LATE RESPONSES OF THE VISUAL CORTEX DURING CONVERGENCE OF PHOTIC AND ELECTRODERMAL EXCITATION

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In experiments on unanesthetized rabbits with electrodes permanently implanted into brain structures over which visual excitation is dispersed the dynamics of changes in the late slow evoked potential (EP) to light was studied in relation to the time of application of nociceptive electrical stimulation to the limb. The experiments showed that suppression of this EP component takes place if the electrodermal stimulus is applied during the latent period of the late response regardless of whether the phase of the photic EP was inhibitory or excitatory.

The study of interaction between heterosensory excitation in synaptic organizations of cortical neurons has yielded conflicting results. According to some workers [3], modulation of unit activity in the visual cortex by nonvisual stimuli (acoustic, electrodermal, or stimulation of the reticular formation) is the result of an orienting reflex and is tonic in character. Other workers [2], however, using various combinations of acoustic and photic stimulation separated by different time intervals, observed both phasic and tonic changes in unit activity. The phasic changes and their dependence on the excitatory and inhibitory phases of the photic response are regarded as the result of the specific action of sound on the unit activity, whereas the tonic changes, according to these same workers, are determined by the nonspecific effect of sound on neurons of the visual cortex.

In the present writers' experiments on waking rabbits, simultaneous photic stimulation and electrodermal stimulation of the limb led to suppression of the late slow surface negative-positive evoked potential (EP) to light [4].

To study the mechanisms of suppression of this late response the investigation described below was carried out. An electrodermal stimulus was applied at various microintervals of time after a flash, i.e., when structures responsible for EP generation were in different phases of excitation.

EXPERIMENTAL METHOD

Experiments were carried out on rabbits with permanently implanted electrodes in brain structures where dispersion of visual excitation takes place, namely the visual, temporal, and sensomotor areas of the cortex, the lateral geniculate body (LGB), hippocampus, bulbar reticular formation, and lateral and ventromedial hypothalamic nuclei. The reference electrode was placed on the nasal bone. Surface electrical activity and the EPs were recorded by a monopolar method on a 16-channel "Alvar" electroencephalograph. Meanwhile EPs in the occipital region and sometimes in LGB were recorded by means of a "Disa Electronic" universal indicator. Flashes were generated by a flash lamp at the rate of 1/sec from an "Alvar" Phonophotostimulator, emitting series of nine flashes. To coincide with the 4th, 5th and 6th flashes or at various time intervals from them, an electric shock (10-15 V, 1 msec) was applied to the hind limb.

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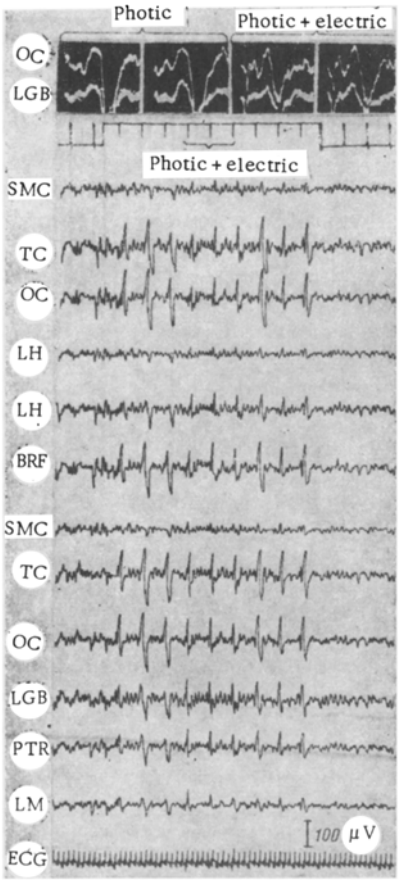

Fig. 1

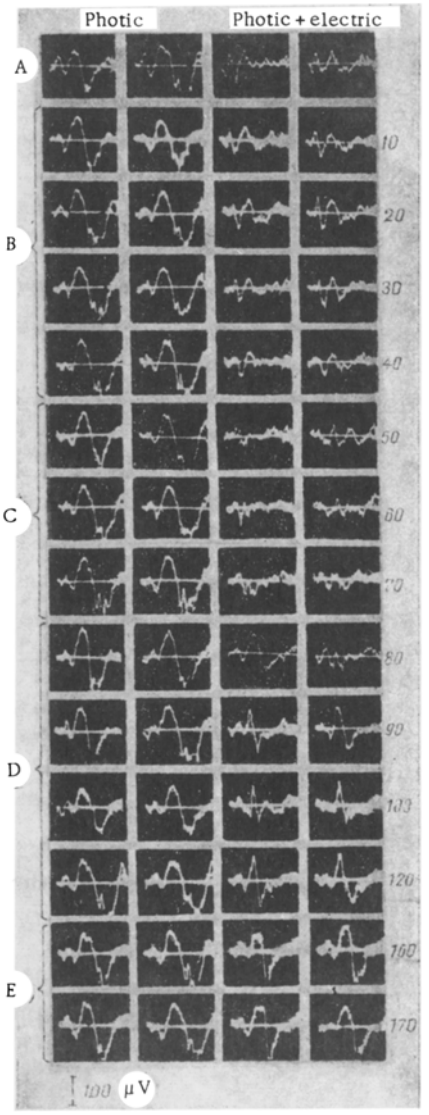

Fig. 2

Fig. 1. Change in slow negative-positive wave of EP to photic stimulation in cortex and subcortical structures during simultaneous application of electric shock of threshold strength. SMC) Sensomotor cortex; TC) temporal cortex; OC) occipital cortex; LH) lateral hypothalamus (two records); TR) pretectal region; BRF) bulbar reticular formation; LM) medial lemniscus; LGB) lateral geniculate body.

Fig. 2. Dynamics of changes in late slow wave of EP in occipital cortex to photic stimulation with simultaneous (A) and delayed (B-E) electrodermal stimulation relative to flash (time interval of delay in msec). Details in text.

EXPERIMENTAL RESULTS

The investigation showed that electrodermal stimulation has an effect on the EP of the visual cortex and other brain structures which depends on the strength of the current and duration of the interval. For example, stimulation of subthreshold strength for suppressing the late slow wave (8-10 V) facilitated the positive phase of the primary response and, inconstantly, the secondary positive wave also. At the same time the configuration of the late slow negative-positive response was modified as the result of shortening of the negative phase and a marked decrease (by 1.5-2 times) in the amplitude of the positive wave. Changes of the same type in the slow wave were also observed in LGB and in other brain structures (Fig. 1).

An increase in strength of the current had an inhibitory action on generation of the late slow wave both in the cortex and in the subcortex, whereas the primary response and secondary positive wave were facilitated in most cases (Fig. 2A).

Application of an electric shock to the skin of the limb 40-45 msec after the flash had the same inhibitory action on the late slow wave as simultaneous application of these stimuli. This part of the EP either became very reduced or disappeared completely, while the secondary positive wave was frequently facilitated (Fig. 2B).

A subsequent increase in the interval between the two heterosensory stimuli to 45-55 msec, i.e., within the latent period of the secondary positive wave, suppressed the generation of this wave and also of the late slow response (Fig. 2C). Finally, the critical interval for liberation of late slow wave from inhibition by the electrodermal stimulus was 75-100 msec, i.e., it fell outside the latent period of the late slow response. Because of fluctuations in the duration of the latent period between 75 and 100 msec, depending on the strength of the stimulating current and on an adaption to nociceptive stimulation, the generation of the late slow wave in these intervals was unstable. However, if it happened to be stable, the configuration of the late response reflected the influence of the electrodermal nociceptive stimulus. This was shown by the fact that the negative and positive phases became shortened and pointed (Fig. 2D). This sharp-frontedness of the EP, as experiments with stimulation of the reticular formation and hypothalamus have shown [4], is the result of increased excitability of the subcortical structures and is evidence of synchronization of multiple streams of excitation at the subcortical level and also on synapses of the cortical cells. This type of change in the configuration of the negative phase of the late slow wave persisted when the electric shock was applied after an interval of between 100 and 140 msec, i.e., as long as it was applied during the ascending part of this wave. With an increase in the interval to 160 msec, the negative phase of the slow wave was no longer changed, whereas its positive phase still reflected subcortical synchronization of excitation rising in the cortex (Fig. 2E). This effect persisted when the electric shock was applied 180-200 msec after the photic stimulus.

These results show that suppression of the late slow wave of the EP to photic stimulation as the result of interaction between photic and electrodermal stimuli took place if the electric shock was applied in the latent period of the late response, regardless of whether this coincided with the inhibitory or excitatory phase of the EP to light. The electrodermal stimulation had an opposite action on the primary and secondary positive waves – they were facilitated. These results are fresh confirmation of differences in the genesis of the primary response and the late slow wave and they are in harmony with Anokhin's view that the EP is determined by the combined action of many streams of ascending excitation, and that its actual configuration is the result of the degree of dispersion of these streams of excitation at different levels of the subcortical pathway [1]. Another interesting fact is that suppression of the late slow wave occurred both in the cortex and in the subcortex, i.e., it was generalized in character. This means that inhibition of the afferent flow determining the genesis of the late response takes place before dispersion of the visual excitation over various structures of the brain. However, the possibility cannot be ruled out that electrodermal, like nociceptive, stimulation has a generalized and emotionally negative action on brain structures through the reticular formation and hypothalamus. This is a specific nociceptive action and not the result of an orienting reflex, mainly because it does not undergo extinction, one of the most characteristic features of the orienting reflex.

Nevertheless, regardless of the mechanisms concerned, electrodermal stimulation has some effect on visual stimulation, and it acts differently on different components of the EP.

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