
f Скорость развития давления	117±15	128±9	−19	
g Индекс сократимости	125±9	120±8		
h Диастолическое давление в аорте	136±12	107±5	−29	0.05
i Максимальная объемная скорость изгнания	136±12	117±4		
j Скорость расслабления	170±33	120±8		
k Индекс расслабления	116±33	107±5		
l Время изгнания	97±1	98±2.5		
m Время расслабления	95±7	106±7		
n Диастолическая пауза	131±14	105±5		

KEY: A,B,C as in Table 1. E. Difference (in %)

a. Pressure building up
b. Contraction rate
c. Structural function index
d. Minute volume
e. Stroke volume
f. Rate of pressure buildup
g. Contractility index
h. Diastolic pressure in aorta
i. Maximal volume rate of ejection
j. Relaxation rate
k. Relaxation index
l. Ejection time
m. Relaxation time
n. Diastolic pause

of norepinephrine action. In hypokinetic animals the effect of norepinephrine was less pronounced in respect to all parameters; however a statistically reliable difference was observed only when comparison was made of the increase in pressure in the left ventricle and aorta. In the hypokinetic animals these indices had dropped by 29-32% (Table 4). Inasmuch as the value for pressure increase in the ventricle is regularly associated with the degree of increase in vascular resistance, the results obtained allow us to suppose, that the reduced inotropic effect of norepinephrine should not be regarded merely as an expression of reduced cardiac adrenoreactivity; it may be conditioned by the reduced rise in vascular resistance resulting from the lowering of adrenoreactivity of the vascular bed.

Excitation of the vagus nerve showed a negative chronotropic effect, which increased with increased intensity of the excitation (Figure 1). No matter what the intensity of the excitation, the magnitude of the effect on the heart of hypokinetic animals was about twice that in the controls. The threshold excitation value showing the minimal effect in these animals was likewise reduced by 22% (p < 0.05).
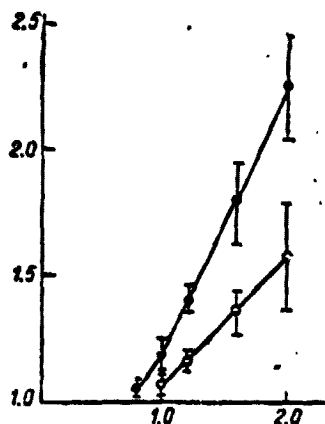
7

Fig. 1. Dependence of value of
negative chronotropic effect of
vagus nerve in controls (light
circles) and hypokinetic animals
(dark circles) upon intensity of
nerve excitation (in volts).
Effect was measured by ratio
between maximal intercontraction
interval at nerve excitation and
value of initial interval (M+m).

All in all the data testify to the fact,
that under the influence of prolonged hypo-
kinesia there is a reduction in the rate of
contraction and relaxation of the cardiac
muscle with unaltered contractile power.
Hypokinetic animals are distinguished by
greater depression of the contractile func-
tion when the contraction rate is increased,
by decreased adrenoreactivity but height-
ened cholinoreactivity.

Evaluation of Results. Longterm hypokinesia
leads to deadaptation to physical load. For
this reason the results obtained are best
compared with data on the contractile func-
tion of the heart in a context of adaptation
to physical load. It has been shown that
the cardiac muscle of animals adapted to physical loads is distinguished by an in- /1143
crease in the speed of contraction and relaxation [8], reduced depression of func-
tion when the rate is increased [2], increased adrenoreactivity [20] but unaltered
maximal contractile power [2,8].

These data attest to the fact, that the effects of adaptation and deadaptation
on the contractile function of the heart are opposed but share a common trait in that
both these shifts in the regime of motor activity affect for the most part the rate
of shortening of the cardiac muscle and have little effect on its contractile force.

In adaptation to physical loads acceleration of the process of myocardial con-
traction, as is now positively known [13,19], is conditioned by an increase of ATPase
myocin activity, while the acceleration of relaxation is influenced by an increase
in the "calcium pump" capability of the SR to absorb Ca^{++} ions from the miofibrils
[15]. Therefore the physiological changes established by the present study of them-
selves let us hypothesize the presence of corresponding ATPase myocin changes and
"calcium pump" changes in the myocardial cells of animals under prolonged hypokinesia.
Actually, the biochemical study we made on the animals used in our experiments have
shown, that the ATPase activity of actomyocin in control animals was 53+8, whereas in

hypokinetic animals it was 30 ± 5 µM/mg/min and had dropped by 43% ($p<0.05$), i. e. to the same degree as the contraction rate.

At this time there are no data on the capability of SR from the hearts of hypokinetic animals to absorb Ca^{++}. Our data on the reduction in the relaxation index permit us to theorize, that a reduction in the rate of absorption of Ca^{++} by fragments of reticulum from the hearts of these animals is a probability.

A comparison of the changes described above in respect to the contractile function of the heart in deadaptation with the changes observed in compensatory cardiac hypertrophy induced by constriction of the aorta in animals or by diseases of the circulatory system in humans reveals a great similarity between these two conditions. Thus, in compensatory hypertrophy one observes a reduction in the maximal speed and power of contraction [1,17], a reduction in the speed and index of relaxation [5,6], a reduction in the activity of the Ca^{++} pump [16], a reduction in the concentration of catecholamines in the myocardium and a reduction in cardiac adrenoactivity [7,9].

Thus the data obtained agree with the theory referred to before, that changes in cardiac function and metabolism in deadaptation and transadaptation are characterized by a pronounced commonality and opposed to changes due to training, i.e. in optimal adaptation of the heart. The mechanism of these relationships may be explained on the basis of the theory of the decisive role played by structural relationships in longterm adaptation and this is the subject of special study.

REFERENCES

1. Kapel'ko, V. I., Mekhanicheskiye svoystva gipertrofirovannoy serdechnoy myshtsy [Mechanical Properties of the Hypertrophicized Cardiac Muscle], Kardiologiya 11, 11, 89-96 (1971).

2. Kapel'ko, V. I. and L. M. Giber, Reaktsiya serdtsa na funktsional'nuyu nagruzku adaptirovannykh k fizicheskim nagruzkam krys [Cardiac Reaction to Functional Loading of Rats Adapted to Physical Loads], Fiziol. zh. SSSR 63, 4, 597-599 (1977).

3. Kovalenko, Ye. A., Patofiziologiya dlitel'noy gipokinezii [Pathophysiology of Longterm Hypokinesia], Kosm. biol. aviakosm. med. 10, 1, 3-15 (1976).

4. Meyerson, F. Z., Obshchiy mekhanizm adaptatsii i deadaptatsii serdtsa [General Mechanism of Cardiac Adaptation and Deadaptation], Kardiologiya 16, 9, 5-14 (1976).

5. Meyerson, F. Z. and V. I. Kapel'ko, Rol' vzaimosvyazi mezhdu siloy sokrashcheniya i skorostyu rasslableniya miokarda v prisposoblenii serdtsa k vozrastayushchey nagruzke [Role of the Interrelationship between Contractile Power and Rate of Myocardial Relaxation in Adaptation of the Heart to Increasing Load], Kardiologiya 13, 2, 19-30 (1973).

6. Meyerson, F. Z. and V. I. Kapel'ko, Sokrashcheniye i rasslableniye serdechnoy myshtsy pri kompensatornoy gipertrofii i trenirovannosti. Metabolizm miokarda. Mater. 2-go sov.-amer. simpoziuma [Contraction and Relaxation of the Cardiac Muscle in Compensatory Hypertrophy and Training. Myocardial Metabolism. Materials of the Second Soviet-American Symposium] Published by "Meditsina" 124-145 (1977).

7. Meyerson, F. Z. and N. P. Larionov, Depressiya sokratitel'noy funktsii i snizheniye effektivnosti ispol'zovaniya kisloroda pri kompensatornoy gipertrofii serdtsa [Depression of the Contractile Function and Reduction in Effectiveness of Oxygen Administration in Compensatory Cardiac Hypertrophy], Kardiologiya 15, 4, 107-114 (1975). /1144

8. Meyerson, F. Z., V. I. Kapel'ko and K. Pfayffer, Sokrashcheniye i rasslableniye serdechnoy myshtsy pri adaptatsii k fizicheskim nagruzkam [Contraction and Relaxation of the Cardiac Muscle in Adaptation to Physical Loads], Fiziol. zh. SSSR 62, 5, 793-795 (1976).

9. Meyerson, F. Z., B. N. Manukhin, M. G. Pshennikova and L. S. Rozanova, O mediatornom obmene miokarda pri kompensatornoy giperfunktsii i gipertrofii serdtsa [On the Mediator Metabolism of the Myocardium in Compensatory Hyperfunction and Hypertrophy of the Heart], Patol. fiziol. i eksper. terapiya 7, 1, 32-36 (1963).

10. Pometov, Yu. D. and B. S. Katkovskiy, Izmeneniye serdechnogo vybrosa i gazoobmena v pokoye pri gipokinezii [Change in Cardiac Ejection and Gas Exchange at Rest in Hypokinesia], Kosm. biol. aviyakosm. med. 6, 4, 39-46 (1972).

11. Pruss, G. M. and V. I. Kuznetsov, Sokratitel'naya funktsiya miokarda pri dli- tel'noy gipodinamii [Contractile Function of the Myocardium in Longterm Hy- podynamia], ibid. 8, 6, 45-49 (1974).

12. Baldwin, K. M., W. W. Winder and J. O. Holloszy, Adaptation of Actomyocin ATPase in Different Types of Muscle to Endurance Exercise, Amer. J. Physiol. 229, 2, 422-426 (1975).

13. Bahn, A. K. and J. Scheuer, Effect of Physical Training on Cardiac Actomyosin Adenosine Triphosphatase Activity, Amer. J. Physiol. 223, 6, 1486-1490 (1972)

14. Meerson, F. Z. and A. M. Breger, The Common Mechanism of the Heart's Adaptation and Deadaptation: Hypertrophy and Atrophy of the Heart Muscle, Basic Res. Cardiol. 72, 2/3, 228-234 (1977)

15. Penpargkul, S., D. I. Repke, A. M. Katz and J. Scheuer, Effect of Physical Training on Calcium Transport by Rat Cardiac Sarcoplasmic Reticulum, Circul. Res. 40, 2, 134-138 (1977).

16. Sordahl, L. A., W. B. McCollum, W. G. Wood and A. Schwartz, Mitochondria and Sarcoplasmic Reticulum Function in Cardiac Hypertrophy and Failure, Amer. J. Physiol. 224, 3, 497-502 (1973).

17. Spann, I. E., R. A. Buccino, E. H. Sonnenblick and E. Braunwald, Contractile State of Cardiac Muscle Obtained from Cats with Experimentally Produced Ven- tricular Hypertrophy and Heart Failure, Circul. Res. 21, 3, 341-354 (1967).

18. Veragut, U. P. and H. P. Krayenbühl, Estimation and Quantification of Myocardial Contractility in the Closedchest Dog, Cardiologia 47, 2, 96-112 (1965).

19. Wilkerson, J. E. and E. Evonuk, Changes in Cardiac and Skeletal Muscle Myosin ATPase Activities after Exercise, J. Appl. Physiol. 30, 3, 328-330 (1971).

20. Wyatt, H. L., W. Parmley, B. Rabinowitz, J. Tyberg, L. Chuck and M. Kligerman, Enhanced Cardiac Response to Catecholamines in Physically Trained Cats, Cir- culation 54, 4, Suppl. 2, 34, (1976).

copyright holder: "Fiziologicheskiy zhurnel SSSR"
1978