POSTSYNAPTIC INHIBITION IN THE GENERAL CORTEX OF THE TURTLE FOREBRAIN

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Postsynaptic inhibition in the general cortex of the turtle forebrain was investigated by recording unit activity intracellularly. Depending on the type of IPSPs recorded in response to electrical stimulation of the contralateral optic nerve and cortical surface the neurons were subdivided into three groups: 1) with long direct IPSPs, 2) with long and short direct, and also recurrent IPSPs, 3) with short direct and recurrent IPSPs. It is concluded that inhibitory pathways of the short direct and recurrent IPSPs have a common final component, a stellate interneuron. Compared with the recurrent collateralsof the principal neurons, the direct afferents make contact with more distal portions of the dendrites of this cell. Synapses formed on dendrites of the principal neurons by axons of the stellate cells are nearer to the soma than synapses responsible for generation of the long direct IPSP.

INTRODUCTION

Morphological investigations show that the principal cells of the general cortex of the turtle receiving afferent fibers and giving off efferent fibers are arranged in a compact layer in the lower third of the cortex [1, 2, 6, 7]. These cells are pyramidal or piriform neurons with a wide and narrow zone of ramification of the dendrites [2]. The same layer also contains short-axon cells of the stellate type. Horizontal cells are located in the neuropil. Their dendrites diverge in a tangential direction and the axon branches in the region of the dendrites of the principal neurons [2].

Previous investigations [3-5] showed that in about 60% of cells an IPSP is formed in response to electrical stimulation of the optic nerve, most commonly after an EPSP. Depending where the recording is taken, either an EPSP1 and EPSP2, or both together may be recorded. The EPSP1 arises in the rostral zone of the cortex and the EPSP2 (with longer latency than EPSP1) in the caudal zone. Both EPSPs may appear together in the central zone of the cortex. The IPSP of neurons in this zone of the cortex arises as a result of at least two afferent excitatory volleys.

In this investigation the structure of the inhibitory pathways of the turtle's general cortex was analyzed, for it is evidently an important component of the mechanisms of response of cortical neurons to adequate stimuli.

METHOD

Experiments were carried out on pond turtles (Emys orbicularis) immobilized by intramuscular injection of 2% diplacin* solution (0.2-0.3 ml). In the course of the experiment a weak jet of air was introduced continuously into the lungs through the trachea. The dura and arachnoid mater were removed and the pia torn where the microelectrode was inserted.

*1,3-di(β-platynecliumethoxy)benzene hydrochloride.

Fig. 1. Effect of depolarization change of membrane potential on IPSP amplitude: 1-6) unit responses to stimulation of ON, 7-10) to stimulation of CS. Records below odd numbers, controls; under even numbers, during depolarization. Here and in Figs. 2 and 4, calibration: 20 mV, 100 msec. Broken line shows level of membrane potential.

Bipolar nichrome electrodes were used for electrical stimulation. The contralateral optic nerve (ON) and cortical surface (CS) were stimulated close to the microelectrode.

Potentials were recorded intracellularly by glass microelectrodes (resistance 300-500 MΩ) filled with 2 M potassium citrate solution. The cells were polarized by passing a current through the microelectrode by means of a bridge circuit. The signal was led to a preamplifier (R_{in} \sim 100 GΩ) and dc amplifier with transmission band of 0-15 kHz, connected in series.

RESULTS

Altogether 273 neurons in the nuclear layer of the general cortex were recorded (resting potential not below 40 mV). IPSPs were recorded in 207 neurons (64 in the rostral, 87 in the central, and 56 in the caudal zones of the cortex) in response to stimulation of ON and CS. To simplify the task, IPSPs arising in neurons of only the rostral or only the caudal zones of the cortex will be analyzed, for in these cases the IPSPs arise chiefly to one of the afferent excitatory volleys reaching the cortex.

Depending on the duration of the IPSPs, the effect of polarization on them, and the character of summation of these potentials during paired stimulation the neurons could be divided into three groups.

In the neurons of group 1 (21 cells) a long IPSP (~800 msec, sometimes 1500-2000 msec) with a latent period (LP) of about 50 msec in the rostral and about 70 msec in the caudal zones of the cortex was recorded in response to stimulation of ON. With a change in membrane potential through depolarization (either artificially or as a result of damage to the cells) the IPSP increased steadily (Fig. I: 1, 2). During paired stimulation of ON this IPSP showed good summation by duration (but not summation by amplitude) irrespective of spike generation in response to the testing stimulus (Fig. 2: 1, 2). In response to stimulation of CS, a long IPSP, also showing good summation, was recorded in the neurons of this group (Fig. 2: 9, 10).

In the neurons of the second group (63 cells) the duration of the IPSPs, as in those of the first group, was about 800 msec, and their LP was about 50 msec in the rostral and about 70 msec in the caudal zone of the cortex. The complex composition of this IPSP was apparent on depolarization: the initial phase (duration 300 msec) increased much more than the late phase (Fig. 1: 3, 4). The same heterogeneity and complex composition of this IPSP also were observed during paired stimulation. Good summation in amplitude (but not in duration) of the early component (duration 70-80 msec) was observed, but summation of the late component (LP about 120 msec in the rostral zone and 150 msec in the caudal zone of the cortex) was by duration. Summation took place in both cases regardless of whether a spike was generated in response to the testing stimulus or not (Fig. 2: 3, 4).