Cortical control of specific and nonspecific sensory projections to the cerebral cortex¹

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Activation of the frontal association response field of the cerebral cortex of cat by a brief train of electric shocks is shown to induce a subsequent marked depression of nonspecific evoked cortical association responses and an enhancement of the later components of evoked primary sensory responses. Both effects have been reported to accompany behavioral attending. The two effects of cortical stimulation can be differentiated by drug actions: strychnine abolishes the depression of association responses and picrotoxin abolishes the enhancement of primary responses. It is suggested that the frontal association response field may play a crucial role in the control of specific and nonspecific sensory projections to the cerebral cortex.

When an organism "attends" to a sudden sensory stimulus, two seemingly opposite changes occur in cortical responses evoked by the stimulus. The later components of primary sensory responses recorded from the human scalp increase in amplitude (Davis, 1964; Haider, Spong, & Lindsley, 1964). Nonspecific evoked responses recorded from association cortex of the cat, on the other hand, are markedly depressed (Shaw & Thompson, 1964; Thompson & Shaw, 1965). This report will show that a single maneuver, electrical activation of the frontal cortex, can produce both types of changes in cortical activity.

Method

Cats were anesthetized with 70 mg/kg of α -chloralose (IP), and the cerebral cortex exposed. Cortical evoked responses were recorded with a gross monopolar surface electrode, using standard techniques. Series of individual responses and computer (Enhancetron) averaged responses were obtained. The electrical stimulus to the frontal cortex was a train of 10 pulses, of .2 msec. duration each at a frequency of 250/sec., from a Tektronix 161 pulse generator delivered through an isolation transformer to two silver ball electrodes placed 2 mm. apart on the cortex. Peripheral stimuli were light flash, free field click, or skin shock, presented at various times (0 to 500 msec.) after onset of the 40 msec. cortical shock train. In drug experiments chloralosed animals were first immobilized with Flaxedil (IV) and then administered IV doses of strychnine (0.2 mg/kg) or picrotoxin (2 mg/kg).

Results

Electrical stimulation of frontal cortex produced a marked depression of nonspecific association responses and an enhancement of the later components of primary sensory responses. The effects began about 10 msec. after onset of cortical shock train, reached a maximum

at 50 to 80 msec. and were over by about 300 msec. The areal distribution of relative effectiveness of cortical shock train on the frontal cortex was virtually identical to the relative amplitude distribution of evoked association responses in this region (Thompson, Johnson, & Hoopes, 1963), with maximum effectiveness about 3 mm. anterior to the cruciate sulcus. The effects could still be obtained by precruciate stimulation after ablation of somatic sensory area I. Stimulation of somatic sensory area I was relatively ineffective.

Data shown in Fig. 1 illustrate the effect of increasing cortical stimulus intensity on the amplitude of specific and nonspecific cortical evoked responses. Peripheral stimuli were given 70 msec. after onset of the 40 msec. cortical shock train. Inset drawings in Fig. 1 identify the components of the association and primary evoked responses plotted in the graphs. The initial positive component (P_1) of nonspecific responses to all three types of peripheral stimuli, recorded from the middle suprasylvian and anterior lateral association areas, was markedly depressed in an equivalent manner by the cortical shock train. In contrast, the initial positive component (P_1) of primary responses recorded from areas AI to click, VI to light flash, and SI to forepaw shocks, was not

Fig. 1. Effect of intensity of cortical shock train to frontal cortex on primary and association cortical responses evoked by sensory stimuli. Various components of the evoked responses (identified by insets—positive up) are plotted separately. Abscissa scale refers to stimulator (Tektronix 161) settings.

depressed. In fact there was a slight (10%) but statistically significant increase in response amplitudes following strong cortical shocks. The subsequent negative component (N_1) of all three types of primary response exhibited a substantial increase in amplitude with increasing cortical shock intensity. Finally, the afterpositivity (P_2) of the primary visual response (the only type of response where P_2 could be obtained reliably) was markedly enhanced by the cortical shock train.

The increases in later components of primary cortical responses following cortical shock are strikingly comparable to the increases in human evoked response components during "attention" recently described by Spong, Haider, & Lindsley (1965). Response components from their data were identified in terms of waveforms, polarities, and peak latencies that corresponded to the response components defined in our experiments. Thus, for the primary visual evoked response, their data indicated an increase of about 75% in the N_1 component, compared to a 70% increase in our experiments, and an increase of about 150% in the P_2 component, compared with an increase of 120% in our experiments (see Fig. 1).

The actions of two inhibitory blocking agents, strychnine and picrotoxin, on the effects of cortical shock on association and primary responses were explored. Strychnine totally abolished the depression of association responses following cortical shock, but did not alter the enhancement of primary responses. Picrotoxin, on the other hand, abolished the enhancement of primary responses, but did not alter the depression of association responses.

Discussion

The results of these experiments indicate that electrical activation of the frontal association response field of the cortex can reproduce both the depression of association cortical responses and the enhancement of the later components of primary cortical responses that accompany behavioral attending. This region of the cortex may well play a crucial role in the control of both specific and nonspecific input to the cerebral cortex. Krauthamer & Albe-Fessard (1965) recently reported that electrical activation of the basal ganglia depresses cortical association responses without influencing primary responses. It may be that this system is involved in the control of association responses by the frontal cortex. Consistent with this is their finding that strychnine abolished the effects of basal ganglia stimulation. The possibility that our results for cortical association responses might be an

artifact of stimulus current spread to the basal ganglia was ruled out by undercutting the stimulated cortex: all effects of stimulation were eliminated.

Since strychnine appears to abolish most forms of postsynaptic inhibition and picrotoxin markedly reduces presynaptic inhibition (Eccles, 1964), the drug effects obtained in our experiment suggest that depression of cortical association responses following cortical stimulation may involve postsynaptic inhibition and the enhancement of primary responses may involve presynaptic inhibition. However the time course of the depression of association responses is strikingly parallel to the time course of presynaptic inhibition on primary afferent fibers of the spinal cord following comparable stimulation of the somatic-motor cortex (Andersen, Eccles, & Sears, 1964). In contrast to the effects described here, however, this type of presynaptic inhibition is maximal with stimulation of somatic sensory area I, and cannot be elicited by precruciate stimulation after ablation of somatic sensory area I. In any event, the diametrically opposed actions of strychnine and picrotoxin on the two cortical stimulation effects described here suggest that rather different neural mechanisms may be involved.

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Note

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