# EFFECT OF STIMULATION OF THE HYPOTHALAMIC AND AMYGDALAR MOTIVATION CENTERS ON VESTIBULAR NYSTAGMUS

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Chronic experiments on rabbits with electrodes implanted into hypothalamic and amygdalar structures showed that weak electrical stimulation of the food center of the lateral hypothalamus, giving a food response to preliminary testing, induces marked facilitation of vestibular (especially rotatory) nystagmus, whereas stimulation of the ventromedial hypothalamic nucleus inhibits it. The effect of amygdalar stimulation was manifested more often as facilitation, but sometimes as inhibition of the nystagmus. The results are evidence of the modulating influence of the hypothalamic and amygdalar motivation centers on the vestibular system.

KEY WORDS: hypothalamus, food behavior; vestibular nystagmus.

Recent investigations have shown the importance of afferentation from interoceptors of the alimentary tract and the level of food excitation in the reflex regulation and functional tuning of the vestibular system [1, 2, 4, 5]. It was decided to investigate the effect of artificial stimulation of hypothalamic and amygdalar structures directly concerned with the formation of food motivations and with the regulation of food intake [6, 9, 10, 12, 13, 15, 17], on vestibulo-somatic reflexes [rotary (RN) and postrotatory (PRN) nystagmus]. Such an investigation would also clarify certain mechanisms of interaction between the vestibular system and the autonomic nervous system and, in particular, with the parasympathetic and sympathetic hypothalamic centers. This investigation was devoted to the study of these problems.

### EXPERIMENTAL METHOD

Chronic experiments were carried out on adult cats (2.8-3 kg) with electrodes (30-40  $\mu$  in glass insulation) implanted into the motivation centers of the lateral and ventromedial portions of the hypothalamus and the amygdalar complex based on coordinates of a stereotaxic atlas [19]. These brain structures were stimulated for 20-60 sec with square pulses (1-2.5 msec, 100 Hz, 1-5 V) by a monopolar technique. After preliminary testing of the behavioral responses the animals were fixed to the platform of an electric turntable and rotated in the horizontal plane up to an assigned angular velocity (180 deg/sec). After rotation at a uniform rate in a clockwise direction for 20-60 sec, the turntable was suddenly stopped ("stop stimulus"). RN and PRN were recorded on the 4-ÉÉG-I ink-writing electroencephalograph. The experiments were carried out in complete darkness. The corneo-retinal potential was recorded with needle electrodes inserted into the skin at the angles of the eye. The following parameters of RN and PRN were studied: duration (in sec), number of beats in the nystagmus response, the amplitude (in  $\mu$ V), the frequency (number of beats per second), and energy (in conventional units); the energy of nystagmus is the product of amplitude and frequency over a given period of time [3, 18].

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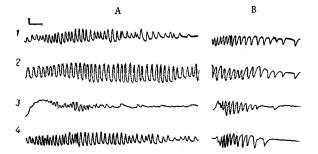


Fig. 1. Effect of hypothalamic and amygdalar stimulation on vestibular nystagmus in rabbits: A) RN; B) PRN; 1) initial nystagmus response; 2) response to electrical stimulation of food center of lateral hypothalamus; 3) of ventromedial hypothalamus; 4) of central amygdalar nucleus. Calibration: 100  $\mu$ V and 1 sec.

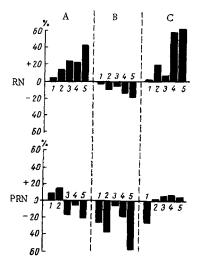


Fig. 2. Changes in parameters of RN and PRN under the influence of stimulation of the lateral hypothalamus (A), the ventro-medial hypothalamus (B), and the amygdala (C). Mean changes given in % of control: 1) duration of nystagmus response; 2) number of beats; 3) frequency, 4) amplitude, 5) energy of nystagmus.

The scheme of the experiments was as follows. Vestibular nystagmus was recorded in a rabbit. The region of the lateral and ventro-medial hypothalamus or of the corpus amygdaloideum from which behavioral responses had been evoked in that rabbit to preliminary testing were then stimulated. Immediately after (or during) stimulation the vestibular nystagmus was again investigated. Usually five or six rotations were carried out in one experiment. The location of the stimulating electrodes in the brain was verified histologically. The total number of experiments was 72.

### EXPERIMENTAL RESULTS AND DISCUSSION

Electrical stimulation of the food center of the lateral hypothalamus, which evoked an orienting and investigatory response in previously fed animals, followed by searching for food and eating it, led to a significant (P < 0.05) increase in the duration, the number of beats, and the frequency of the nystagmus (Figs. 1A and 2A). The amplitude of the RN was increased (by 21.6%) whereas that of the PRN was reduced (by 6%). Changes in energy, as a combined index of the intensity of the nystagmus, took the form of a significant increase (by 42%) for RN and a decrease (by 21%) for PRN.

An increase in the strength of stimulation of the lateral hypothalamus, evoking responses of aggressive or defensive type in the rabbits, as a rule led to inhibition of vestibular nystagmus.

Stimulation of the ventro-medial hypothalamic nuclei in fasting animals evoked an emotional response of refusal of food

during and for 20-40 sec after the end of stimulation; in some experiments responses of protective type were observed. Experiments to study the effect of stimulation of the ventro-medial hypothalamic nuclei on vestibular nystagmus showed that the effects evoked differed sharply from those of weak stimulation of the lateral hypothalamus: there was a regular decrease in all parameters of RN and PRN (Figs. 1B and 2B). The amplitude of the nystagmus fell significantly (by 12.8% for RN and by 19.5% for PRN), the duration, number of beats, and frequency of the nystagmus were reduced, and the energy of the nystagmus fell (by 18.7% for RN and by 57.7% for PRN).

Reciprocal relations clearly exist between the lateral and ventro-medial hypothalamic nuclei in their influence on the tuning of the vestibular system.

Stimulation of the nuclei of the corpus amygdaloideum (especially its basolateral part), which evoked responses resembling automatisms of food or defensive type (movements of mastication, tapping with the paws, etc.) in the rabbits during preliminary testing, led in most experiments to an increase in the duration, the number of beats, and the amplitude of RN (Figs. 1 and 2C); in some experiments the increase in amplitude occurred 10-15 sec after the beginning of rotation, whereas during the first few seconds the amplitude of the nystagmus was reduced. The energy of nystagmus, especially RN, was increased, sometimes very considerably (for example, by 95% in rabbit No. 3 and by 58% in No. 4). After amygdalar stimulation the nystagmus, especially RN, often became irregular in rhythm and amplitude, and the peaks of the waves were sometimes bimodal and trimodal. The latent period of the nystagmus response was increased almost three-fold compared with the control tests; during the first few seconds of rotation wandering of the eyes was frequently observed.

The changes found in the nystagmus responses were of short duration; as a rule 5 min after the end of electrical hypothalamic or amygdalar stimulation they were no longer detectable.

It is an interesting fact that in a state of hunger (fasting for 24-48 h) such marked activation of nystagmus should occur in the rabbits. Electrical stimulation of the food center of the lateral hypothalamus evoked further facilitation of the nystagmus. A different picture was observed in rabbits immediately after taking food. In these animals, marked inhibition of vestibular nystagmus occurred after feeding (in the phase of "sensory satiation"). The amplitude and frequency of the oscillations of RN and PRN were significantly reduced and the duration of the response also was shortened. Changes of this type in vestibular nystagmus have also been observed previously in animals and man after taking food [5, 8]. In response to electrical stimulation of the food centers of the hypothalamus only slight changes took place in the nystagmus response.

The modulating effect of the food center on the vestibular system is evidently of functional importance in the production of a state of "central motivation excitation" and the achievement of the result of a current behavioral act. There is nothing surprising about this if it is remembered that not only is the general level of motor activity increased in the hungry animal (for under natural conditions such an animal would have to hunt or seek food), but the tuning of its sensory systems, especially those with the function of orienting the eyes and head in space, is modified (to correspond to the current behavioral act), and their responses to specific sensory stimuli are rendered more acute. Results similar to those now described were obtained in experiments conducted in Anokhin's laboratory [6, 7]. It has been shown, for example, that stimulation of the food center of the lateral hypothalamus considerably increases the ability of visual cortical neurons to converge excitation of different modalities and increases the powers of discrimination of the central visual system.

The hypothalamus and corpus amygdaloideum probably exert their influence on the vestibular system through the mesencephalic reticular formation, the modulating effect of which on vestibular nuclei and on the vestibulo-oculomotor systems has been described previously [14, 16]. The "sympathetic" region of the hypothalamus has been shown to be closely connected with the vestibular nuclei through the dorsal longitudinal fasciculus [11].

Analysis of the results of the present experiments also suggests that activation of the "parasympathetic" regions of the hypothalamus evokes facilitation of vestibulo-oculomotor responses, whereas stimulation of the "sympathetic" regions inhibits them. In this respect the present results are in a large measure of agreement with those of some previous investigations in which neurotropic drugs were used to analyze the central mechanisms of vestibular nystagmus [2, 3]. The further study of the effectiveness and the characteristics of the central autonomic influences, particularly those of the limbic structures of the brain, on the vestibular system will shed further light on the mechanisms of motion sickness.

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