THE APPEARANCE OF ASYMMETRY IN THE ELECTROENCEPHALOGRAM OF THE CEREBRAL HEMISPHERES AFTER UNILATERAL INJURY OF THE LUNGS

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(Received October 11, 1958. Presented by Active Member AMN SSSR V. N. Chernigovskii)

In previous research in this laboratory [8], it was shown that thermal injury of the lungs produces disturbance of both conditioned and unconditioned reflexes. It would therefore be expected that injury of the lungs would also involve a change in the electrical activity of the cerebral cortex. The aim of the present investigation was to study the electrical activity of the cerebral cortex during burn injury of the lung.

In the clinical literature, there are several papers describing the changes in the electrical activity of the cerebral cortex in pneumonia, tuberculosis and bronchial asthma [1, 5, 7, 9, 11, 13, 14, 22, 25-27, 28]. In these clinical investigations, the changes in the electrical activity of the cerebral cortex could have been the result of several disturbances affecting the body during diseases of the lungs and exerting a direct influence on the cortex– anoxemia, toxemia, fever.

There are only two experimental papers in which changes are described in the electrical activity of the brain in association with lesions of the lungs.

In one of these [17], changes in the electrical activity of the brain were observed during an attack of experimental asthma in guinea pigs. In another work [3], changes in the electrical activity of the brain were noted in pneumonia in rabbits. The pneumonia was induced by stimulation of various nerve formations (by suboccipital injection of turpentine emulsion, or by crushing the superior cervical sympathetic ganglia), which could themselves affect the electroencephalogram.

EXPERIMENTAL METHOD

In our experiments, the lung was injured by injection of hot water (80-90°) into its substance, in a volume of 3-4 ml from a syringe through the chest wall. As previous laboratory investigations showed, the disturbances of the bodily functions arising under these circumstances were due to a pathological flow of impulses from the lesion to the central nervous system.

Various investigations [2, 6, 12, 15, 18] have shown that the electrodes in common use, when inserted into the various subcortical structures, into the cortex, or epidurally, cause considerable functional and morphological changes in the vicinity of the lead. For this reason, certain workers [4, 6] tap the cortical potentials through the thin bone of the cranium. We also adopted this method of tapping the cortical potentials. For this purpose, we used buried electrodes, consisting of hollow steel cylinders, 2.5 mm in diameter and 2.5 mm long. Inside each cylinder, there was a screw thread to which the wire taking the potentials was screwed before the experiment.

At the base of the cylinder was a needle-shaped projection, 0.75 mm long. Before insertion, the base of the electrodes, as well as the needle-shaped projection, were coated with an insulating layer of BF-2 glue or of soluble plexiglas. As soon as they had been coated with glue, the electrodes were driven into the previously exposed skull bones by light blows of a hammer, and then fixed with phosphate cement. The short,needle-shaped projection of the electrode enabled the cortical potentials to be tapped through a thin layer of bone of the skull.

The potentials were tapped by bipolar leads (interelectrode distance 6-8 mm)from the frontoparietal and occipital areas of each hemisphere and fed into an amplifier with a linear frequency characteristic of 0.2 to 200 cps. The recordings were made on an ink apparatus with a linear frequency characteristic of 0 to 70 cps.

The animal was kept in a screened room, in a narrow box which restricted its movements but which did not interfere with its natural position. Having the animal free and unfixed in this way enabled the various stages of the change from alertness to sleep to be observed without the use of drugs. After the animal had become accustomed to the experimental conditions, in the course of 2-3 days, a recording was made of the action potentials of the brain and of respiration for a period of $1\frac{1}{2}$ - 2 hours. The lung was then injured, after which the electroencephalogram and respiration were recorded for 4-5 hours and then for an hour on the following 2-3 days. The experments were carried out on 13 rabbits.

EXPERIMENTAL RESULTS

In the intact rabbit accustomed to the experimental conditions, the electroencephalogram showed periodic bursts of fairly frequent (15-20 oscillations per second) high-voltage (80-100 μv) waves – the so-called sleep "spikes" (Fig. 1, A).

In the frontoparietal area, the voltage of the spikes was higher than in the occipital area. In addition, the spikes in the frontoparietal area were usually from two to three times more frequent than in the occipital area. Irrespective of the location of the electrodes, the sleep spikes in the intact rabbit almost always appeared symmetrically, i.e.,at the same time in both hemispheres; in only a few rabits were one or two or, in extreme cases,three asymmetrical spikes seen amid 100-200 symmetrical spikes. Asymmetry was shown by the fact that the appearance of a spike in one hemisphere was accompanied by an indefinite rhythm in the other.

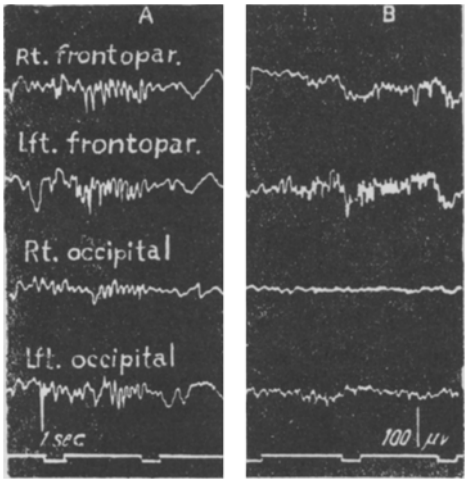

Fig. 1. So-called sleep "spikes" in the electroencephalogram of an unanesthetized rabbit. A) before injury to the lung; B) 35 minutes after injury to the left lung.

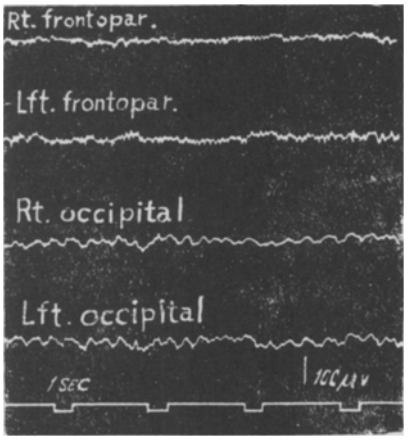

Fig. 2. Desynchronization in the electroencephalogram of the frontal areas of the brain of the rabbit after injury to the lung.

Thermal injury to the lung led to desyncronization, usually lasting 10-30 minutes (Fig. 2). Desynchronization was characterized by the disappearance of the slow, high-voltage waves and spikes and by the appearance, mainly in the frontoparietal area, of well-developed low-voltage rapid (30-40 oscillations per second) rhythms. In the occipital leads,clear oscillations appeared, with a frequency of 5-7 per second.

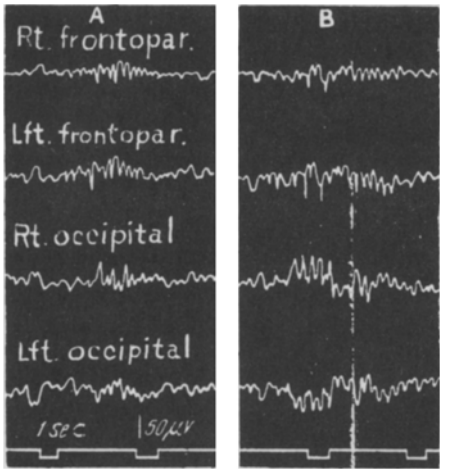

Fig. 3. Spikes in the electroencephalogram of the unanesthetized, vagotomized rabbit. A) before injury to the lung; B) 20 minutes after injury to the right lung.

Novocain infiltration of the site of puncture neither prevented nor shortened the duration of desynchronization. The desynchronization was obviously the result of stimulation of the afferent pathways of the lung. In view of the fact that the main afferent pathways from the lung pass along the vagus nerve, it might be thought that the desynchronization which we observed was the result of impulses passing along the vagus nerve to the brain. This was the conclusion reached by Moruzzi et al. [29] from their experiments. It was shown in these experiments on the "encephale isole" preparation of the cat that stimulation of the vagus nerve caused desynchronization of the activity of the whole cortex.

Usually, from 10 to 30 minutes after injury to the lung, desynchronization gave way once again to a slower, synchronous rhythm, which was replaced by sleep spikes. In contrast to the intact rabbits, however, in which these spikes appeared simultaneously in both hemispheres in the overwhelming majority of cases, in rabbits with injury to the lung an obvious asymmetry was observed in their features (see Fig. 1, B). This asymmetry was shown by the fact that with the appearance of spikes in the ipsilateral hemisphere, oscillations were observed in the contralateral hemisphere, which revealed some degree of desynchronization. Asymmetry was usually observed during the transition from the stage of waves with an indefinite rhythm to the stage of spikes. At this time, half or even two thirds of all the spikes were asymmetrical: in the electroencephalogram of the contralateral hemisphere they were absent. The number of asymmetrical spikes then gradually decreased, and 2-3 hours after injury, asymmetry of the spikes was revealed only from time to time as a delay in their appearance in the contalateral hemisphere by comparison with the ipsilateral. From 3 to 4 hours after injury to the lung, the symmetry of the bioelectrical activity of the hemispheres was usually restored.

The asymmetry just described was not the result of stimulation from which impulses proceed along afferent nerves supplying the tissues damaged by the needle of the syringe as it entered the lung, for insertion of the needle alone, without infliction of a burn to the lung, caused no asymmetry of the spikes.

It was naturally suggested that the cause of the asymmetry was impulses passing from the site of injury to the lung along the vagus nerve to the brain.

In order to investigate this suggestion, in the next series of experiments, the lung was injured after a preliminary bilateral vagotomy. Division of both vagus nerves in the cervical region itself caused desynchronization, which lasted for many hours if the operation was performed without anesthesia. If the operation was carried out under local anesthesia, then only 40-60 minutes after division of the vagus nerves, the spikes reappeared, and were even more clearly shown than before the division: the spikes had a greater amplitude and were seen more frequently than in the intact rabbits. Injury to the lungs of the vagotomized animals did not cause even a hint of asymmetry of the spikes in even one experiment (Fig. 3).

The facts presented demonstrated first, that asymmetry of the spikes apppearing after injury to the lung was in fact the result of a spread of impulses from the site of injury in the lung along the vagus nerve, and secondly, that the afferent pathways from the lungs to the frontoparietal and occipital areas of the cortex were crossed.

According to the modern view, desynchronization is the result of stimulation of the reticular formation, which has a generalized action on the whole cortex. Among the many papers devoted to investigation of the reticular formation, in a few, is described the appearance of a more or less limited desynchronization. Jasper and Penfield [23] and, later, Gastaut [20] for instance, described a local desynchronization appearing in the cortical representation of the upper limb of the unanesthetized human subject during movement of the

corresponding limb. Gellhorn et al. [10, 21] reported desynchronization of the activity of the sensomotor area of the cortex in response to nociceptive and proprioceptive stimulation of the hindlimb in cats and monkeys under moderate barbiturate anesthesia. Ectors [16] described the local desynchronization of cortical activity in response to weak visual, tactile, or olfactory stimulation. Moruzzi and Magoun [24] observed desynchronization of the activity of the ipsilateral cortex only in response to weak stimulation of the reticular formation of the brain stem in cats.

In the experiments of French et al. [19], stimulation of the brain stem of monkeys caused desynchronization of the activity of mainly, the frontoparietal area of the cortex. In some cases, local desynchronization of the activity of the frontoparietal area of the cortex was observed in response to stimulation of the sciatic or splanchnic nerves; such stimulation never produced desynchronization of the activity of the temporal or occipital areas of the cortex.

Our results also showed that peripheral stimulation may lead to limited (to the hemisphere contralateral to the injured lung) desynchronization.

It may be concluded from our experimental findings that the change in the EEG observed during lesions of the lungs may be the result not only of anoxemia, toxemia, or fever, as several authors have pointed out, but also of nervous impulses from the site of injury.

SUMMARY

A pronounced asymmetry of the bioelectric activity occurs during the first 10–30 minutes following thermal injury of the lung. It is manifested by the appearance of sleep spindles in the hemisphere, ipsilateral to the injured lung. Oscillations without a definite rhythm are observed in the contralateral hemisphere, testifying to a certain desynchronization. Lung injury inflicted to previously vagotomized rabbits does not cause the above asymmetry. Thus, the impulsation transmitted along the vagus from the site of lung injury may cause a limited desynchronization in the hemisphere contralateral to the injured lung.

A conclusion is drawn that the EEG changes observed in lung affections may be due not only to hypoxemia, toxemia, or fever (as pointed out by a number of authors), but also to impulsations from the focus of injury.

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