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THE RESEARCH ASPECT OF THE EPILEPTIFORM SYNDROME

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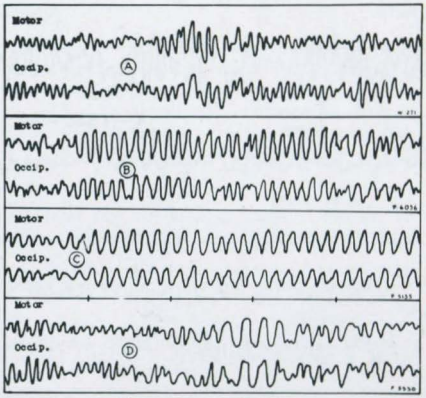
During the Second Great War, routine electroencephalograms done on members of the Royal Canadian Air Force revealed that 10-15 out of every 1000 (approximately 1 percent) showed electro-cerebral activities indistinguishable from those associated by Gibbs et al with psychomotor epilepsy Fig. 1 and Fig. 2. This group



Elements of Psychomotor Patterns

Fig. 1

of individuals certainly represented an above average sample of the general population. Their records showed no physical or psychiatric abnormalities. Many were re-interviewed by a psychiatrist and/or neurologist. There was only one in a group of over 2000 who gave a history of attacks which in any way resembled psychomotor states. Goodwin[1] reported the findings in the electroencephalograms. He noted that the slow square topped waves were occurring at exactly one-half, and the fast variant at double the concurrent alpha rate. Typical examples of the slow and fast variants are shown in Fig. 3.

Then 550 presumably normal individuals were chosen at random. These did not have the thorough screening utilized by the R. C. A. F. group. It was found that the recordings of 10 percent of these subjects contained traces or more of

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the alpha variants. Subjects showing alpha variants were thoroughly examined, and those having clinical signs or symptoms associated with the EEG changes came under the broad headings of psychoneurotics and/or epileptics.

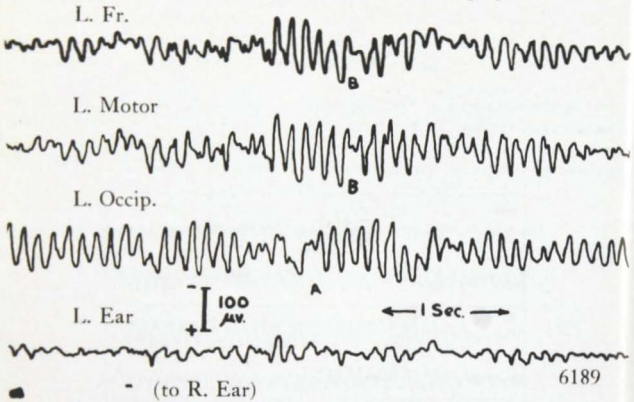


Fig. 2

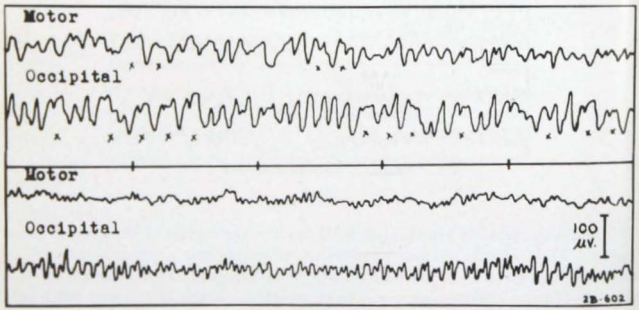


Fig. 3

Table 1 shows the percentage of the patients in these groups demonstrating alpha variants in their EEG. It was not surprising to find 73 percent of epileptics with this EEG abnormality, but we did not understand the high incidence in the psychoneurotic group.

After some 12 years of observation the so-called psychoneurotics in this group proved to have developed episodic disorders, 95 percent of which had varying degrees of impaired consciousness. Several intractable migraine cases in the group responded to appropriate anti-epileptic therapy.

TABLE 1

INCIDENCE OF THE ALPHA VARIANTS (FAST AND SLOW) IN THE ELECTROENCEPHALOGRAMS OF VARIOUS POPULATIONS

GROUP	NO. OF CASES	PER CENT WITH ALPHA VARIANTS
SELECTED NORMAL	550	10%
PSYCHONEUROTICS*	94	45% ($P < .01$)
EPILEPTICS*	134	73% ($P < .01$)

*FINAL DIAGNOSIS.

There have also been several cases of fugues or compulsive behaviour which one would expect in psychomotor attacks. The psychoneurotic features in many cases were either initiated by or contributed to through an episodic impaired consciousness which produced understandable anxiety. This often disappeared under appropriate anti-epileptic medication. Proctor[2] reported the response to sodium dilantin, sodium amytal and phenobarbiturate, singly or in combination (Table 2).

TABLE 2

CLINICAL RESULTS (Preliminary Report November 28, 1946)
Response of 132 patients with psychomotor syndrome to various types of medication.

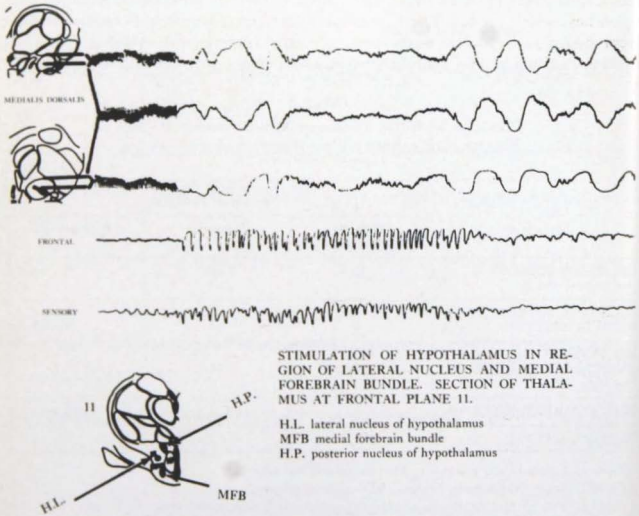
Medication	Number of Patients		
	Markedly Improved	Improved	Unimproved
DILANTIN SODIUM (see notes 1 and 2)	27	10	5
DILANTIN SODIUM plus SODIUM AMYTAL	2	3	0
DILANTIN SODIUM plus PHENOBARBITURATE (see note 3)	32	14	1
PHENOBARBITURATE	1	7	14
SODIUM AMYTAL	0	7	9

Note 1: 4 cases in this group may have had grand mal attacks.
Note 2: Drug withdrawn in 7 cases. All 7 patients relapsed.
Note 3: 80% of this group are mixed psychomotor-idiopathic grand mal. None are purely idiopathic grand mal.

It is noteworthy, that the patients responded selectively to sodium dilantin and showed a very poor response to sodium amytal or phenobarbiturate. The latter two drugs should have been beneficial if we had been dealing primarily with a psychoneurotic syndrome. In discussing our findings with Dr. Wilder Penfield in 1947, he agreed that we may be dealing with the phenomena which Gowers[3]

described in 1907 as the borderlands of epilepsy. Also he agreed that the term epileptiform syndrome was as suitable as any for this phenomena.

As our series has enlarged, we have been more and more impressed with the predominance of impaired consciousness in the patients' symptomatology. We have been unable to produce consistently the alpha variants by stimulation through scalp electrodes in the human. We have, therefore, embarked on an investigative problem using monkeys (Macacca Mulatta), endeavoring to determine the site in the brain or brain stem which might produce this episodic impairment of consciousness and concurrent alpha variant activity at the cortex of the brain. Dr. R. S. Knighton has very kindly collaborated in this aspect of our research. It was a happy coincidence that while working at the Montreal Neurological Institute, he had noted alpha variant activity occurring at the cortex on stimulating the hypothalamus of cats (Fig. 4). It was not possible to measure critically changes in consciousness in the cat at a time when the animal was under nembutal sedation.



STIMULATION OF HYPOTHALAMUS IN REGION OF LATERAL NUCLEUS AND MEDIAL FOREBRAIN BUNDLE. SECTION OF THALAMUS AT FRONTAL PLANE 11.

H.L. lateral nucleus of hypothalamus
MFB medial forebrain bundle
H.P. posterior nucleus of hypothalamus

Fig. 4

In the past year a primate laboratory has been established at the Henry Ford Hospital, and we are gradually solving the technical problems in the production of an efficient intra-cerebral electrode. At present we are using a glass-covered, enameled, stainless steel wire No. 50, the enamel being scraped off for about 2 mms. from the tip of the electrode.

The matter of evaluating minimal changes in consciousness presented a problem, and we have to thank Dr. Harry Harlow of the University of Wisconsin who kindly arranged the training of one of our psychologists in psychometric testing of monkeys.

The monkey is tested for some months until a plateau of performance is established. Then, the animal is tested before and after the stimulation by the implanted electrode and comparison of his performance prior to and immediately following stimulation used as a measure of impairment of consciousness.

The accurate placement of electrodes intra-cranially is technically difficult, but Dr. Knighton, using a Horsley-Clarke sterotaxic unit, is most proficient.

We obtain confirmation of the actual location of the electrode by means of Xray and finally by coagulation at the point of the electrode, sacrificing the animal and perfusing with ferro cyanide solution to mark where the point of the needle has deposited iron on coagulation.

The animal on which we are presently working appears to have the electrode placed in the mid-brain. On stimulation, which requires a very small potential of approximately 2/10 of a volt, the head and eyes deviate to one side. The pupils contract or dilate (depending upon the polarity), and, finally, the animal emits a short cry on termination of the stimulus.

The EEG from this area shows the expected 5 to 6 per second high voltage activity. We have not been able to discern any change in this activity beyond 20 seconds after stimulus with the above voltage for approximately 2 seconds.

We believe there is a slight impairment of consciousness following the stimulus that lasts for approximately 2-3 seconds. We have yet to obtain a record from scalp electrodes in the occipital and temporal regions during the period of stimulus, as we have not produced other than an efficient monopolar electrode. We feel that at least a bi-polar electrode for proper stimulation in this area of the brain to drive the appropriate areas of cortex will be necessary.

We have endeavoured to set out the nature of our research in this problem, and feel that it should throw some light on these borderline epileptic states which we have called epileptiform syndromes. We have in mind to explore the results of stimulation of the various reticular formations within the brain, hoping that this will help us to understand better the role this formation plays in the state of consciousness. We believe epilepsy is not a well defined state, and this syndrome enlarges our conception of its limits.

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