EFFECT OF ELECTRICAL STIMULATION OF THE MEDULLA ON THE CARDIOVASCULAR, RESPIRATORY, AND MOTOR SYSTEMS

K. A. Smirnov

UDC 612.1+612.2+612.74]-06:612.828.014.421

Stimulation of medullary structures under chronic and acute experimental conditions evokes complex responses including cardiovascular, respiratory, and motor components. In unanesthetized dogs the arterial pressure was increased in response to stimulation of all structures tested. Injection of hexobarbital in several cases converted pressor responses into depressor. A decrease in the frequency of stimulation from 100 to 10 and 2 pulses/sec was accompanied by disappearance or a marked diminution of the responses.

Most information on the functional organization of the vasomotor center has been obtained in anesthetized or decerebrate animals. However, some investigations [1-3, 6-8, 10-12] have conclusively demonstrated that the character and magnitude of the vasomotor reflexes and the action of direct stimulation of ponto-medullary structures on the cardiovascular and somatic systems are dependent on anethesia and decerebration. The writer has previously shown [4] that during electrical stimulation of medullary structures under chronic experimental conditions elevation of the arterial pressure and holding of the breath in inspiration are most frequently observed.

In this investigation the effects of stimulation of bulbar structures on the cardiovascular, respiratory, and motor systems were compared under chronic and acute experimental conditions.

EXPERIMENTAL METHOD

Electrodes were implanted in medullary structures of dogs weighing 8-12 kg. After the dogs had recovered, a second operation was performed, during which a polyethylene catheter filled with heparin in a dilution of 1:3 and closed with a stopper was inserted into the central end of the carotid artery. The free end of the catheter was brought out through an incision in the skin in the dorsal region. The experiments began next day. During the experiments the dogs were kept in a special frame [13]. Medullary structures were stimulated with square pulses with frequencies of 2, 10, and 100 pulses/sec, duration 1 msec. The intensity and duration of stimulation depended on the animal's behavior. The arterial pressure was measured by a "Barovar" electromanometer, and respiration was recorded by means of a pneumatic capsule, fixed to the chest wall, and a photoelectric sensor element. Recordings were made on a "Cardiovar" ink-writing instrument. Motor responses were assessed visually. After the end of the chronic experiment, an acute experiment was performed under hexobarbital anesthesia (50 mg/kg), and when it was ended, the dog's medulla was removed along with the electrode and fixed in 10% formalin solution. The sections were identified with the aid of the atlas of Lim et al. [9]. Altogether 50 experiments were performed on 12 dogs.

EXPERIMENTAL RESULTS

Stimulation of all test structures of the medulla under chronic and acute experimental conditions was accompanied by the appearance of complex responses including cardiovascular, respiratory, and motor com-

Group for Investigation of Vasomotor Reflexes, I. P. Pavlov Institute of Physiology, Academy of Sciences of the USSR, Leningrad (Presented by Academician V. N. Chernigovskii.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 69, No. 6, pp. 3-5, June, 1970. Original article submitted October 31, 1969.

TABLE 1. Action of Electrical Stimulation of Medullary Structures on Cardiovascular, Respiratory, and Motor Systems

Structures	Coordinates (from atlas of Lim et al. [9])	Chronic experiment				Acute experiment			
		AP	P	R	motor response	AP	P	R	motor response
Gigantocellular nucleus of reticular formation	C6;3;4	+	T	I	Cries	+	B	I	Opens mouth
	C6;4;5	+	T	I	Holds out paw, cries	+	T	I	Holds out paw, opens mouth
	C7;4;9	+	B	I	Holds out paw, becomes absolutely still	–	B	I	Holds out paw
	C7;1,5,4	+	T	I	General restlessness	–	B	I	Opens mouth
Parvocellular nucleus of reticular formation	C7;5;4	+	B	I	Turns head to the left	+	B	I	Turns head to the left
	C8;2;2	+	B	Q	Opens mouth, cries	+	O	I	Holds out paw, opens mouth
	C8;2,2;5	+	T	I	Opens mouth, turns head to the left	+	O	I	Opens mouth, turns head to the left
Near nucleus of tractus solitarius	C10;4;2	+	T	I	Cries	–	B	E	Turns head to the left
Caudal pontine nucleus of reticular formation	C3;2;2	+	T	I	Inclines head forward	–	I	I	Turns head to the left
Ventral nucleus of reticular formation	C10;4;5,4	+	T	I	Cries	+	T	I	Opens mouth
Inferior vestibular nucleus	C7;5;2	+	O	Q	Turns head to the left, opens mouth	+	I	I	Turns head to the left, opens mouth
Accessory cuneate nucleus	C9;4,5;2	+	T	S	Cries	–	I	Q	Opens mouth and closes mouth

Legend: AP) arterial pressure; R) respiration; P) pulse; +) elevation of AP; –) lowering of AP; B) bradycardia; T) tachycardia; O) no change in pulse; I) holding breath in inspiration; E) holding breath in expiration; Q) quickening of respiration; S) slowing of respiration.

ponents. No isolated changes affecting the respiratory, cardiovascular, or motor systems were observed. The complex character of these responses cannot be regarded as a stimulation artefact, due to the spread of loops of current through adjacent nervous structures, because all components mentioned above were always represented even in response to electrical stimulation at the threshold level. A response of this type lies at the basis of the functional organization of the reticular formation of the brain stem.

In response to stimulation of bulbar structures in the chronic experiments the arterial pressures always rose, while respiration as a rule stopped in inspiration. Quickening of the heart rate was observed twice as often as slowing. Changes in arterial pressure, pulse, and respiration of this type were produced by stimulation of both medial and lateral structures, in some cases (in response to stimulation of certain parts of the gigantocellular nucleus, the accessory cuneate nucleus, and neurons in the immediate proximity of the spinal nucleus of the trigeminal nerve) anesthetizing the animals caused pressor responses with tachycardia to change into depressor responses with bradycardia. Different opinions have been expressed in the literature regarding the causes of this action of anesthetics. The most likely hypothesis is that of Khayutin [5], according to whom a "demasking" of the inhibitory component of physiological responses, which exists also in waking animals, takes place under the influence of anesthesia. It must be remembered, however, that in some cases the chronic experimental conditions do not permit a precise answer to the question whether the pressor responses are the result of direct stimulation of medullary structures or of the animal's motor excitation. In this case square pulses of different frequencies and intensities were used to stimulate the cardiovascular bulbar structures. In all experiments (both chronic and acute) lowering the frequency of stimulation from 100 to 10 or 2 pulses/sec was accompanied by disappearance or considerable diminution of the response.

LITERATURE CITED

1. A. V. Val'dman, in: Current Problems in the Physiology and Pathology of the Nervous System [in Russian], Moscow (1965), p. 325.
2. L. A. Kitaev-Smyk, in: Abstracts of Proceedings of the 2nd Conference of Junior Scientists of the Institute of Pharmacology and Chemotherapy, Academy of Medical Sciences of the USSR [in Russian], Moscow (1960), p. 15.
3. G. V. Kovalev, in: Current Problems in the Pharmacology of the Reticular Formation and Synaptic Transmission [in Russian], Leningrad (1963), p. 237.
4. K. A. Smirnov, Fiziol. Zh. SSSR, No. 12, 1406 (1968).
5. V. M. Khayutin, Functional Organization of Vasomotor Reflexes. Doctoral Dissertation [in Russian], Moscow (1962).
6. V. M. Khayutin, Acta Physiol. Acad. Sci. Hung., 29, 145 (1966).
7. F. Bergman and J. Gutman, Acta Physiol. Lat.-Amer., 16, 49 (1966).
8. J. Gutman, M. Chaimovitz, V. Ginath, et al., Arch. Internat. Physiol., 70, 33 (1962).
9. R. K. S. Lim, Liu Chan-nao, and R. L. Moffitt, A Stereotaxic Atlas of the Dog's Brain, Springfield (1960).
10. A. J. Mandell and L. M. N. Bach, Am. J. Physiol., 190, 330 (1957).
11. T. Minami, Fukuoka Acta Med., 49, 153 (1958).
12. T. Nisicimi, Folia Pharmacol. Jap., 52, 586 (1956).
13. H. Sheppard, Texas Rep. Biol. Med., 24, 228 (1966).