ANTAGONISTIC CHARACTER OF DESCENDING INFLUENCES OF MEDIAL AND LATERAL HYPOTHALAMIC NUCLEI ON EXCITABILITY OF SPINAL MOTONEURONS

A. R. Chubakov

UDC 612.826.4-064:612.832

Electrical stimulation of medial hypothalamic structures causes marked inhibition of monosynaptic and polysynaptic spinal reflexes, whereas stimulation of lateral parts of the hypothalamus is accompanied by facilitation of these reflexes.

Medial and lateral regions of the middle hypothalamus have opposite effects on animal behavior [1,7, 11,16]. Reciprocal relationships between these parts of the hypothalamus have been described [5,6,12-14],

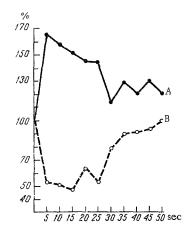


Fig. 1. Effect of stimulation of ventromedial and lateral divisions of hypothalamus on extensor monosynaptic potentials: A) dynamics of changes in monosynaptic potential during stimulation of lateral part of hypothalamus and after end of stimulation; B) dynamics of changes in monosynaptic potential during stimulation of ventromedial division of hypothalamus and after end of stimulation. Each curve shows mean results of 10 experiments. First 25 sec along abscissa corresponds to period of hypothalamic stimulation; stimulation absent from 25 to 50 sec. Ordinate: amplitude of monosynaptic potential (in % of initial background level); abscissa: time from beginning of hypothalamic stimulation (in sec).

especially in the regulation of feeding behavior. The writer has found differences in motor disturbances following destruction of lateral and medial hypothalamic structures.

In the investigation described below, descending effects of electrical stimulation of these structures on reflex activity of the spinal cord were accordingly analyzed. Conflicting views are expressed in the literature on this question [2, 9, 10].

EXPERIMENTAL METHOD

Acute experiments with recording of monosynaptic and polysynaptic spinal reflexes were carried out on 20 curarized adult cats. Preliminary operations (laminectomy, tracheotomy, exposure of the skull, dissection of the nerves, introduction of a cannula into a forelimb vein) were carried out under ether anesthesia. The animal's head was fixed in a stereotaxic apparatus and the ether anesthesia was stopped. The animal was given an injection of d-tubocurarine (0.6 mg/kg body weight) and artificial respiration applied. Mono- and polysynaptic potentials evoked by application of single stimuli to the central end of the divided tibial nerve (3-4 V, 0.1 msec, once every 5 sec) were recorded from ventral L7-S1) roots of the spinal cord with bipolar silver electrodes. Photographic recordings of the potentials were made with a "Disa" indicator. The hypothalamic structures

Laboratory of Physiological Analysis of Endogenous Neurotropic Substances, Institute of Normal and Pathological Physiology, Academy of Medical Sciences of the USSR, Moscow. (Presented by Academician V. V. Parin.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 70, No. 11, pp. 22-24, November, 1970. Original article submitted January 26, 1970.

• 1971 Consultants Bureau, a division of Plenum Publishing Corporation, 227 West 17th Street, New York, N. Y. 10011. All rights reserved. This article cannot be reproduced for any purpose whatsoever without permission of the publisher. A copy of this article is available from the publisher for \$15.00.

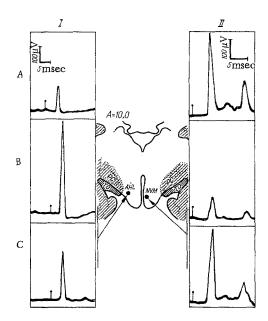


Fig. 2. Changes in monosynaptic and polysynaptic spinal reflexes during stimulation of ventromedial and lateral hypothalamic nuclei: I) changes in monosynaptic potential during stimulation of lateral hypothalamic nucleus; II) changes in monosynaptic and polysynaptic potentials during stimulation of ventromedial hypothalamic nucleus; A) initial potential; B) during stimulation; C) after end of stimulation.

were stimulated with regular pulses (5-9 V, 100 Hz, 1 msec) by means of bipolar electrodes with interelectrode distance 0.2 mm, introduced stereotaxically by reference to coordinates of an atlas of the cat's brain [15]. The animal's body temperature was maintained between 36 and 37° throughout the experiment by means of heating lamps. Potentials started to be recorded 2-2.5 h after the end of ether anesthesia. The location of the stimulating electrodes in the hypothalamic structures of each animal was verified histologically in transverse sections through the brain stained by Nissl's method.

EXPERIMENTAL RESULTS

Experimental stimulation of medial (ventro- and dorsomedial nuclei) and lateral structures of the middle hypothalamus differed in its effects on reflex activity of the spinal cord (Fig. 1). Stimulation of the ventromedial nucleus constantly produced inhibition of the extensor monosynaptic spinal reflex (P < 0.001). Different types of poststimulation effects were observed. Most frequently, inhibition during stimulation was followed by facilitation of monosynaptic reflexes after its end. In some cases, inhibition of reflexes continued after the end of stimulation, or they returned immediately to their original level. Electrical stimulation of the dorsomedial nucleus of the hypothalamus gave rise to an effect similar to that of stimulation of the ventromedial nucleus.

By contrast to stimulation of the medial structures, electrical stimulation of the lateral hypothalamus at the

same frontal level always produced facilitation (P < 0.01) of the extensor monosynaptic spinal reflex. In this case the monosynaptic potential reached 200-300% of its initial value (Fig. 2). Changes in polysynaptic reflexes during stimulation of these hypothalamic structures occurred in the same direction as the changes in monosynaptic reflexes.

In these experiments, facilitatory and inhibitory hypothalamic effects on reflex activity of the spinal cord were always bilateral. Since hypothalamic effects modified both monosynaptic and polysynaptic extensor potentials in the same direction, on the basis of results indicating that the polysynaptic component of every reflex response is, at least partially, flexor in nature [4, 8], the absence of reciprocity of these effects on extensor and flexor spinal motoneurons can be postulated. This agrees with data obtained by Baklavadzhyan et al. [2,3], who describe the general and nonreciprocal influence of the hypothalamus on extensor and flexor spinal reflexes. However, these workers, when they studied descending influences of the anterior, lateral, and posterior hypothalamic nuclei and mammillary bodies, observed only their facilitatory influences on these reflexes in unanesthetized cats. In contrast to these observations, in the present experiments structures were located in the hypothalamus, stimulation of which in unanesthetized cats was accompanied by inhibition of monosynaptic and polysynaptic spinal reflexes. These structures were the ventromedial and dorsomedial hypothalamic nuclei.

The discovery that influences of the medial and lateral hypothalamic nuclei on reflex activity of the spinal cord in waking cats are opposite in character is in agreement with the view that they act antagonistically in the regulation of the feeding behavior of animals [5,6,13].

LITERATURE CITED

- 1. E. V. Abzianidze, Fiziol, Zh. SSSR, No. 6, 664 (1969).
- 2. O. G. Baklavadzhanyan, Autonomic Regulation of Brain Electrical Activity [in Russian], Leningrad (1967).
- 3. T. K. Kipriyan, Dokl. Akad. Nauk Armyansk. SSR, 42, No. 2, 124 (1966).
- 4. P. G. Kostyuk, Fiziol. Zh. SSSR, No. 4, 398 (1960).

- 5. K. V. Shuleikina, Fiziol. Zh. SSSR, No. 8, 1025 (1964).
- 6. B. K. Anand and J. R. Brobeck, J. Neurophysiol., 15, 421 (1952).
- 7. S. Balagura, R. Wilcox, and D. Coscina, Physiol. Behav., 4, 629 (1969).
- 8. R. M. Eccles and A. Lundberg, Arch. Ital. Biol., 97, 199 (1959).
- 9. R. Emmers, Arch. Ital. Biol., 99, 322 (1961).
- 10. S. Feldman and J. Wagman, Exp. Neurol., 5, 250 (1962).
- 11. J. R. Knott, W. R. Ingram, and R. E. Correll, Arch. Neurol. (Chicago), 2, 247 (1960).
- 12. M. Le Moal and B. Cardo, C. R. Soc. Biol., 162, 405 (1968).
- 13. Y. Oomura, K. Kimura, H. Ooyama, et al., Science, 143, 484 (1964).
- 14. A. Sclafani and S. Grossman, Physiol. Behav., 4, 533 (1969).
- 15. R. S. Snider and W. T. Niemer, A Stereotaxic Atlas of the Cat Brain, Chicago (1961).
- 16. M. D. Wheatly, Arch. Neurol. Psychiat., 52, 296 (1944).