INVESTIGATION OF BREATHING RHYTHM IN RATS UNDER ACUTE EMOTIONAL STRESS

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KEY WORDS: irregularity of the rhythm of breathing; pneumogram; emotional stress; negative emotional tension.

The rhythm of breathing is a sensitive somatovegetative parameter which reflects changes in the state of the bodily functions [2, 3, 9]. The degree of respiratory arrhythmia has been shown to reflect quantitatively the intensity of motivations and emotional reactions [4, 5]. In particular, the rhythm of breathing is substantially disturbed during negative emotional strain [1, 6, 8, 10]. However, in the investigations cited, attention was concentrated mainly on changes in the frequency and depth of respiration, and for that reason correlation between the intensity of negative emotional tension and the degree of irregularity of the respiratory rhythm has remained unstudied. The investigation described below was undertaken to study this problem.

A model of acute emotional stress in rats during stochastic stimulation of the ventromedial hypothalamus and electrodermal stimulation (EDS) [7, 11] was chosen as the experimental model. The irregularity of the rhythm of breathing was analyzed quantitatively [4].

EXPERIMENTAL METHOD

Experiments were carried out on 11 male Wistar rats weighing 250-300 g. The animals were scalped, and a metal plate, by means of which the animal's head could be fixed in a stereotaxic apparatus without the use of ear clips, was secured to the skull with "Norakril" glue. To create a conflict situation the rats were placed in a small Plexiglas box, restraining their movements. Electrical stimulation of the ventromedial hypothalamic nucleus (VMH) was applied to the fixed animals, alternately with EDS. Stimulation of VMH and EDS continued for 3 h according to a specially devised stochastic scheme with an alternating current of threshold values, with voltage 1.4-6 V, frequency 50 Hz, and pulse duration 1 msec. The order of stimulation was determined in accordance with the law of random numbers. The duration of each stimulation of VMH and EDS was 30 sec and 1 min. Threshold voltages of the

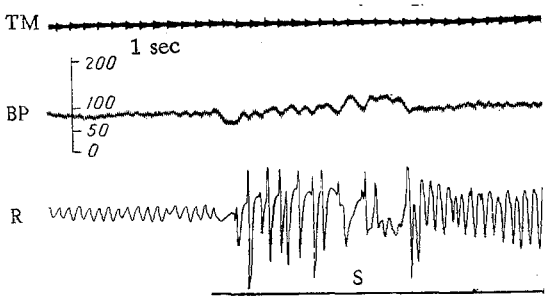

Fig. 1. BP and pneumogram during EDS in an animal resistant to emotional stress. TM) Time marker, BP) arterial pressure (scale graduated in mm Hg), R) respiration, S) period of stimulation of hypothalamus.

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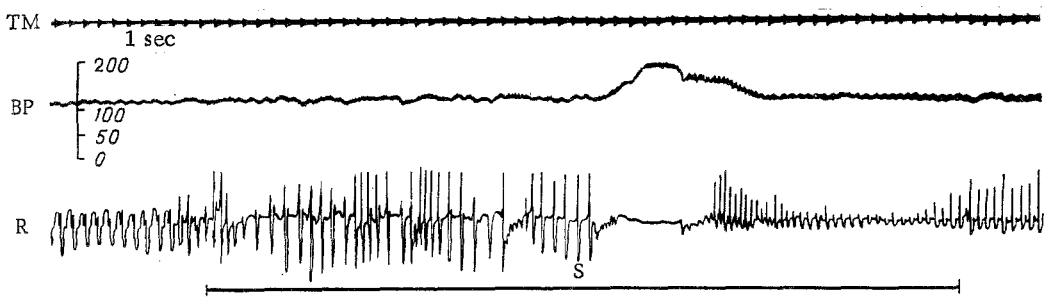

Fig. 2. BP and pneumogram during stimulation of VMH in animal predisposed to emotional stress. Legend as in Fig. 1.

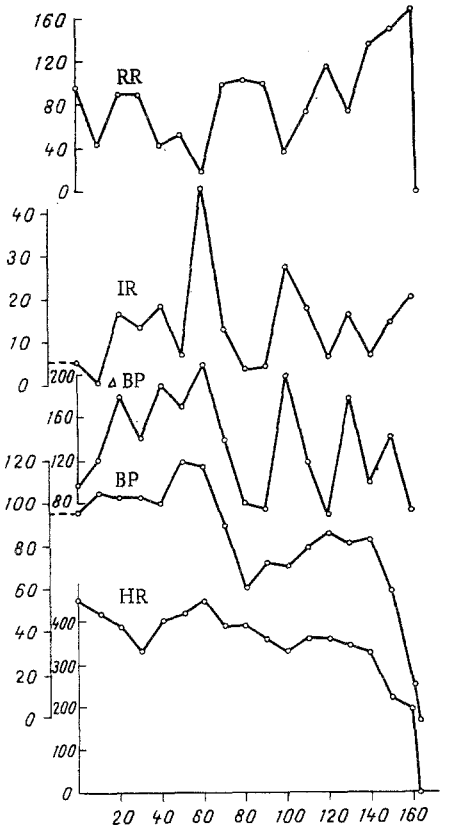

Fig. 3. Dynamics of changes in IR, RR, and relative increase in BP (ΔBP) during stimulation of VMH, and of BP and HR in pauses between stimulations, in rat predisposed to emotional stress during implementation of stress stimulation program. Abscissa, time (in min); ordinate, IR (in %), RR (cycles/min), ΔBP (in %), BP (in mm Hg), and HR (beats/min).

current used to stimulate VMH and for EDS were chosen depending on the change in blood pressure (BP) produced, which was not less than 20% of its initial level.

BP of the rats was recorded in the descending aorta through a catheter inserted into the caudal artery, by means of a transducer (Nihon Kohden, Japan). During the experiments both the general BP level and the change in BP to each episodic stimulation of VMH and EDS were recorded. Throughout the experiment also, the pneumogram of the rats and changes in respiration in response to each episodic stimulation of VMH and EDS were recorded continuously by means of a thermistor transducer, fixed in front of the animal's nose, and the ECG was recorded in standard lead II, by means of needle electrodes inserted subcutaneously into the animal's limb. The parameters chosen for testing were recorded on a polygraph (Nihon Kohden). BP, the heart rate (HR), the respiration rate (RR), and the irregularity of the rhythm of breathing (IR) were then determined in the control and every 10 min during the experiment. The parameters of breathing were calculated in time intervals of 24 sec. The degree of IR was calculated by the equation [4, 5]:

$$IR = \frac{1}{n-1} \sum_{i=1}^{n-1} \frac{|T_i - T_{i+1}|}{T_i + T_{i+1}} \cdot 100\%,$$

where T_i is the duration of the i-th respiratory wave, i the serial number of the respiratory wave in a series of respiratory cycles, and n the number of respiratory waves studied.

EXPERIMENTAL RESULTS

Before the experiment began the value of RR differed in each individual animal, but breathing was virtually uniform. Six rats survived the 3-h stress program (they were called "resistant") but five animals died at different stages of stress (they were called "predisposed" to acute emotional stress). BP, HR, RR, and IR changed in the resistant animals only during stimulation, and in the pauses between stimulations the changes were not significant (Fig. 1). During episodic stimulations of VMH and during EDS, BP and IR increased whereas HR and RR changed in opposite directions. The maximal value of IR during stimulation of VMH was 22 ± 3%.

In animals predisposed to emotional stress (i.e., in rats which died at different stages of stress) HR and IR did not change significantly in intervals between stimulations (Fig. 2), but BP and HR fell gradually, and in animals which died it fell to zero (Fig. 3). During EDS and stimulation of VMH the character of changes in the somatovegetative parameters was similar to changes in the analogous parameters in resistant animals. A distinguishing feature of the predisposed animals was that during stimulation of VMH reversible respiratory arrest developed, but only at times of a considerable rise of BP (Fig. 2). Under these circumstances breathing became much more irregular. The maximal duration of respiratory arrest in the different animals was 16 ± 3 sec, and the corresponding value of IR was 38 ± 9%. After a marked increase in IR, further stimulation of VMH was not accompanied by any significant episodes of respiratory arrest and rise of BP until death supervened.

The degree of arrhythmia of respiration, one of the objective physiological parameters of respiration, thus depends on the degree of negative emotional strain and correlates with cardiovascular changes in the body during acute emotional stress. The most marked respiratory arrhythmia was observed during stimulation of the hypothalamic emotiogenic centers, which may be connected with the spread of excitation from VMH to the respiratory center in the medulla. In animals which died during the stress program during VMH stimulation, transient episodes of respiratory arrest accompanied by considerable rises of BP were recorded, and under these circumstances the irregularity of respiration increased to 38 ± 9%. The intensity of the hemodynamic changes combined with the increase in irregularity of respiration during stress-inducing stimulation is an unfavorable sign in animals exposed to a stress situation.

The results of this investigation showed that animals differ in individual resistance of their cardiovascular and respiratory functions to emotional stress. Vascular reactivity and disturbance of the respiratory rhythm during episodic stimulation of VMH and also during EDS in the course of emotional stress can be used as criteria of resistance or predisposition of the animal to acute emotional stress.

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THE "NO REFLOW" PHENOMENON IN THE CEREBRAL CORTEX IN THE EARLY POSTISCHEMIC PERIOD

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During the first minutes of the resuscitation period after total profund cerebral or general ischemia the volume blood flow of the brain tissue rises sharply [3, 4]. Meanwhile investigations have demonstrated local postischemic hypoperfusion of the cerebral cortex as the earliest manifestation of cerebrovascular disturbance [7, 9]. The contradictory nature of these data and the absence of agreement on the nature of the phenomenon of occlusion of the cerebral vessels in the early postischemic period motivated a morphological and functional investigation of the microcirculation of the cerebral cortex during ischemia and in the early recovery period.

EXPERIMENTAL METHOD

Experiments were carried out on 50 male albino rats weighing 180-220 g anesthetized with ether. Ischemia was produced by a model of clinical death lasting 5 min from blood loss through the external iliac artery followed by resuscitation. Material for analysis was taken from the experimental animals at the end of clinical death and 5, 10, and 30 min after resuscitation. Ten animals served as controls.

The cerebral vessels were injected with gelatin medium with high-grade purified Mark PM-50 soot. Perfusion was carried out intravitally at 37°C under a pressure of 120 mm Hg for 20 min. The brain, perfused with ink, was fixed in 96° alcohol and embedded in celloidin. The angioarchitectonics of the cortex and deep brain formations was studied in cleared preparations 200 μm thick. The vascular network of the sensomotor cortex was subjected to morphometric analysis: The density of the vascular network was determined [1] in sections 20 μm thick and the internal diameter of the vessels measured. Filling of the vessels with blood was studied by a histochemical method of hemoglobin determination [8]. For ultrastructural analysis the cortex was fixed by perfusion with 2.5% glutaraldehyde and immersion in the same fluid and postfixation in 1% OsO_4. Oriented pieces of neocortex were embedded in Epon-812. Ultrathin sections were stained with uranyl acetate and lead citrate and examined in the EVN-100LM electron microscope. The numerical results were subjected to statistical analysis by Student's test.

EXPERIMENTAL RESULTS

At the end of ischemia a small decrease in density of the vascular network was found in the cerebral cortex (Fig. 1). The mean diameter of the vessels increased to 7.02 ± 0.47 μm compared with 5.12 ± 0.12 μm in the control ($P < 0,01$). Some microvessels were highly constricted, others extremely dilated, i.e., there was a wide range of variations of their diameter. The residual content of blood in the vessels was minimal.

Evidence of slight swelling of the pericapillary astroglia, and moderate translucency of the cytoplasm of some and hyperchromia of other endotheliocytes of the blood capillaries were observed electron-microscopically during ischemia.

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