

Relation of Cerebral Dysrhythmia to Eclampsia.*

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The etiology of eclampsia is unknown. Many aspects of the clinical picture of the eclamptic syndrome suggested the necessity for electroencephalographic studies. This report deals with the electroencephalographic findings and family histories of a group of 17 patients in whom a diagnosis of eclampsia was made because of convulsive seizures associated with the latter part of the puerperium.

These patients were studied from one week to 5 years after their hospital admissions. In one instance the tracing was taken one week after the eclampsia, in 2 cases the recording was made 10 days after the eclampsia, and in the remaining instances the electroencephalograms were made from 3 months to 5 years after the patients' admission for eclampsia. The intervals are adequate in most, if not in all instances, to exclude post-convulsive changes as a cause for the abnormal tracings.

The electroencephalographic technic consisted of 6 lead monopolar tracings, using the lobe of the ear as a reference point. The leads were taken from both frontal, parietal and occipital regions. Short periods of hyperventilation were included in each record. From the scalp electrodes the potentials were led off to Grass amplifiers and recorded on a 3-channel ink writer.

Preliminary analyses of the records, using the standards suggested by Gibbs,¹ Jasper,² Davis,³ and others,⁴ which include frequency, amplitude, wave form, and general stability, indicated that 13 out of 17 tracings were abnormal, *i. e.*, 77%. Two tracings were within normal limits and 2 were considered borderline.

Similar tracings were taken from 10 patients who had had mod-

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¹ Gibbs, F. A., *J. Psychology*, 1937, **4**, 365.

² Jasper, H., and Kershman, J., *Arch. Neurol. and Psych.*, 1941, **45**, 903.

³ Davis, P. A., *J. Neurophysiol.*, 1941, **4**, 92.

⁴ Gibbs, F. A., and Gibbs, E. L., *Atlas of Electroencephalography*, Lew A. Cummings Co., Cambridge, Mass., 1941.

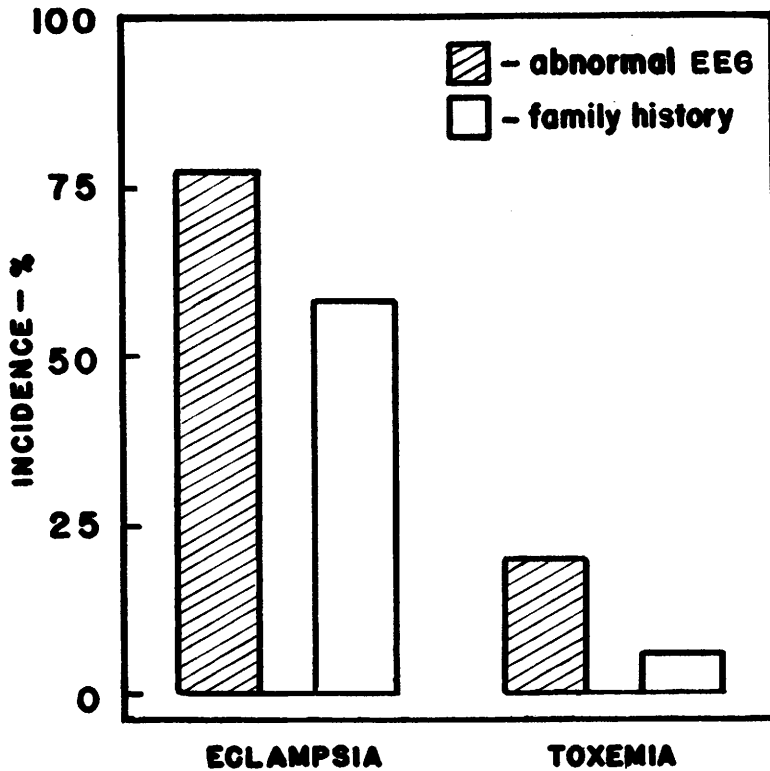


FIG. 1.

Illustrating the incidence of cerebral dysrhythmia and a family history of convulsive disorders in patients who have had eclampsia and toxemia of pregnancy.

erately severe to severe toxemia of pregnancy without convulsions (Toxemia—grade II). In this group there was only one record that was abnormal and 2 that were borderline or questionable.

A careful history taken on both groups of patients revealed that in 10 of 17 women with eclampsia there was a history of convulsive seizures either in the patient prior to her eclampsia or in some member of her immediate family. In only 2 patients was there a history of convulsive seizures prior to the pregnancy in which the diagnosis of eclampsia was made. In the group with toxemia alone there was only one instance with a family history of convulsions.

This preliminary study suggests that there may be a primary cerebral dysrhythmia present in those patients having the syndrome of eclampsia, and that the associated toxemia may be the "trigger mechanism" that exaggerates the inherent dysrhythmia to the degree that convulsions appear. In accord with this is the fact that many of the methods that have been successful in the therapy of eclampsia have depended upon the use of anticonvulsant measures.

The above data do not refer in any regard to the etiology of the toxemia, but do suggest that those patients who develop convulsions (eclampsia) in the course of their toxemia may have a primary cerebral dysrhythmia.

It may be inferred from the preceding that a careful history together with an electroencephalogram may be of great importance in determining those patients who may develop eclampsia. Furthermore, it is apparent that the proper therapy of eclampsia and pre-eclampsia may include those measures generally employed in the treatment of cerebral dysrhythmia. In accord with this, studies are now in progress to elucidate the action of dilantin and other anticonvulsants in the eclamptic and pre-eclamptic states.

Summary. Seventy-seven percent of 17 eclamptic women had electroencephalograms indicative of cerebral dysrhythmia. Fifty-eight percent of this series gave a family history of convulsive disorders.

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Effect of a Vacuum on Destruction of Bacteria by Germicides.

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The presence of a vacuum was found to have no effect on the destruction of bacteria by germicides. Regardless of whether the organisms were mixed with the germicidal dilutions and kept at atmospheric conditions or placed under a vacuum, the final results were the same.

Entirely different results were obtained, however, when gaseous germicides were employed. Tests carried out over a period of about 15 years have shown that the efficiency of the gases was greatly increased in the presence of a vacuum. In fact, under certain conditions organisms could not be destroyed unless a vacuum was employed.

Experimental. The gases which were tested included formaldehyde, methyl bromide, methyl formate, ethylene oxide, and carbon disulfide. With the exception of formaldehyde no one of the gases effected a sterilization after an exposure period of 2 hours. These non-germicidal gases are important for the destruction of insects