longation of poststress reparative DNA synthesis demonstrated in the present investigation. Injuries accumulating with age evidently do not leave untouched the genes which control DNA repair, and this must be reflected in the correctness of reparative synthesis. When the results of this investigation are analyzed, it can be accepted that repair of myocardial DNA in the older age group takes place in several stages and may perhaps include correction (resynthesis) of individual repaired DNA regions initially insufficiently correctly synthesized, and this requires extra time. This hypothesis is in agreement with the character of the time course of poststress reparative synthesis of myocardial DNA from the comparative age aspect (Fig. 2). During the first days after stress, in the course of induced reparative synthesis for a period of 2 days 88% of radioactivity is incorporated into DNA of animals of the younger age group, compared with only 33% of additional radioactivity incorporated into DNA of animals of the older group in the course of induced synthesis for 3 days. Consequently, prolongation of poststress DNA repair takes place with age.

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EFFECT OF EMOTIONAL-PAINFUL STRESS ON MYOCARDIAL CONTRACTILITY IN PROLONGED HYPOKINESIA

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KEY WORDS: myocardium; hypokinesia; stress.

Prolonged hypokinesia leads to arrest of growth and, in particular, to cessation of the increase in weight of the heart in rats [1, 4, 5]. A reduction in weight of the heart by more than one-third is due to the fact that the heart muscle consists of cardiomyocytes with a smaller volume than in the control. The ratio of area of sarcolemma to weight of the cell is increased for these cardiomyocytes. The quality of coupling of excitation with contraction and the functional capacity of the myocardium are increased correspondingly [4]. The problem of how acute stress is reflected in cardiac contracility of these animals has not hiterto been studied.

The aim of this investigation was to study the effect of emotional painful stress (EPS) on contractility of the heart muscle of animals previously exposed to a state of prolonged hypokinesia.

EXPERIMENTAL METHOD

Experiments were carried out on 64 male Wistar rats weighing 180-200 g, divided into four groups: 1) control (n = 21), 2) EPS (n = 10), 3) hypokinesia (n = 21), and 4) hypokinesia + EPS (n = 12). Hypokinesia for 60 days was produced by keeping the animals in special restraining cages. At the end of the period of hypokinesia the body weight of the

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TABLE 1. Effect of EPS on Contractility of Papillary Muscle of Albino Rats Exposed to Hypokinesia for 2 Months (M ± m)

Parameter	Control	EPS	Difference, %	Hypokinesia	Hypokinesia + EPS	Difference, %
Load at rest, g/mm²	0,92±0,13	0,99±0,13	+7	0,76±0,06	0,85±0,11	+12
Area of cross section, mm²	0,86±0,09	0,76±0,10	—2	0,80±0,05	0,83±0,05	+4
Amplitude of contraction, % of initial length of muscle	4,80±0,49	1,83±0,21*	—62	11,63±0,47*	8,93±0,36**	—23
Veloc. of contraction, m.u./sec	0,65±0,02	0,19±0,01*	—71	1,56±0,07*	1,09±0,03**	—30
Veloc. of relaxation, m.u./sec	0,60±0,01	0,13± ,01*	—75	1,45±0,06*	1,06±0,03**	—27

Legend. *) Values differing significantly from control, ** from corresponding values for "hypokinesia" group.

animals was 206 ± 42 g compared with 382 ± 10 g in rats kept in ordinary animal house cages; the weight of the heart was 607 ± 12 mg compared with 887 ± 15 mg in the control ($P < 0.001$). EPS was induced in the animals in the form of an "anxiety neurosis" [6] and its duration was 6 h. The rats of these groups, and also intact animals, were anesthetized with urethane 2 h after the end of exposure to stress and the heart was removed and placed in a dissecting bath, filled with aerated Krebs–Heneseleit solution, in which the posterior papillary muscle of the left ventricle was dissected. The preparation was placed in a working chamber with a capacity of 100 cm³, the rate of flow of the solution was 20 cm³/min, its temperature 29 ± 1°C, and pH 7.4. The working Krebs–Henseleit solution, aerated with carbogen (95% O_2 + 5% CO_2) in glass columns 120 cm high, had the following composition (in mM): NaCl 118.0; KCl 4.7; $CaCl_2$ 2.5; $MgSO_4$ 1.2; KH_2PO_4 1.2; $NaHCO_3$ 25.0; glucose 5.5. The $NaHCO_3$ solution was aerated beforehand with CO_2 to lower the pH and to prevent precipitation of Ca^{++} when added to the working solutions. The papillary muscle was stimualated by square pulses 5 msec in duration and 20% above threshold in amplitude by means of a type EUS-1 stimulator. The initial frequency of stimulation of 20 beats/min was maintained for 60 min until complete stabilization of the amplitude of contraction at minimal resting load. The optimal load at which the muscle was stretched to the so-called maximal length (L_{max}), resulting in maximal isotonic shortening of the preparation, was then found. The parameters of isotonic contraction were measured with a 51V21 capacitive transducer, a "Disa" oscilloscope (Denmark), and a 1428 mK 11 V photographic attachment, assembled into an apparatus for studying the contractile function of isolated papillary muscles by Sonnenblick's method in Kapel'ko's modification [2]. To enable the results to be compared the amplitude of contraction in each experiment was expressed as a percentage of length of the muscle, and the velocity of contraction and relaxation were expressed in muscle units per second (m.u./sec). The data for myocardial contractility of the animals were subjected to statistical analysis by Student's tests.

EXPERIMENTAL RESULTS

The results (Table 1) consistently confirmed existing data [4] to the effect that prolonged hypokinesia leads to a marked increase in the amplitude of contraction and the velocity of contraction and relaxation of the isolated papillary muscle of the animals, whereas a single exposure to EPS has the opposite action, i.e., depress contractility of the heart muscle. The data in Table 1 also show that the effect of stress on myocardial contractility of animals kept previously in a state of prolonged hypokinesia differed from that in the control. Absolute values of the decrease in amplitude of contraction and the velocity of contraction and relaxation after stress in animals exposed to prolonged hypokinesia were the same as in the control. However, a high level of contractile hypokinesia had the result that these differences, in relative values, were about 2.5 times less than in the control. Ultimately the basic parameters of myocardial contractility of the "small" hearts of animals which had not grown because of prolonged hypokinesia were many times higher after stress than in the control. In fact, the amplitude of contraction in the hypokinesia + stress series was 8.93% of the initial length of the muscle, whereas in the series in which control animals were exposed to stress it was 1.83% of the initial length of the muscle. The velocities of contraction and relaxation were related by a similar ratio.

In other words, these results are evidence that in the case of the "small" hearts of aminals exposed to prolonged hypokinesia and thus possessing a high capacity for realization of their contractile function, depression of that function by stress is less dangerous than for control animals.

When the mechanisms of this phenomenon are analyzed at least two possibilities must be considered. The first is that the powerful adrenergic effect associated with stress induces equal activation of lipid peroxidation, lipases, and phospholipases, i.e., it results in equal realization of the lipid triad of injury to biomembranes characteristic of stress [7]; correspondingly, an equal disturbance of the process of coupling of excitation with contraction leads to an equal decrease in the parameters of contractility of the heart muscle. However, this possibility is not in fact realistic, for equal injury to the sarcolemmal membranes of the cardiomyocytes, affecting a small and large relative area, must inevitably lead to equal (in relative values) disturbances of coupling processes and of myocardial contractile function. A different result was obtained in the present experiments.

The second possibility stems from the fact that prolonged hypokinesia is a chronic stress state which persists 2 months after the animals are placed in restraining cages [4, 5]. There is evidence that during repeated exposure to moderate stress, the resistance of animals [5] and, in particular, of their heart muscle [3], to the harmful action of stress rises significantly. Accordingly prolonged hypokinesia could be the cause of adaptation of this type and could lead to increased resistance of the animals' heart to stress injury.

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UNIT ACTIVITY OF THE GANGLION NODOSUM IN MYOCARDIAL ISCHEMIA

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The writers showed previously [1] that the discharge frequency of afferent neurons and interneurons in the bulbar cardiovascular center increases as a rule in myocardial ischemia (MI). These changes may evidently be linked not only with the increased flow of afferent impulses from the ischemic myocardium [3, 5-7], but also with changes in the firing pattern of different receptors and, in particular, from organs of the vascular and respiratory systems. The latter is particularly important because afferent impulses are sent to the bulbar cardiovascular center both from cardiovascular neurons and also from certain types of respiratory neurons of the ganglion nodosum [2].

The aim of this investigation was to study changes in the firing pattern of different types of ganglion nodosum neurons during the development of MI.

EXPERIMENTAL METHOD

Experiments were carried out on 39 cats of both sexes weighing 3-4 kg under pentobarbital anesthesia (30-40 mg/kg, intraperitoneally) with artificially ventilation by the Vita-I apparatus. The thorax and pericardium were opened and ligatures applied to the circumflex and anterior descending branches of the left coronary artery. Ischemia was produced by compressing the branches of the left coronary artery for 5-10 min. Unit activity in the gang-

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