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Short Communication

The Influence of Stimulation of Limbic Structures on Motivational Behaviors and Memory

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Abstract

The vegetative, somatic and behavioral effects of electrical and chemical stimulation of various structures of t limbic system generally support and supplement the data obtained by damaging them. The contradictory fac often encountered in the literature can be explained in most cases by certain shortcomings of the method of dire stimulation (especially electrical) of brain structures.

Key Words: limbic structures; motivational behavior; memory

Introduction

The vegetative, somatic and behavioral effects of electrical and chemical stimulation of various structures of the limbic system generally support and supplement the data obtained by damaging them. The contradictory facts often encountered in the literature can be explained in most cases by certain shortcomings of the method of direct stimulation (especially electrical) of brain structures [9]. Due to the small size of the anatomically isolated nuclei of such structures as the hypothalamus, amygdala, septum, thalamus, etc, their selective activation becomes difficult, since the stimulating current also spreads to neighboring areas, as a result of which it is difficult to correlate the obtained effect with the excitation of a specific nervous structure. No less difficult in applying this approach are the functional and structural features of the nuclear formations themselves [22]. Neurons with different functional properties, participating in the formation of various motivational phenomena, are often localized in the same nuclei. Electrical stimuli can also affect the conduction pathways of very distant structures, which again complicates the picture [18]. In this regard, the method of chemical stimulation has proven to be more successful, since at certain doses physiologically active substances (mediators of synaptic transmission), firstly, may not involve the conducting pathways in excitation, and, secondly, may selectively excite those nerve populations that have the corresponding synaptic chemosensitivity [31].

The effects of electrical stimulation of various hypothalamic nuclei confirm the position that the regulation of food and drinking motivation is carried out by the interaction of the lateral and ventromedial areas of the hypothalamus [26]. Electrical stimulation of the lateral hypothalamus can cause activation of both food and drinking behavior even in a previously satiated animal, whereas electrical stimulation of the ventromedial hypothalamus causes inhibition of food and drinking. There is an experiment where an injection of norepinephrine into the lateral area of the hypothalamus in rats caused activation of food behavior, and an injection of carbachol caused activation of drinking behavior [12].

There are data in the literature on the activation of drinking and eating in response to electrical and chemical stimulation of extrahypothalamic structures of the limbic system [10]. It was subsequently discovered that stimulation of the ventromedial area of the tegmentum of Tsai in cats causes a food reaction. In this regard, it is interesting to note that powerful projections of the lateral hypothalamus were found in this area, and it can be assumed that the activation of food behavior may be the result of antidromic stimulation of neurons of the "food center" [2].

In terms of identifying the organization of neurophysiological mechanisms of eating behavior, particularly interesting data were obtained with electrical and chemical stimulation of the amygdala. In particular, it was shown that stimulation of the basolateral region of the amygdala causes inhibition of eating behavior, and stimulation of the dorsomedial part causes its activation [17]. The inhibitory effect of the basolateral amygdala on eating reactions is realized through the ventromedial hypothalamus. This is indicated by an increase in the activity of neurons of this hypothalamic nucleus during stimulation of the lateral nucleus of the amygdale [23]. The facilitating effect of the dorsomedial amygdala on eating behavior can be realized either through activation of the lateral hypothalamus or through inhibition of the ventromedial hypothalamus, as a result of which the lateral hypothalamus is released from the inhibitory effect of the ventromedial nucleus [6].

Stimulation of the hippocampus has little effect on eating and drinking behavior. Only isolated cases have been described where activation of eating behavior occurred not in response to stimulation, but in response to the cessation of hippocampal stimulation [7]. This fact is interpreted as a rebound after the cessation of the inhibitory influence of the hippocampus

caused by its stimulation. This seems to be consistent with the data obtained with electrical stimulation and damage to the septum, through which the hippocampal influence on the hypothalamus can be realized. Stimulation of the septum caused a decrease in water drinking in rats, and its damage caused polydipsia [19].

Experiments have shown that there are two functionally distinct zones in the hypothalamus for the implementation of vegetative and somatic reactions [11]. The first, dynamic zone, covers the posterior region of the hypothalamus, the stimulation of which causes symptoms characteristic of the activation of the sympathetic nervous system, and this zone can be considered an ergotropic system. The second, trophotropic zone, the stimulation of which causes symptoms characteristic of the activation of the parasympathetic nervous system, is located in the preoptic region and the anterior hypothalamus [8]. These two zones are in a reciprocal relationship. The work of other researchers has basically confirmed the data that the hypothalamus is the most important formation regulating the vegetative functions of the body [29].

Vegetative and somatic shifts that occur with electrical stimulation of the hypothalamus are components of emotional reactions. It has been shown that stimulation of the hypothalamus can cause a reaction of rage in a cat under light anesthesia [1]. Later, these data were confirmed on non-anesthetized cats. Subsequently, emotional reactions of a defensive type in response to electrical stimulation of the hypothalamus were obtained by many authors [30].

At present, it can be considered reliably established that emotional reactions of the aggressive-defensive type are regulated by the interaction of the ventromedial and lateral areas of the hypothalamus, while emotional reactions of the type of anxiety, attention, worry, etc., are regulated mainly by the posterior hypothalamus. However, in both cases, the participation of extensive structures of the mesodiencephalon is important [34].

At first glance, it seems paradoxical that damage and irritation of the ventromedial hypothalamus produce the same effect - the development of aggression. As was indicated above, the effect of damage can be explained by an increase in the sensitivity of the central gray matter of the midbrain as a result of its deafferentation, since it has been shown that it is the ventromedial hypothalamus that has powerful projections to this formation [14]. This is confirmed by the fact that with electrical stimulation of the central gray matter, a pronounced reaction of rage develops. It has also been shown that damage to the central gray matter significantly blocks the reaction of rage in response to hypothalamic stimulation [16].

Affective behavioral responses can be elicited by electrical stimulation of the amygdala, and these responses are similar to those elicited by stimulation of the hypothalamus. It has been shown that the effects elicited by stimulation of the amygdala are the result of activation of hypothalamic mechanisms, and this influence is mediated through the ventral amygdalofugal pathway of Nauth [28].

Aggression and fear reactions can also be evoked by electrical stimulation of the dorsomedial thalamus. Simultaneous stimulation of the hypothalamus and dorsomedial thalamus causes effective facilitation of the hunting-type aggression reaction, indicating their functional relationship [4].

Electrical stimulation of the septum has yielded contradictory data by different authors, which can be explained by the presence of a topographic localization of the function in it. It has been shown that stimulation of the lateral part of the septum causes a decrease in heart rate, while stimulation of the medial part causes an increase in heart rate [5]. When stimulating the posterior septum in rats, blood pressure decreases, while stimulation of the diagonal bundle increases it. In response to stimulation of the medial septum, a decrease in emotional reactivity was also caused, and in response to stimulation of the lateral septum, an increase in it [20]. All this indicates that through the septum, the rostral structures of the limbic system can have both an inhibitory and a facilitating effect on the mesodiencephalic mechanisms of vegetative and emotional reactions [3].

Vague and contradictory effects were obtained with electrical stimulation of the cortical structures of the limbic system. This is understandable, since electrical stimulation can hardly initiate the normal functioning of such a complex and differentiated structure as the cortex [33]. Most likely, one can expect that electrical stimulation will upset the normal functioning of the mechanisms of the cortex and thereby cause its functional shutdown. The effects of electrical stimulation of the hippocampus have been studied most diligently [31]. In classical experiments, no noticeable changes in vegetative reactions were found with electrical stimulation of the cortical structures of the limbic system. These data were confirmed by others. However, others observed a decrease in respiration and heart rate in response to stimulation of the hippocampus [27]. The inhibitory effect of the hippocampus has been described in relation to the motor reaction caused by activation of the motor cortex, orienting, food and defensive conditioned reflexes, and an aggressive reaction caused by electrical stimulation of the hypothalamus. Based on this, it is believed that the hippocampus has an inhibitory effect on the motivational centers of the hypothalamus [13].

The phenomenon of self-stimulation was discovered by applying the method of electrical stimulation of limbic system structures [15]. It was initially noted that animals were often attracted to the corner of the experimental chamber where testing was performed. Then a special device was mounted, and if the animal could turn on the stimulation itself by pressing a pedal with its paw, it willingly began to stimulate its brain [21]. It was established that self-stimulation is observed if the stimulating electrodes are localized in the motivational structures of the limbic system. The frequency of self-stimulation determines the reinforcing or "rewarding" effect of a particular structure. Subsequently, a test with the choice of the site was developed as a more reliable indicator of the "reward" effect [35]. The highest reinforcing effect was observed when the electrodes were implanted in the lateral hypothalamus, interpeduncular tegmental nuclei, medial forebrain bundle and some subcortical nuclei. A comparatively low effect of self-stimulation is characteristic of the cortical structures of the limbic system [24].

In parallel with the experiments, it was discovered that when stimulating some structures of the limbic system, an effect opposite to self-stimulation is observed: animals begin to avoid the stimulation and learn to switch it off themselves. It is clear that such stimulation is unpleasant or aversive for the animal [32]. The most pronounced aversive effects are observed when stimulating the ventromedial hypothalamus, central gray matter, periventricular nuclei of the hypothalamus, etc. As indicated above, these areas are important in integrating emotional reactions of the aggressive-defensive type [25]. It has been shown that there is a kind of reciprocal relationship between positive and negative (aversive) structures, rewarding stimulation weakens the effect of stimulation of aversive areas, and vice versa. The method of self-stimulation and switching off stimulation most clearly proves the importance of limbic structures and their hierarchy in integrating motivational-emotional reactions [17].

Electrical stimulation of limbic structures can cause not only activation of innate (unconditioned) motivational-emotional reactions, but also acquired (conditioned) reactions [23]. Even before this, the phenomenon of activation of a conditioned reflex in response to electrical stimulation of subcortical structures was demonstrated. Later, this phenomenon was observed during stimulation of such structures of the limbic system as the hypothalamus, reticular formation, amygdala, etc [29]. In response to stimulation of these structures, animals perform complex instrumental learned acts to obtain food. In an experiment where norepinephrine and carbachol were injected into the lateral region of the hypothalamus, activation of not only eating and drinking behavior was observed, but also activation of previously learned instrumental movements to obtain food and drink [34]. These facts indicate that electrical and chemical stimulation of mimotivacional structures of the limbic system can cause activation of a memory trace.

As noted above, the hypothalamus has been shown to be a critical area for regulating the sleep-wake cycle by transecting the brainstem at various levels

[5]. The results obtained using electrical stimulation confirm this position. Both high- and low-frequency electrical stimulation of the preoptic area were found to induce synchronization of the electrocorticogram and behavioral sleep. In this regard, it is interesting to note the presence of powerful projections from the preoptic area to structures such as the raphe nuclei and orbital cortex, which are widely believed to be involved in organizing the slow-wave phase of sleep [19].

Opposite electroencephalographic (desynchronization) and behavioral (awakening) effects are produced by electrical stimulation of the posterior hypothalamus [2]. This suggests the existence of a reciprocal relationship between the anterior and posterior regions of the hypothalamus and the significance of this relationship in regulating the alternation of different phases of the wakefulness-sleep cycle [16]. In addition, an inhibitory effect of the preoptic area on neurons of the activating reticular formation of the midbrain has been demonstrated. In other experiments, synchronization of the electroencephalogram and the sleepy state developed in response to high-frequency electrical stimulation of the basomedial areas of the cerebral cortex [1]. All this indicates that there is a subsystem in the limbic system whose function is to regulate the wakefulness-sleep cycle.

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