

MR 19 06 ✓
G. Glaser

The EEG records which show abnormalities of various types and their topographical pattern in relation to skull lesions are discussed.

diencephalon, electroencephalographic records before and after such treatments have been taken. In most cases no changes have been observed, sometimes only an increase in the voltage has appeared with burst of theta waves and instability on overbreathing.

26. Continuous EEG recording during insulin coma. — C. L. CAZZULLO and V. NEGRI, Milan.

In a group of 15 schizophrenic patients continuous EEG recording during insulin coma were carried out. The observation period lasted from 6 to 8 hours. The EEG patterns are analyzed and correlated to the changes of the state of consciousness and to the blood sugar level.

31. EEG changes induced by injection of cerebrospinal fluid of schizophrenic and epileptic patients. — A. POLINI, Bergamo.

The intravenous injection of 10-20 ml. of C.S.F. taken from an epileptic or catatonic patient causes, in a certain number of cases an activation of the normal and the pathological activity in the EEG. C.S.F. from hebephrenic and paranoid patients causes decrease in frequency and sometimes increase in voltage if injected in normal subjects. The same C.S.F. causes when injected to epileptic patients a diminution of grand mal.

27. EEG and chorea of Sydenham. — R. VIZIOLI and B. MONDOVI, Rome.

In a group of patients with Sydenham's chorea unspecific abnormalities were found. They were all very slight. There was no particular correlations with the fact that the patient had suffered from rheumatism.

32. Electroencephalographic modifications following intravenous injection of nicotinic acid. — C. SERRA and A. SCOPPA, Naples.

The authors have studied the electrical activity during the injection of nicotinic acid and within 20 min. after the intravenous administration of the drug.

The results observed in the 20 subjects examined were as follows:

1. A constant increase of the amplitude of the alpha waves especially in the parietooccipital and central regions (mainly after the administration of 45-60 mg. of the drug). These changes were detectable until the end of the period of observation.

2. In some instances, beside the above mentioned modifications, there were also some paroxysmic discharges of theta and alpha waves in the parietooccipital and temporal regions.

On the basis of the above mentioned findings the authors think that the drug produces some metabolic modifications (especially upon sugar content of the brain tissue) responsible for the EEG modifications.

28. Photic stimulation in photogenetic epilepsy. — F. PAPARO, Rome.

Six cases of photogenetic epilepsy are reported. Four patients were submitted to EEG studies. In three cases there was a pattern typical of idiopathic epilepsy; in the fourth case no abnormality was seen in the resting record or with hyperventilation. Bilaterally synchronous spike and wave complexes appeared only with photic stimulation.

The author examines the value of photic stimulation in idiopathic epilepsy and concludes that diagnosis of photogenetic epilepsy has to be made on the photic origin of the spontaneous clinical seizures. Evidence of EEG and clinical activation with photic stimulation is not restricted to this kind of idiopathic epilepsy.

29. EEG and infantile hemiplegia. — R. VIZIOLI and E. MARCHINI, Rome.

Fifty cases of infantile hemiplegia were studied from the EEG standpoint. Contralateral depressed activity was found in many cases. A low percentage of epileptic specific abnormalities were found also in patients suffering from clinical epilepsy.

The hypothesis that severe involvement of brain may impede the transmission of epileptic activity is stressed.

30. EEG after radar-application. — L. SINISI, Geneva.

In some patients affected by mental illness and treated with micro-waves (radar) focussed on the

33. Electroencephalographic and clinical study of a case of "geloplegia". — R. D'ORIO and G. RICCI, Rome.

(a) Akinetic crises caused only by laughing. Recent association with hypersomnia.

(b) Difficulty to demonstrate EEG changes at rest or with hyperventilation.

(c) Hypersensibility to activation with intravenous Carbazol (1/2 cc. at 5 per cent).

(d) Discussion.

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