THE EFFECT OF ASPHYXIA ON THE ELECTROCORTICAL EFFECTS OF ACETYLCHOLINE

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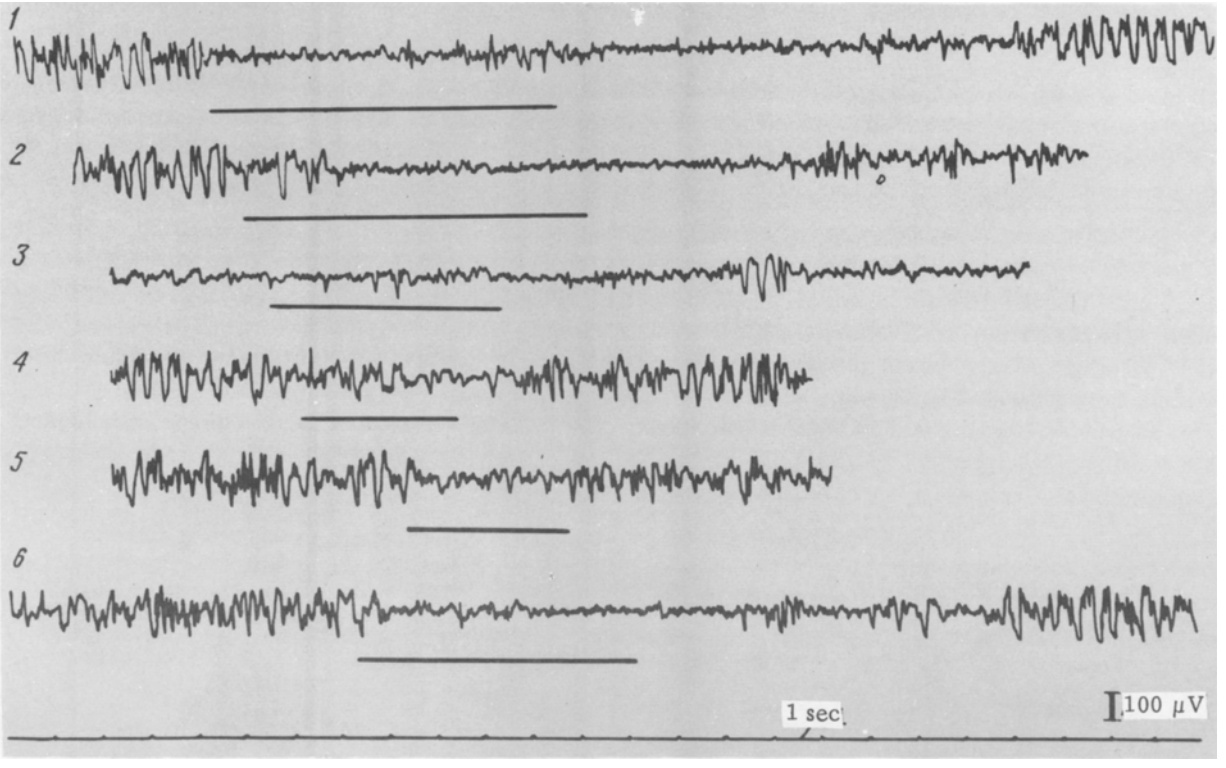
Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 55, No. 3,
pp. 27-29, March 1963
Original article submitted March 5, 1962

In agreement with reports in the literature [5-10], in experiments which we have performed the intracarotid injection of acetylcholine in a dose of 2-10 μg into intact rabbits led to a well marked desynchronization reaction (see figure, 1). Asphyxia caused desynchronization of the cortical rhythms, the degree of which varied with the period of its development. In the initial stage, before marked desynchronization was present, the electrocortical effect of acetylcholine was slightly reduced in duration (see figure, 2). With deepening asphyxia, against the background of some degree of desynchronization of the cortical activity, the injection of acetylcholine increased desynchronization, but this effect was ill defined (see figure, 3). In the profound stages of asphyxia, when slow waves appeared in the electrocorticogram, the electrocortical reaction to acetylcholine was either absent or still less clearly shown.



Changes in the electrocortical effects of acetylcholine. 1) Before asphyxia; 2) 1 min after the beginning of asphyxia; 3) 3 min after the beginning of asphyxia; 4) 30 sec after cessation of asphyxia; 5) 2 min after cessation of asphyxia; 6) 10 min after cessation of asphyxia. The continuous line denotes the time of injection of acetylcholine.

The disappearance of the electrocortical reaction to acetylcholine during asphyxia was a reversible phenomenon, for after stopping the asphyxia, when the electrocorticogram gradually returned to its initial level, the reaction to acetylcholine reappeared. At first it was brief and weak (see figure, 4, 5), but later it became similar to its initial state (see figure, 6). It should be noted that during asphyxia and in the course of the restoration of functions after its cessation, against the background of the desynchronization of cortical activity which it causes, the administration of acetylcholine sometimes gave rise to an electrocortical reaction in the form of a burst of synchronized potentials of high amplitude and slow rhythm.

We were thus able to confirm the development of desynchronization of cortical activity in response to the intracarotid injection of small doses of acetylcholine. Irrespective of the mechanism of this reaction, i.e., whether it was the result of activity directed towards the cholinergic systems of the reticular formation [7-10] or the result of stimulation of the receptors of the blood vessels, its realization was disturbed during asphyxia.

The reason for this disturbance may lie both in oxygen deficiency and in excess of carbon dioxide. We know that hypercapnia may itself cause excitation of the reticular formation and desynchronization of cortical activity [4]. It is probable that the desynchronizing action of acetylcholine is made more difficult with this as a background. Admittedly, in our experiments depression of the reaction of desynchronization to administration of acetylcholine took place before the concentration of carbon dioxide in the inspired air had reached high values. Consequently, the cause of the depression of this reaction, in our opinion, is more likely to be lack of oxygen than excess of carbon dioxide.

The cases in which a burst of synchronized potentials, and not the desynchronization of cortical activity, appeared in response to the injection of acetylcholine call for a special examination. These bursts of potentials have been observed by several writers in the anterior divisions of the brain in response to stimulation of the interoceptors [1, 3] or during the reaction to sensory stimulation against the background of the action of bromides [2]. The most probable explanation of this fact must be sought in N. E. Vvedenskii's teaching of the functional mobility of the tissues, according to which the same stimulus may evoke different reactions depending on the functional state of the central nervous system.

SUMMARY

An inquiry was made into the effect of asphyxia on the electrocortical effects of acetylcholine. Experimental results demonstrated that asphyxia development induced a gradual reversible disappearance of electrocortical effects of acetylcholine. During the process of functional restoration they returned to the intitial condition. Against the background of desynchronization of the cortical activity in asphyxia and functional restoration in intracarotid acetylcholine administration,there occurs sometimes an outbreak of synchronized potentials of considerable amplitude and slow rhythm.

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