association of oxygen with hemoglobin takes place 10 times faster and 50% saturation of the erythrocytes with oxygen takes 0.08-0.09 sec [10]. This rate is quite sufficient for normal saturation of the erythrocytes with oxygen, and changes in it within certain limits will not affect this process.

The affinity of hemoglobin for oxygen is thus increased in young subjects during hyperoxia, and this is reflected in a shift of the oxyhemoglobin dissociation curve to the left, and the tissues are protected against an excessive increase in pO_2, which could have serious consequences. In elderly subjects the affinity of hemoglobin for oxygen under standard conditions does not increase significantly, and under real conditions it does not change at all.

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POSTSTRESS RIGIDITY IN THE LEFT VENTRICULAR MYOCARDIUM

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Under the influence of emotional-painful stress (EPS) a unique syndrone of poststress rigidity has been shown to develop in the arterial myocardium: the atrial muscle responds to an equal applied load by an increase in length which is less by 33-50% than in the control; this is accompanied by depression of the tension developed by the atrium during isometric contraction [1, 8]. The question of the importance of this phenomenon for cardiac function remains unanswered, for it was not known whether poststress rigidity and the accompanying disturbances of contractile function are realized in the mycardium of the ventricles of the heart, which play the decisive role in its pumping functions.

The aim of this investigation was to study the extensibility and contractile function of the papillary muscles of the left ventricle of animals exposed to stress and to compare the disturbances found with those observed in the atrial myocardium.

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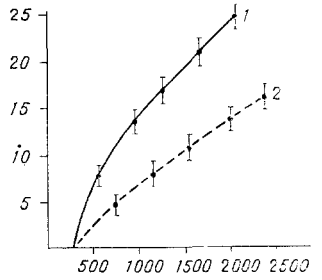

Fig. 1

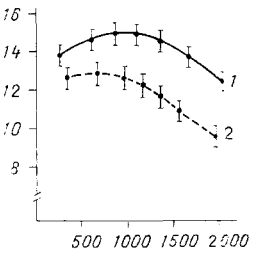

Fig. 2

Fig. 1. Effect of EPS on extensibility of isolated papillary muscle: 1) control; 2) EPS. Abscissa, magnitude of stretching load (in mg/mm^2); ordinate, increase in length of the muscle (in % of initial length).

Fig. 2. Effect of EPS on amplitude of contractions of isolated papillary muscle: 1) control; 2) EPS. Abscissa, magnitude of stretching load (in mg/mm^2); ordinate, amplitude of contractions (in % of initial length of muscle).

EXPERIMENTAL METHOD

Experiments were carried out on male Wistar rats weighing 150-200 g. EPS was produced in the form of an anxiety neurosis [9] once only, in the course of 6 h. The rats were decapitated 2 h after the end of exposure to stress. The posterior papillary muscle of the left ventricle was isolated and placed in a constant-temperature chamber with Krebs–Henseleit solution (95% O_2, 5% CO_2, pH 7.4, 29°C) containing 5.5 mM glucose. The muscles contracted under isotonic conditions for 1 h in response to electrical stimulation with a frequency of 20 Hz and with a load of 150 mg; the muscle was then gradually stretched by increasing the load to 1500 mg. Since the muscles differed in thickness, the stretching load was calculated as the ratio of the load to the cross-section of the muscle. Extensibility of the muscle was assessed as the increase in its length in response to an increase in load every 250 mg; the length of the muscle was expressed as a percentage of its initial length, i.e., its length when carrying a load of 250 mg. The length of the muscle was recorded by measuring the displacement of the lever of the instrument used to record isotonic contractions, to which the muscle was fixed. This displacement was measured by a capacitive transducer and recorded on a "Disa" indicator by means of a "Crossor" camera, by the method described previously [4]. The contractile function of the muscle was evaluated and the amplitude of contraction, calculated as the ratio between the amount of shortening of the muscle and its initial length, in percent.

EXPERIMENTAL RESULTS

Exposure to EPS led to a decrease in extensibility of the papillary muscles. As Fig. 1 shows, in rats exposed to stress the increase in the length of the muscle in response to a single stretching load was on average 33-50% less than in the control. For example, whereas the length of the muscle in the control rats in response to a load of 100 mg/mm^2 increased by 14% of its initial length, in rats exposed to stress it increased by 6.5%, i.e., by about half as much.

The decrease in extensibility of the myocardium in these animals was combined with depression of the amplitude of contractions. For instance, Fig. 2 shows that in rats exposed to stress the amplitude of contractions was reduced on average by 18-24% compared with the control. Moreover, increases in the amplitude of muscular contraction during stretching and flattening out on a plateau in rats exposed to EPS were about 60% less than in the control.

It can thus be concluded from these results that the decrease in extensibility and impairment of realization of the Frank–Starling mechanism are typical manifestations of post-stress injury to the myocardium both in the atria and in the ventricles.

When the probable mechanism of this phenomenon is considered, the idea put forward previously, that during diastole the residual actomyosin cross-linkages are preserved in the myofibrils, and these may affect the resting tension, must be borne in mind [5, 12]. An in-

crease in the number of "residual" cross-linkages, whatever the cause, must evidently lead to an increase in the resting tension and to a decrease in extensibility of the heart muscle in diastole. Disconnection of the actomyosin cross-linkages that have been formed, i.e., for the purpose of relaxation, requires two basic conditions: first, the supply of ATP, synthesized in the mitochondria and the glycolysis system, to the myofibrils and second, removal of calcium from the myofibrils by the Ca-pumps of the sarcoplasmic reticulum (SPR) and sarcolemma [10, 12]. In EPS injury to the membranes of SPR [3], the sarcolemma [6], and the mitochondria [2] and also a decrease in the glycogen reserves and in its synthesis [7] in the myocardium have been demonstrated. It can be postulated that to some degree this disturbs ATP transport into the sarcomeres and the removal of calcium from them. As a result the number of "residual" actomyosin cross-linkages increases and the decrease in extensibility of the myocardium in animals exposed to EPS, revealed by this investigation, takes place. This hypothesis also explains the second fact which was discovered, namely depression of the force of contraction. This depression is possibly associated with the fact that an excess of "residual" cross-linkages, existing in diastole, leads to a decrease in the number of free active centers forming new cross-linkages during diastole in the myofibrils and, as a result of this, to a decrease in the developed tension. The physiological importance of the phenomena discovered is that as a result of a decrease in extensibility of the myocardium, the diastolic volume may be reduced, the diastolic pressure raised, and ventricular filling disturbed. Together with the reduction in the force of contraction, this situation evidently leads to the disturbance of the pumping function of the heart as a whole and it may play the principal role in the development of heart failure in stress situations.

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