EFFECT OF INHALING COLD AIR ON AUTONOMIC FUNCTIONS AND ON THE STATE OF THE CENTRAL NERVOUS SYSTEM IN RABBITS WITH HYPERTHERMIA

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Experiments on rabbits showed a decrease in amplitude of the EEG waves and of excitability and reactivity of the cerebral cortex as well as a sharp increase in the respiration and heart rates during hyperthermia. Inhalation of cold air, at a temperature of between −10 and −20° by such animals through a mask caused rapid (within 1-5 min) recovery of cortical excitability and reactivity and of the respiration rate, and a gradual (within 60-80 min) recovery of amplitude of the EEG. These results confirmed the view that afferent impulses play an essential role in the development of disturbances of the regulatory function of the CNS in hyperthermia and also the view that the thermal resistance of the body can be increased by the local action of cold.

The writers' previous investigations [2, 9], suggested that an excessive flow of afferent impulses from peripheral heat receptors plays an important role in the mechanism of disturbances which develop during hyperthermia and which determine the limits of tolerance to overheating.

Having regard to the possible tonic action of afferent stimuli from cold thermoreceptors [3, 5, 6] on the central nervous system, the investigation described below was carried out to study the effect of inhalation of cold air on the autonomic functions and state of the central nervous system during hyperthermia.

EXPERIMENTAL METHOD

In the experiments of series I, 7 rabbits were exposed for 2-2.5 h to hyperthermia at a temperature of 40-45°C and a relative humidity of 20-30%. Next, against this background of severe hyperthermia, air at a temperature of between −10 and −20°C was introduced for 60-80 min into the mask through which the rabbit breathed (volume velocity 30-35 liters/min). In the experiments of series II, 7 rabbits inhaled cold air for the first 1.5-2 h of their exposure to hot and humid conditions, and thereafter inhaled air at a temperature of 40-45° C. In the course of the experiment the rectal temperature, respiration rate, pulse rate, systolic arterial pressure, and electrical activity of the occipital and parietal lobes of the brain were recorded. The respiratory excursions of the chest were recorded by means of a carbon powder rheostat transducer [1]. To determine the heart rate the ECG was recorded in standard lead II. The systolic blood pressure was measured by a bloodless method in the animals's forelimb [7]. The EEG of the occipital and parietal regions were recorded by monopolar leads. Livanov's method of reactivity curves [4] was used to determine cortical excitability and reactivity.

EXPERIMENTAL RESULTS

Exposure to heat caused an increase in body temperature on the average by 2.5°C, a sharp increase in the respiration and heart rate, a decrease in the systolic blood pressure, and a decrease in cortical electrical activity, excitability, and reactivity. Evidence of the decrease in cortical excitability and reac-

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TABLE 1. Effect of Inhalation of Cold Air on Autonomic Functions and State of the Cerebral Cortex of Rabbits (M±m)

Indices	initial data	Experiments of series I					
		before inhalation of cold air	inhalation of air at a temperature of between −10 and −20°C				
			1 min	5 min	10 min	60 min	
Rectal temperature (in deg)	38.6±0.14	41.1±0.19	41.2±0.13	41.2±0.21	41.2±0.26	39.7±0.20	
Respiration rate (per min)	54±6	306±18	217±23	211±20	186±19	139±12	
Heart rate (beats per min)	252±10	294±11	344±13	332±9	328±10	330±12	
Systolic blood pressure (in mm Hg)	112±4	83±8	–	119±9	115±9	118±10	
Mean amplitude of biopotentials (in μV):							
in occipital cortex	125±8	63±5	121±12	82±8	75±6	106±11	
in parietal cortex	116±6	70±8	120±12	103±9	80±7	113±10	
Bioelectrical cortical response to repetitive photic stimulation of increasing intensity:							
latent period (in sec)	20.2±0.33	23.5±0.42	–	19.5±0.31	20.3±0.30	21.1±0.44	
duration (in sec)	8.0±0.25	4.1±0.53	–	8.5±0.37	8.2±0.42	7.5±0.45	
amplitude of biopotentials during response (in μV)	190±18	156±12	–	160±19	172±21	187±19	

Indices	Experiments of series II				
	initial data	before inhalation of warm air	inhalation of air at a temperature of 40–45°C		
			1 min	5 min	10 min
Rectal temperature (in deg)	38.4±0.11	39.4±0.16	39.4±0.15	39.6±0.12	39.7±0.13
Respiration rate (per min)	62±5	116±18	288±14	365±15	380±19
Heart rate (beats per min)	259±7	270±9	314±12	348±8	341±10
Systolic blood pressure (in mm Hg)	121±5	130±7	–	89±4	98±7
Mean amplitude of biopotentials (in μV):					
in occipital cortex	117±4	123±15	109±12	106±10	99±8
in parietal cortex	122±5	111±6	97±8	93±9	89±7
Bioelectrical cortical response to repetitive photic stimulation of increasing intensity:					
latent period (in sec)	19.8±0.17	20.1±0.36	–	20.6±0.41	20.8±0.42
duration (in sec)	8.2±0.21	8.5±0.37	–	7.9±0.44	7.6±0.38
amplitude of biopotentials during response (in μV):	157±12	150±9	–	139±14	130±12

tivity was given by an increase in the latent period of the bioelectrical response to repetitive photic stimulation of increasing intensity, and a decrease in its duration and in the amplitude of the potentials (Table 1, experiments of series I).

Inhalation of cold air produced an immediate slowing of respiration and an increase in the heart rate. The rectal temperature remained at its maximal level for a further 5-10 min, and then gradually fell, while the systolic blood pressure rose after inhalation of cold air for 4-6 min, and the latent period of the cortical electrical response to photic stimulation was shortened and the duration of the response itself was increased (Table 1, Fig. 1). The amplitude of the cortical potentials increased during the first few

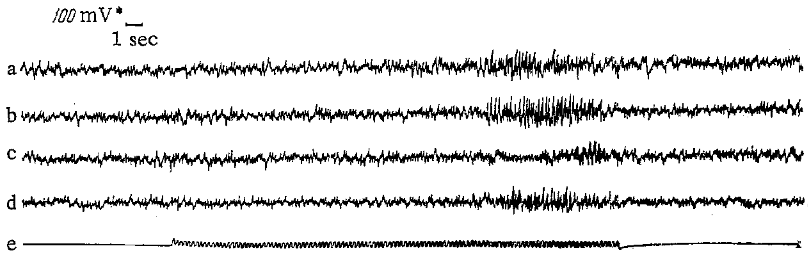

Fig. 1. Changes in occipital biopotentials of a rabbit during repetitive photic stimulation of increasing intensity: a) normal, rectal temperature 38.2°C; b) rectal temperature 40.8 °C; c) rectal temperature 41.1°C; d) after inhalation of cold air for 5 min at a rectal temperature of 41.5°C; e) marker of photic stimulation.

minutes of inhalation of cold air, but it then began to fall again. It did not return to its initial level until the 60th minute of inhalation of cold air.

In the experiments of series II, no marked changes in the functional state of the cardiovascular and central nervous systems were observed until the animal was changed over from breathing cold to breathing warmed air. The rectal temperature then rose by 1°C and the respiration rate increased to 116 per min.

Inhalation of warmed air caused an immediate and sharp increase in the respiration rate (which was more than doubled) and heart rate, a decrease in cortical electrical activity, and a fall of blood pressure. No definite changes in cortical excitability and reactivity could be detected (Table 1).

Hence, inhalation of cold air during simultaneous exposure to a high external environmental temperature can prevent the development of disturbances due to hyperthermia in animals. Changing over to inhalation of warm air causes an immediate and considerable increase in the respiration rate, a fall of blood pressure, etc. This fact is evidence of the special role of afferent impulses from thermoreceptors in the development of disturbances of the regulatory function of the central nervous system during hyperthermia.

During inhalation of cold air, information from the cold receptors of the respiratory tract helps to restore the normal functions of the body. This is shown by the fact that the change to inhalation of cold air immediately evokes responses aimed at restoring the normal activity of vitally important systems of the body. Since the possible heat emission via the respiratory organs is extremely limited [8, 10], and the rectal temperature did not fall during the first minutes of inhalation of cold air, it can be considered that the role of a change in the heat balance of the body in the manifestation of the initial positive effect is less important.

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