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Brain Stem, E. E. G., and Cortical Vasomotor Reactions

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ABSTRACT

The data here presented indicate that stimulation of the brain stem reticular formation is accompanied by vascular changes leading to increase of cortical temperature. From comparison with other factors whose influence on cerebral vessels is known, it seems likely that either increase in blood flow or vasodilation may occur. Such vascular responses generally, but not always, accompany arousal EEG patterns. The study of possible neurohumoral mechanisms responsible for such phenomena is complicated by many factors. My results indicate that acetylcholine rather than adrenergic compounds may be involved.

A great deal of information has accumulated on the brain wave changes produced by stimulation of the brain stem reticular formation, but even today the anatomic and functional significance of the structures involved is not clearly understood.

First, there is the problem of anatomical location and organization of the structures mediating the EEG arousal response, secondly, the problem of effects other than the EEG changes which accompany the arousal response, and finally, the question of neurohumoral transmission of such responses. I planned to investigate these problems and I present some of my results.

MATERIAL AND METHODS

The animals used were cats. In a group of these, electrolytic or surgical lesions were made at different levels of the brain stem reticular formation and the EEG was recorded with implanted electrodes for several days following the lesion. These and other cats, without lesions, were used for acute experiments during which, in addition to the brain waves, the EKG, vascular changes of the cortex, blood pressure steady cortical potential, and the effects of various drugs were studied. The cortical vascular changes were measured with thermistors, the steady potential with non-polarizable electrodes, and the blood pressure with a calibrated strain gage.

OBSERVATIONS

This chart is from an animal in which a large lesion of the left reticular formation in the caudal

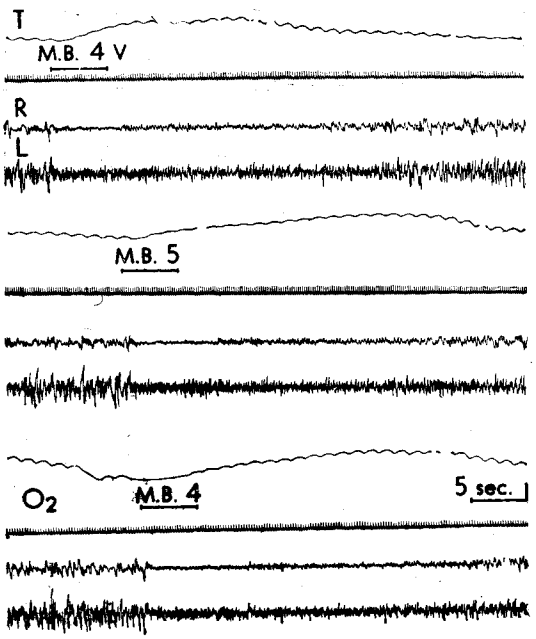

Fig. 1

part of the midbrain was made one week previously. It can be seen that the left hemisphere shows a more synchronized brain wave pattern than the right. Stimulation of the midbrain in front of the lesion on either side results in a clear arousal response of both hemispheres and an increase in cortical temperature, which seems to last longer when the stimulus is increased in intensity. The lower records were taken following oxygen breathing during the time that cortical temperature was falling. Again the midbrain stimulation produced an increase of cortical temperature and a clear arousal reaction. This animal had the spinal cord transected at C_2 and was under artificial ventilation.

This picture shows the changes in cortical temperature (T), steady potential (SP), and brain waves produced by midbrain reticular formation stimulation

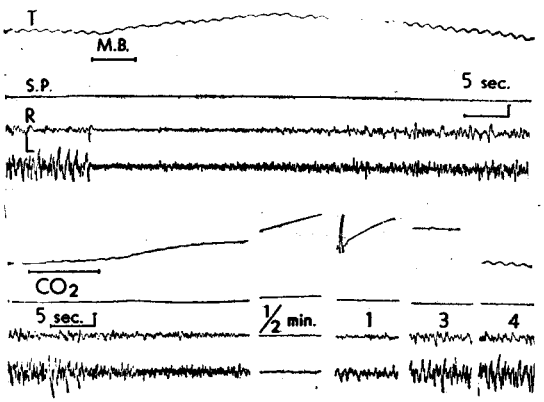

Fig. 2

and by a brief administration of CO_2. This latter procedure resulted in more pronounced and much longer-lasting changes in both cortical temperature and steady potential. The direction of change is, however, similar. It is interesting to note that, following CO_2, there is an arousal response not as clear as following midbrain stimulation and that the cortical temperature returns to its initial level later than the brain wave pattern. Similar changes were seen comparing midbrain stimulation to those produced by apnea.

This picture shows that increase in cortical temperature may occur following brain stem stimulation without prominent changes in brain wave pattern.

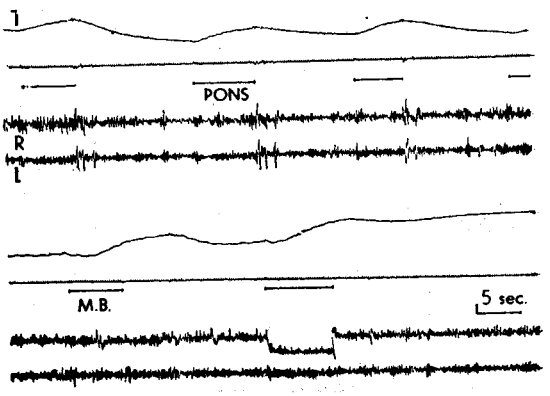

Fig. 3

A certain degree of arousal is possibly present following each of the stimulations to the pons, in the upper records, but entirely absent following stimulation of the midbrain, which produces clear increases in cortical temperature.

These and many other data indicate that brain stem stimulation results in cortical vasomotor changes, generally, but not always, concomitant with an arousal response. The possibility was considered that such vascular reactions may depend on the release of neurohumours since several lines of evidence indicate the presence of adrenergic and cholinergic systems in the reticular formation.

I have seen repeatedly that, when epinephrine or norepinephrine is injected systemically, the cortical temperature increases with the increase of systemic blood pressure, in animals with spinal cord either intact or transected.

Intracarotid injections of small amounts of norepinephrine (10 gamma) generally did not produce clear changes in brain wave pattern or in cortical temperature. In some instances, however, we saw the cortical temperature increase following the injection of norepinephrine in the carotid artery before the systemic pressor effect became evident.

This picture shows one of these instances and it can be seen that such increase in cortical temperature occurs for these two rather large doses (20 and 10 gamma), that it is more prominent and rapid on the injected side (the right), and that it precedes the raising of the systemic blood pressure.

In my experience intracarotid injections of acetyl-

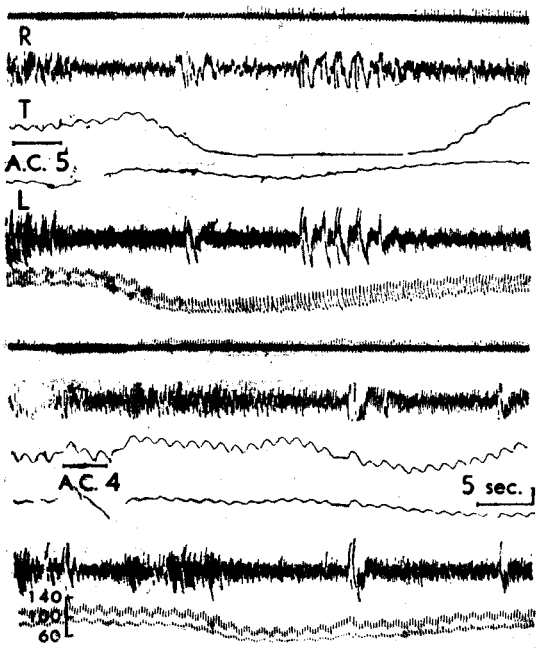

Fig. 4

choline, even in very small doses (3—4 gamma), produced an arousal reaction in most instances. At the time of the fall in systemic blood pressure the brain temperature did not follow closely such a systemic fall, and when it did occur it was generally delayed. It was evident, however, that on the side of the carotid cannulation the systemic fall blood pressure was more promptly reflected in the decrease of cortical temperature.

This chart shows the effects of the intracarotid injection of 50 and 12 gamma of acetylcholine. The arousal response and the behavior of the blood pressure and brain temperature is similar in both instances. Cortical temperature increases initially more rapidly on the injected side (the right), then falls with a delay of a few seconds after the systemic drop in blood pressure. On the left side there is no evidence of such a phenomenon.

In this example, smaller dosages of acetylcholine were injected with essentially similar results, indicating that the limited blood supply of the right hemisphere on the side of the carotid injection was

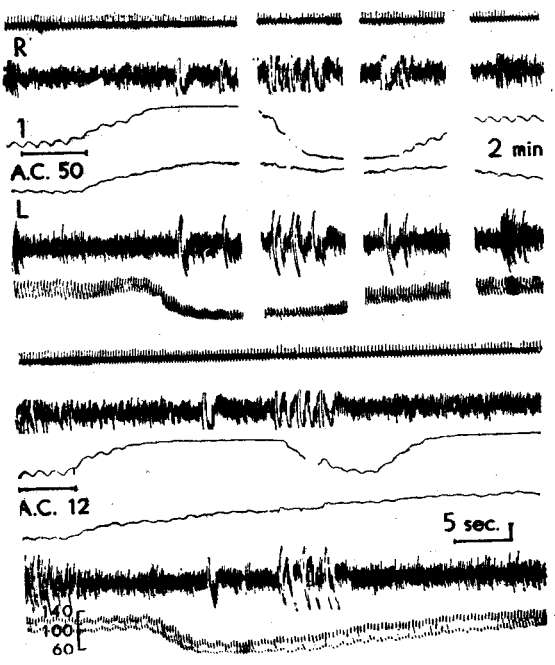

Fig. 5

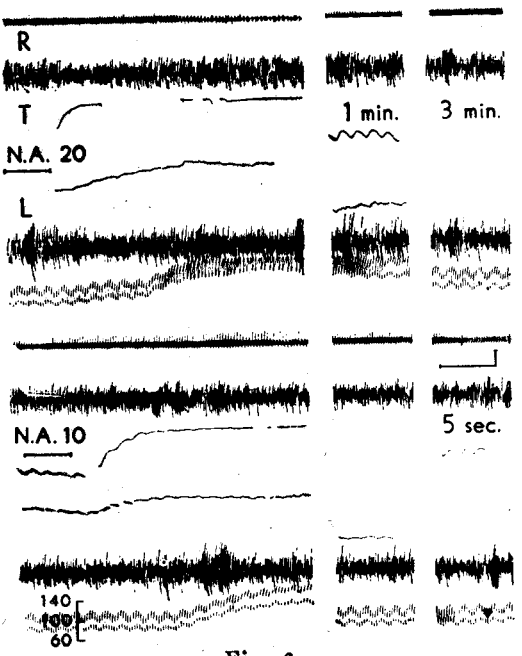

Fig. 6

responsible for the fall in brain temperature, which follows the drop in systemic blood pressure.

I have seen that atropinization abolishes this arousal response to acetylcholine. In addition, the systemic fall in blood pressure is reduced, and the decrease of cortical temperature is simultaneous with this fall.