ELECTROENCEPHALOGRAPHIC CHANGES IN HYPO- AND HYPERTENSIVE RESPONSES TO HYPOTHALAMIC STIMULATION

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Changes in cortical and subcortical electrical activity during hypo- and hypertensive vascular responses to electrical stimulation of the hypothalamus were investigated before and after injection of the monoamine oxidase inhibitor vetrazin, in experiments on rabbits anesthetized with urethane. Vetrazin completely blocked the hypertensive response and its electroencephalographic manifestation but had no effect on hypotensive hypothalamic responses. The results are interpreted from the standpoint of differences in the neurochemical mechanisms of hypothalamic pressor and depressor responses.

KEY WORDS: blood pressure; hypothalamus; monoamine oxidase inhibitor vetrazin.

Stimulation of various hypothalamic structures. such as the ventromedial nucleus, the medial and lateral preoptic regions, etc., induces changes in the blood pressure [3, 4, 9, 10] and causes generalized changes in the electrical activity of various brain formations [4, 7, 9, 10].

The object of this investigation was to study the electroencephalographic changes accompanying hypo- and hypertensive vascular responses to stimulation of various parts of the hypothalamus. The monoamine oxidase inhibitor vetrazin [5] also was used in an attempt to discover whether the chemical mechanisms of the electroencephalographic response accompanying pressor and depressor vascular responses to hypothalamic stimulation are the same or different.

EXPERIMENTAL METHOD

Acute experiments were carried out on 20 rabbits anesthetized with urethane (1 g/kg body weight intravenously). Urethane was chosen deliberately as the anesthetic. Previous investigations in the writer's laboratory showed that urethane selectively blocks the waking state, but has virtually no action on ascending activating influences of the hypothalamus on the cortex [1, 7].

The stimulating electrodes were inserted into the preoptic region and into the ventromedial nucleus of the hypothalamus. The blood pressure, heart rate, and the EEG of the cortex and subcortical formations (septum, amygdala, hypothalamus, mesencephalic reticular formation) were recorded.

Parameters of stimulation which induced a pressor or depressor response exceeding the original blood pressure by 20-25% were as follows: a) for pressor points 1.5 V, 50 Hz, 0.1 msec, 3 sec; b) for depressor points 7 V, 8 Hz, 0.1 msec, 10 sec.

Vetrazin was injected into the auricular vein of the animal in a dose of 20 mg/kg body weight, in aqueous solution, at the rate of 1 ml/min. The hypothalamus was stimulated before and 30 min after injection of vetrazin. After each experiment the animal's brain was removed from the skull and fixed in formalin solution. Sections were cut on a freezing microtome to a thickness of 100-120 μ. The location of the electrode tips in the brain was determined by reference to brain atlases by the projection method.

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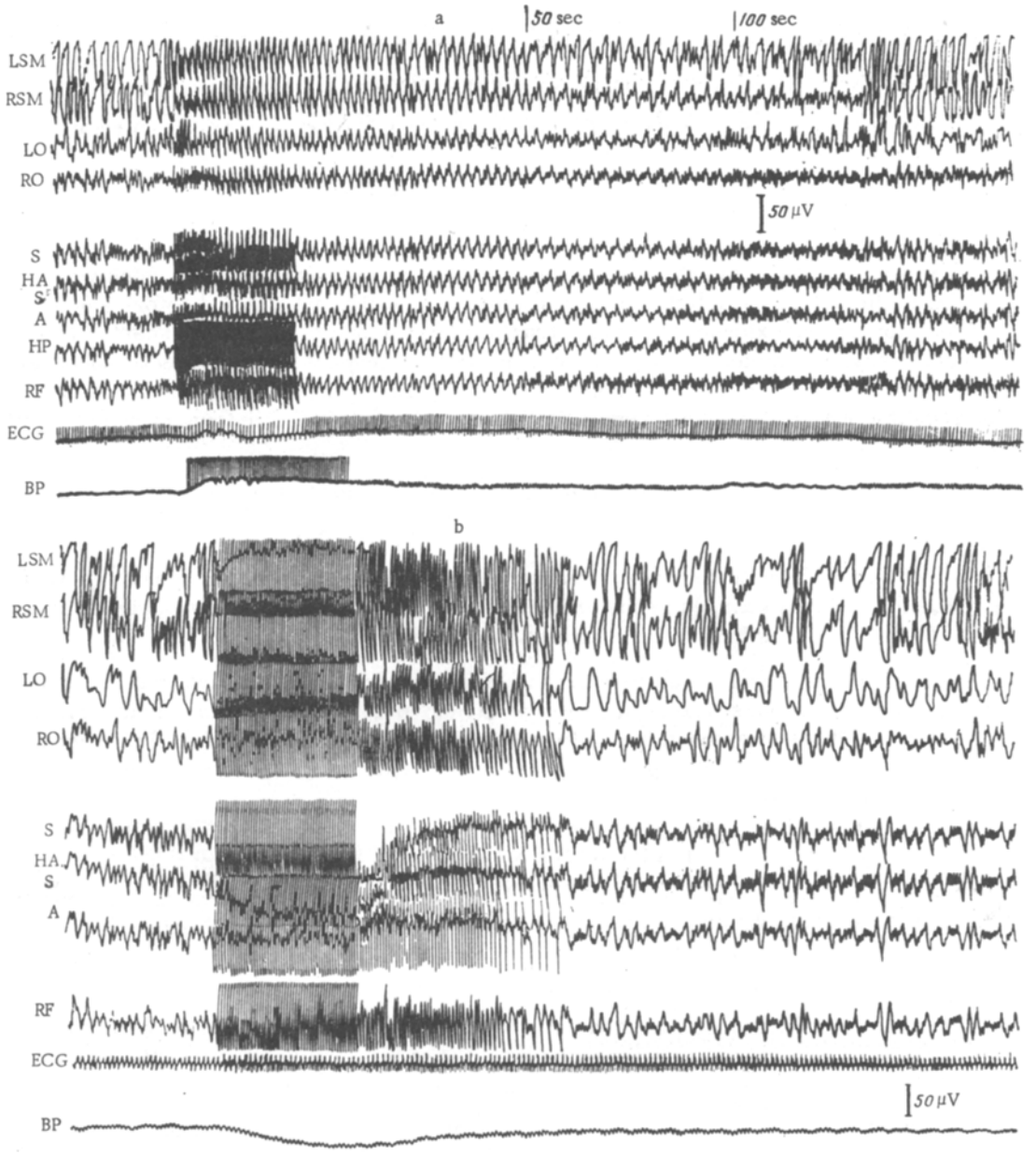

Fig. 1. EEG changes accompanying pressor (a) and depressor hypothalamic responses (b). From top to bottom: LSM) left sensomotor cortex; RSM) right sensomotor cortex; LO) left occipital cortex; RO) right occipital cortex; S) septum; HA) anterior hypothalamus; HP) posterior hypothalamus; A) amygdala; RF) mesencephalic reticular formation; ECG) electrocardiogram; BP) blood pressure.

EXPERIMENTAL RESULTS AND DISCUSSION

In all experiments stimulation of the ventromedial hypothalamic nucleus caused a rise of arterial pressure, whereas stimulation of the preoptic region caused the pressure to fall. The hypertensive response was sometimes accompanied by bradycardia during and immediately after the end of stimulation. A fall in heart rate was also observed during stimulation of the preoptic region of the hypothalamus.

Stimulation of the hypothalamic pressor points led to after-changes in brain electrical activity. In 78% of cases regular, high-amplitude (150 μV) pointed waves with a frequency of 4-7 Hz were observed. In 22% of cases a special kind of high-amplitude rhythm was recorded with a volley at the peak.

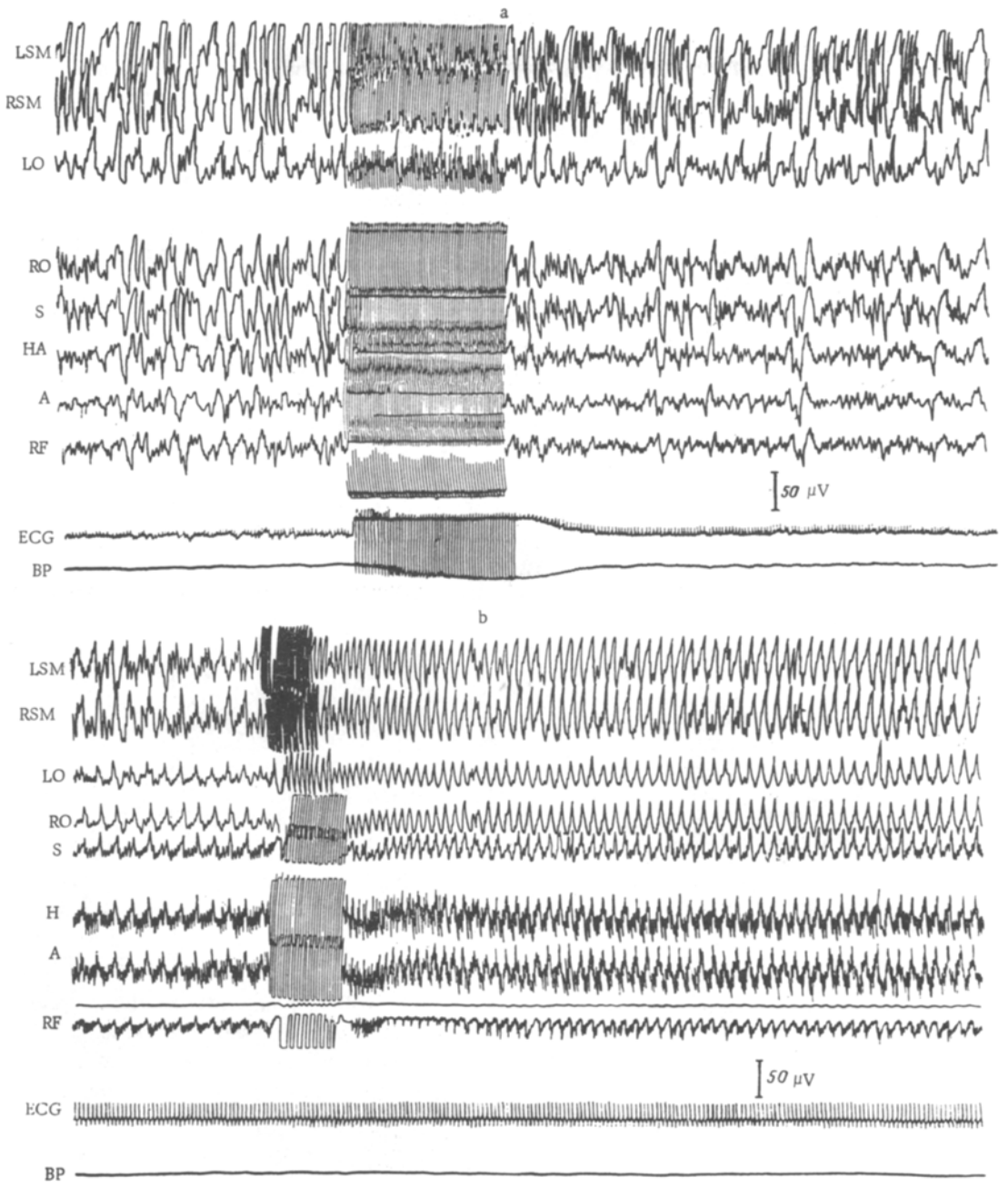

Fig. 2. Changes in EEG and depressor (a) and pressor (b) hypothalamic responses after injection of vetrazin. Legend as in Fig. 1.

During stimulation of the preoptic region evoked depressor responses in 17% of cases were accompanied by brief low-frequency desynchronization of the EEG, which was soon replaced by the original rhythm and was most marked in the occipital recordings. In 33% of cases immediately after the end of stimulation an epileptiform rhythm was recorded. In 50% of cases no visible changes were seen in the EEG in response to stimulation of the preoptic region (Fig. 1). Changes which were observed on the EEG during stimulation of the pressor points were generalized, affecting the whole cortex and subcortical formations, and they lasted much longer than the changes observed during depressor responses.

During stimulation of the preoptic region clearer changes were observed in the cortical than in the subcortical recordings of the EEG.

Injection of vetrazin increased the frequency of the EEG rhythm. Vetrazin completely blocked the pressor vascular response but had no appreciable effect on the depressor response (Fig. 2). Vetrazin is known to be a

monoamine oxidase inhibitor and to block α-adrenergic receptors [6]. It can accordingly be considered that the autonomic and EEG phenomena accompanying the pressor response are based mainly on adrenergic mechanisms, whereas the depressor responses are based on a different neurochemical mechanism. It follows from the work of Val'dman et al. [2] and of Teplov and Vasil'eva [8] that the electroencephalographic and autonomic manifestations of depressor responses evoked by stimulation of the hypothalamic nuclei have a predominantly cholinergic mechanism.

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EFFECT OF A HIGH-VOLTAGE CAPACITOR DISCHARGE ON OPTICAL PROPERTIES OF FROG HEART MUSCLE

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A high-voltage capacitor discharge caused a sharp increase in the intensity of flux of plane-polarized light passing through a strip of frog heart muscle. This may indicate changes in the optical properties of the tissue due to conformational changes in the membrane proteins.

KEY WORDS: high-voltage discharge; heart; optical properties.

Experiments on a cell membrane model based on the use of frog skin showed that an electric discharge induces changes in its transmembrane potential, in the parameters of the volt–ampere characteristic curve, and in permeability to sodium, potassium, and calcium ions [1-3].

All these changes are evidently a reflection of momentary structural changes in the cell membranes at the time of the electric discharge.

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