

Respiratory failure appears when the activity of the respiratory center is blocked by cold. This can be explained [3] by inhibition of the reticular structures of the medulla, which are closely connected functionally with the bulbar respiratory zone. When gradual blocking of the various parts of the respiratory center takes place during cooling, a disturbance of the coordinated regulation of the respiratory act develops and pathological forms of respiration appear. The degree of this disturbance of coordination is evidently determined by the method of cooling. In general hypothermia, when the brain is cooled by blood flowing from the internal organs, the depth of cooling and the order of inhibition of the various parts depend on the character of their blood supply. Variation in the time of extinction of functions is more marked than during craniocerebral hypothermia, when the source of cold is applied to the head. In the latter case, the lowering of the brain temperature, due mainly to the thermal conductivity of the brain tissue and its distance from the source of cold, takes place much faster and more uniformly than in the first case [4] and it is this which determines the long preservation of adequate respiration even during deep hypothermia.

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EFFECT OF EXTIRPATION OF AREA SI ON CORTICAL INTERACTION AND INTRAHEMISPHERIC RELATIONS

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Interaction between different cortical areas of the same hemisphere and intrahemispheric relations during application of stimuli of different modalities were studied by the evoked potentials method in acute experiments on unanesthetized cats immobilized with listhenon. In the intact brain influences of the somatosensory areas on visual cortical responses were shown to be mainly facilitatory in character, whereas these effects disappeared as the result of extirpation of area SI. It is concluded that functional reorganization of interhemispheric relations plays a role in the mechanisms of compensation after injury.

KEY WORDS: interhemispheric relations; compensation.

Despite many investigations [1-6, 9-13] the precise mechanisms of interhemispheric relations in the brain have not yet been explained. Nevertheless this problem is interesting from the standpoint of analysis of possible pathways of interaction and of replacement of the functions of individual systems and structures following injury. The study of disturbed brain functions from this aspect can shed light on the neurodynamic mechanisms of relations between symmetrical brain zones and may also make a contribution to the study of

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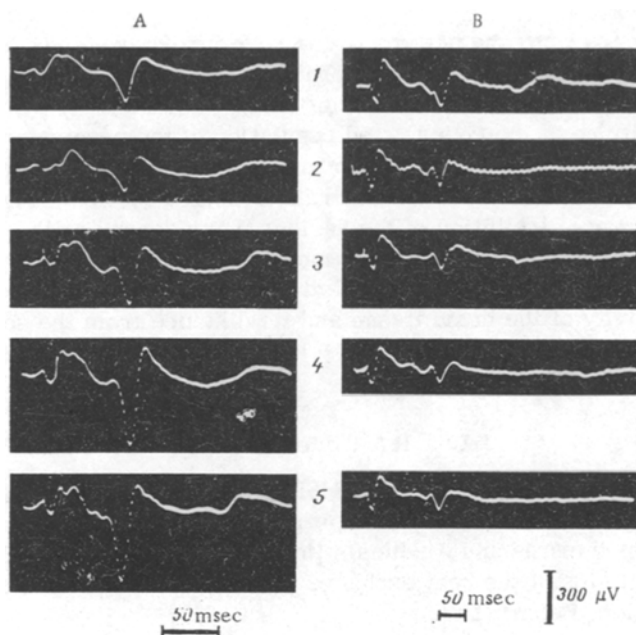


Fig. 1. Visual cortical activity during interaction between somatic and photic stimulation. A) Before, B) after subpial extirpation of area SI. 1) Response to photic stimulation. 2-5) Interval between photic and somatic stimulation 0, 20, 40, and 70 msec respectively.

the pathogenesis of repair and compensatory processes under experimental and clinical conditions. The object of this present investigation was to study interaction between homonymous and heteronymous cortical areas under normal conditions and after stimulation of a pathological process by operative removal of certain cortical areas.

EXPERIMENTAL METHOD

Acute experiments were carried out on unanesthetized cats immobilized with listhenon. The animal was fixed to a stereotaxic apparatus. Fixation points and operative incisions were infiltrated with 0.5% procaine solution. Cortical somatosensory areas I and II were widely exposed bilaterally and the parietal and visual cortex unilaterally. Unipolar derivation of potentials from the visual cortex was by a silver electrode 200 μ m in diameter. The indifferent electrode was secured to the frontal bones. The radial nerve contralaterally to the zone of recording was stimulated by pulses 0.1 msec in duration and with an amplitude 50% above the threshold.

The somatosensory area was stimulated through a coaxial electrode by single pulses 0.1 msec in duration. A single series consisting of 5 pulses, each 0.1 msec in duration and with a frequency of 0.5-1 kHz also was used. The retina was illuminated by flashes with an energy of 0.3 J and duration 100 μ sec, against the background intensity of illumination of 5 lx. The eye was atropinized. The frequency of photic stimulation was 0.2 Hz.

Summation of the evoked potentials, 30 at a time, was carried out on the ATAK-401 analog computer.

EXPERIMENTAL RESULTS AND DISCUSSION

During interaction between the afferent (somatic and visual) flows of excitation in the visual cortex phenomena of facilitation and depression of electrical activity were observed. Whereas the primary response (PR) showed very little change, the greatest change was exhibited by the so-called extrapotentia (ExP) of the visual response (according to some workers this is an element of α -like activity [5, 7, 8]). Depending on the intervals between the somatic and visual stimuli the degree of the effect of somatic stimulation on the photic responses varied. For instance, if the stimuli were applied simultaneously the amplitude of the visual potential was reduced by 15-20%; this effect remained until the interval between the stimuli increased to 15 msec,

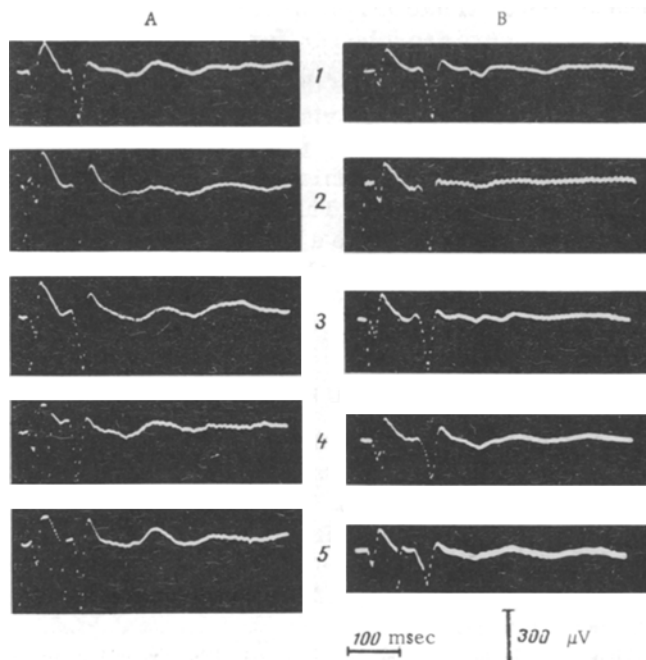


Fig. 2. Interaction between transcallosal and photic stimuli in visual area with cortex intact (A) and after experimental injury (B). 1) Response to photic stimulus. 2-5) Interval between photic and transcallosal stimuli 0, 20, 40, and 60 msec respectively.

after which the amplitude of the ExP was gradually restored (Fig. 1A). Under these circumstances it increased to approximately twice its initial value. As Fig. 1A shows, the duration of ExP also varied: it increased with a decrease in the amplitude of the response and vice versa. At maximal amplitude its duration was 18-19 msec, and at minimal amplitude 22-23 msec.

Investigation of interaction between visual and transcallosal excitation revealed considerable modulation of the photic response in the visual cortex by the transcallosal stimulus. The change in amplitude of the visual response occurred against the background of an almost constant facilitatory action. The facilitatory effect was stronger the longer the intervals by which the transcallosal stimulus preceded the photic stimulus (Fig. 2A). For instance, if the transcallosal stimulus precedes the photic by 100-200 msec the amplitude of ExP was 175% of the amplitude of the response recorded to the photic stimulus alone. The increase in the negative phase of ExP could reach 300%. The latent period was 17-18 msec for PR and 84-87 msec for ExP.

To analyze the mechanisms of transcallosal modulation experiments were carried out with extirpation of area SI. Extirpation of this area on the same side as the visual responses were recorded caused a marked decrease in the amplitudes of PR and ExP during the first minutes, but after 15-20 min a tendency toward their recovery appeared, somewhat later in the negative phases. Recovery of the responses was observed after 0.5-2 h.

After extirpation of cortical area SI the latent period of ExP increased to 105-115 msec (Figs. 1B and 2B). A decrease in facilitatory transcallosal influences also was observed.

The experiments showed that an evoked potential consisting of a positive-negative complex with a latent period of 18 ± 1 msec was recorded in the visual cortex in response to flashes; a significant increase in the amplitude of the response was observed after interaction with somatic or transcallosal stimuli. This interaction between two afferent flows evidently forms a focus of enhanced excitability in the cortical structures of the visual analyzer, as a result of which the amplitude of the primary and secondary responses, including the ExP, is increased.

The organization of the pathways transmitting influences of the somatosensory and transcallosal stimuli on the visual response differs. Although their action on the secondary components of the visual response (especially on ExP) is similar, their effect on PR is different. Stimulation of the radial nerve changed the

amplitude of PR more than transcallosal stimuli (Figs. 1A and 2A). This may be due to the fact that in the latter case subcortical structures participate to a lesser degree in the formation of PR.

It can thus be concluded from these experiments that transcallosal stimuli have a modulating effect on the secondary components of evoked responses in the visual cortex. After the creation of an experimental pathological focus (extirpation of or injury to area SI) these interhemispheric influences are disturbed and no longer affect the electrical activity of the opposite hemisphere. These facts are evidence that extirpation of area SI modifies the configuration of the visual ExP. This, in turn, may be connected with a disturbance of transcallosal impulses which, in the intact brain, have a facilitatory action on the secondary components of the evoked response. The results suggest that transcallosal interhemispheric influences are considerably disturbed following injury to cortical area SI and that the dynamics of their changes may play an important role in the restoration of disturbed brain functions.

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