CORTICOFUGAL EFFECTS ON THE SOMATOSENSORY CORTEX ON TRANSMISSION IN THE POSTERIOR VENTRAL NUCLEUS

E. Lang* and V. L. Glants

UDC 612.825.1:612.826.5

Responses to electrical stimulation of fibers of the medial lemniscus arising in the thalamo-cortical fibers running to the primary somatosensory cortex were investigated in acute experiments on cats anesthetized with pentobarbital. Application of penicillin to the somatosensory cortex and the consequent development of paroxysmal activity in it considerably reduced the amplitude of responses in the thalamo-cortical fibers. The first somatosensory area was much more effective than the second in this respect. The development of paroxysmal activity in the projection cortex also appreciably reduce after-inhibition in the posterior ventral nucleus, as shown by application of paired stimuli to the medial lemniscus.

KEY WORDS: cerebral cortex; thalamic nuclei; regulation of the afferent flow.

Corticofugal connections of the posterior ventral nucleus (PVN) are known to exist [4, 9, 10, 14]. These connections provide the morphological basis for the modulating effects of the somatosensory cortex on the transmission of afferent impulses through PVN [5, 13]. Both excitatory and inhibitory effects from the somatosensory areas of the cortex on single relay neurons of PVN have been discovered [7, 16].

The object of this investigation was to study the effect of cortical projection zones on responses of thalamo-cortical cells sending axons to the first somatosensory area (SI) to stimulation of fibers of the medial lemniscus (ML). The participation of the first and second (SII) cortical somatosensory areas in the formation of after-processes in PVN also was studied.

The response of thalamo-cortical fibers (TCF) recorded close to area SI reflects the discharge of relay cells of PVN projecting to this part of the cortex. The conditions of recording the focal potential in a bundle of parallel TCF can be regarded as similar to the conditions of recording the action potential of a nerve trunk. The amplitude of the response thus recorded is a linear function of the number of fibers involved [12]. Consequently, changes in the amplitude of the focal response of TCF give some indication of the relative change in the number of thalamo-cortical relay cells of PVN responding to stimulation of ML.

EXPERIMENTAL METHOD

Acute experiments were carried out on 28 cats anesthetized with pentobarbital (40 mg/kg, intraperitoneally). The femoral vein and trachea were cannulated, flaxedil was injected intravenously and the animal was artifically ventilated. The skull was trephined and subcortical concentric bipolar electrodes (interelectrode distance 1-1.5 mm) were inserted with the aid of a system of stereotaxic coordinates. Penicillin solution (concentration 100,000 units/ml) was applied epipially to the exposed areas of cortex. The amplitude of the bioelectrical responses was recorded from the screen of a cathode-ray oscilloscope. Statistical analysis of the results was carried out with the FACOM computer.

*Anatomical Institute, Budapest University, Hungary.

Institute of Normal and Pathological Physiology, Academy of Medical Sciences of the USSR, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR, A. M. Chernukh.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 79, No. 1, pp. 3-6, January, 1975. Original article submitted February 13, 1974.

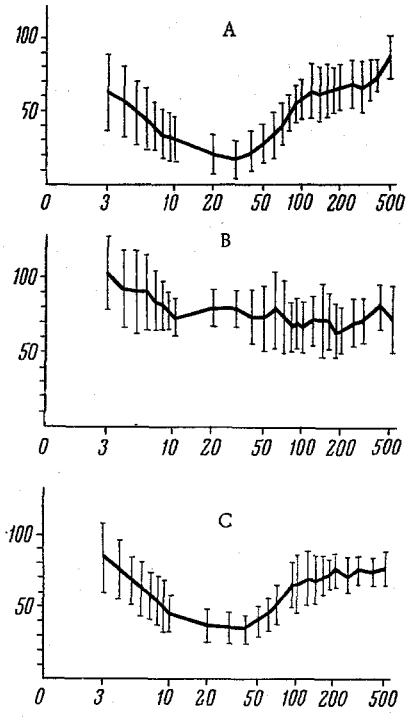

Fig. 1

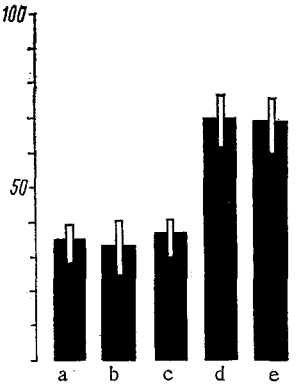

Fig. 2

Fig. 1. Curves of recovery of amplitude of testing responses in thalamo-cortical fibers running from area SI. A) Averaged curve from 24 experiments on animal anesthetized with pentobarbital; B) recovery curve after application of penicillin to areas SI and SII and development of paroxysmal activity therein; C) recovery curve after removal of areas SI and SII and application of penicillin to them. Abscissa, interval between conditioning and testing stimuli (in msec; logarithmic scale); ordinate, amplitude of testing response in percent of amplitude of conditioning response.

Fig. 2. Change in amplitude of responses in thalamo-cortical fibers after procedures on somatosensory cortex. Initial amplitude of response taken as 100%: a) after application of penicillin to area SI; b) after application of penicillin simultaneously to areas SI and SII; c) after removal of area SII and application of penicillin to both projection areas; d) after removal of area SI and application of penicillin to both projection areas; e) after simultaneous removal of areas SI and SII following application of penicillin to both of them.

EXPERIMENTAL RESULTS

The response of stimulation of ML and TCF was recorded after a latent period of 0.7-1.1 msec. The duration of the response was about 2 msec, and its amplitude 400-800 μV. Application of identical paired stimuli to the region of ML, with intervals of between 3 and 500 msec between stimuli provided information on the character of the change in the second (testing) response relative to the first (conditioned).

The maximal decrease in amplitude of the testing response was observed when the interval between stimuli was 25-35 msec (Fig. 1A). As the interval increased to 500 msec the amplitude of the testing response reached 88 ± 11% of that of the conditioned response.

In five cases ipsilateral thermocoagulation of the somatosensory cortical areas was carried out. The removal of areas SI, SII, or both these areas caused no change either in the amplitude of the response or in the curve of recovery of the testing response to paired stimulation of ML.

After application of penicillin solution simultaneously to areas SI and SII and the development of paroxysmal activity in these cortical projection areas, the amplitude of the response of TCF to stimulation of ML fell appreciably to a mean value of 33 ± 7.3% of its initial level (Fig. 2b). The recovery curve of the testing response in this case showed significant changes over the whole range of intertrial intervals studied (Fig. 1B).

Immediately after thermocoagulation of area SI and against the background of paroxysmal activity in both somatosensory areas, the amplitude of the response in TCF running to SI increased sharply on the average of 70% of the amplitude of the response observed before penicillin application (Fig. 2d). However, isolated removal of area SII only, after the application of penicillin, caused virtually no change in the amplitude of the response in fibers running to SI (Fig. 2c). The change in the TCF response after removal of both somatosensory areas against the background of cortical paroxysmal activity was virtually indistinguishable from the effect of isolated removal of area SI.

Meanwhile the character of the recovery curve of the testing response after removal of both areas became similar to the character of the recovery curve before penicillin application (Fig. 1C).

In three experiments penicillin was applied to the exposed white matter after extirpation of the somatosensory cortical areas. In these cases no change in the amplitude of the TCF response or in the recovery curve of the testing response to paired stimulation of ML could be discovered.

During barbiturate anesthesia the afferent flow is regulated at the thalamic level chiefly by internal mechanisms of the relay nucleus, because barbiturate anesthesia significantly reduces the regulatory influence of the projection cortex [1, 2, 3] and the nonspecific structures of the brain [8, 11]. It it understandable, therefore, that thermocoagulation of the cortical projection areas in an animal under barbiturate anesthesia will have no significant effect on the course of after-inhibition in PVN. To activate the projection cortex under these conditions use was made of the seizure effect induced by the application of penicillin to its surface. Seizure waves on the EEG correspond to high-frequency discharges of cortical neurons superposed on a depolarization shift of membrane potential [6, 15]. This creates conditions under which the cortico-thalamic pathways are activated, although under pentobarbital anesthesia the nonspecific structures most probably remain blocked, so that the role of corticofugal influences on the transmission of impulses in PVN can be traced more clearly. Generation of seizure activity in the cortical projection zones leads to a decrease in amplitude of the TCF response to stimulation of ML, which points to a decrease in the number of relay cells in PVN responding to the synchronized volley. Since cogulation of the projection cortex after the appearance of seizure activity leads to restoration of the amplitude of TCF responses to stimulation of LM, it can be concluded that the seizure activity of the cortical neurons inhibits the transmission of afferent impulses in PVN. This is all the more probable because application of penicillin to the white matter after preliminary removal of the cortex was ineffective. Isolated extirpation of areas SI and SII after preliminary application of penicillin revealed differences in the relative contribution of these areas to the regulation of transmission of afferent impulses through PVN. The results showed that in the case of thalamic neurons sending axons to area SI, the inhibitory corticofugal influences from this area were far more effective than those from area SII.

The curve of recovery of excitability of the thalamo-cortical relay cells capable of responding to ML stimulation after the development of paroxysmal activity in the cortex differed significantly from the recovery curve before penicillin application. After-processes in the neurons of this group differed significantly from the after-process in the original population of relay neurons. A statistically significant decrease in the degree of after-inhibition took place for all intertrial intervals between the conditioning and testing stimuli.

LITERATURE CITED

1. V. B. Golovchinskii, in: Proceedings of the 10th Congress of the I. P. Pavlov All-Union Physiological Society [in Russian], Erevan (1964), No. 1, p. 215.
2. V. B. Golovchinskii, Zh. Vyssh. Nerv. Deyat., No. 6, 1098 (1965).
3. R. A. Durinyan, V. L. Glants, and A. G. Rabin, Zh. Vyssh. Nerv. Deyat., No. 6, 1256 (1971).
4. Z. V. Eliseeva, Morphology of Corticothalamic Structures of the Somatic Analyzer of the Cat, Candidate's Dissertation, Moscow (1969).
5. A. G. Rabin, Fiziol. Zh. SSSR, 51, 159 (1965).
6. C. Ajmone-Marsan, in: Basic Mechanisms of the Epilepsies, Boston (1969), p. 229.
7. P. Andersen, K. Junge, and O. Sveen, Nature, 214, 1011 (1967).

8. P. B. Bradley and B.J. Key, Electroenceph. Clin. Neurophysiol., 10, 97 (1958).
9. S. Ramon-y-Cajal, Histologie du Système Nerveux de l'Homme et des Vertèbres, Vol. 2, Paris (1911), pp. 519 and 993.
10. J. G. Dusser de Barenne, Ass. Res. Nerv. Ment. Dis. Proc., 15, 274 (1935).
11. H. Gangloff and M. Monnier, Arch. Exp. Path. Pharmak., 231, 211 (1957).
12. B. Katz, Nerve, Muscle, and Synapse, New York (1966).
13. T. F. Ogden, Electroenceph. Clin. Neurophysiol., 12, 620 (1960).
14. T. P. S. Powell, Brain Res., 10, 369 (1968).
15. D. Prince and B.J. Wilder, Arch. Neurol., 16, 194 (1967).
16. H. Shimazu, N. Janagisawa, and B. Garoutte, Jap. J. Physiol., 15, 101 (1965).