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EEG Studies in Episodic Unconsciousness, Seizure Disorder and Syncope

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A total of 524 subjects from amongst those who reported to Institute of Aviation Medicine, Bangalore, with a history suggestive of suspected or clinical epilepsy or syncope were assessed, for the purpose of finding out the efficacy of the different provocative techniques employed in the electroencephalographic evaluation and to have an objective insight into the relative merits of clinical and EEG diagnosis. Electroencephalograms of all the subjects were obtained and a thorough clinical evaluation was carried out. The study interalia revealed—(a) No EEG abnormality was found in subjects with a history of simple, uncomplicated episodic unconsciousness (syncope), (b) of the interictal EEGs of clinical epileptics, only 18.7% were abnormal, 8.8% borderline and 72.5% were regarded as normal, (c) the EEGs of suspected epileptics revealed that 18.8% of these were abnormal, 13.3% borderline, and 67.9% normal. (d) Provocative techniques like hyperventilation and photic stimulation were found to be very useful in eliciting information that are diagnostically significant, (e) Clinical diagnosis was found to be superior to EEG diagnosis.

Introduction

Now a days Electroencephalographic examination has become a part of the routine investigation procedure of all cases of seizure disorder or episodic unconsciousness. Institute of Aviation Medicine has been conducting EEG studies for more than two decades and lot of valuable information has been collected by now. We report our experience of 524 subjects studied by us. The study while bringing out the value of resting EEG, highlights the efficacy of routine provocative techniques like hyperventilation and photic stimulation.

Material and Method

524 subjects from amongst those personnel reporting to the Institute of Aviation Medicine, with the history of seizure disorder or episodic unconsciousness were selected for the study. These subjects were referred from medical Evaluation Centre of the

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Institute and Command Hospital Air Force Bangalore. The seizure cases were divided into two groups—Suspected and Clinical epileptics. The term "Suspected" was used to include those subjects where no definite history of fits was available and Clinical epileptics comprised of those who had positive history of fits with a clear cut description.

EEGs were recorded on a 16 channel EEG machine (Grass Model 6), subject lying in a recumbent position on a comfortable couch in the EEG lab. The International 10-20 system was followed in the placement of surface contact electrodes on the scalp. Both bipolar and monopolar montages were used for the EEG recording. Antero-posterior and transverse runs were taken in the bipolar montage. All provocative techniques photic stimulation and hyperventilation were used in the bipolar runs Resting record for all the montages were taken with visual stimulation. The subject was asked to hyperventilate for a period of 3 minutes and simultaneous EEG recording was done and the EEG recording continued for another 3 minutes after stoppage of hyperventilation. Intermittent photic stimulation was carried out with varying flash flicker frequency (5-26HZ) by the photic stimulator (Grass Model PS-2) and the duration of each exposure to flash flicker was 5-6 seconds with their eyes open and closed during the exposure.

These EEG records were subjected to visual analysis by more than one electroencephalographer as per standard technique. The results were classified into Normal, Borderline and Abnormal.

The following criterion of evaluation was followed :—

Normal (N)

Dominant alpha rhythm posteriorly with no excess of slow or fast activity, no asymmetry, no local or paroxysmal abnormality.

Border Line (BL)

Dominant alpha with slight excess of Theta and minor asymmetry. No clear local or paroxysmal abnormality.

Abnormality (ABN)

Excess or dominant Theta and delta rhythm, distinct asymmetry, local or paroxysmal abnormalit (spikes and/or sharp waves).

Results

Results are given in Tables 1—6 (attached). Th legends used are N=Normal EEG, BL=Borderlin EEG and ABN for abnormal EEG.

Discussion

The EEG is a valuable tool in the diagnosis convulsive disorder. Berger[1] (1932), Gibbs, Dav and Lennox[2] (1935) were the pioneers to describ abnormalities in the electroencephalograms epileptics between seizures and a statistical surv showed that 40% of subjects have abnormal EE in the interictal phase. The specific changes lik spikes and sharp waves do not pose much di problem but the nonspecific changes some tim constitute a great diagnostic and categorisatio problem. However, it is known that 10 to 15% apparently healthy individuals have abnormal EE and 10 to 20% of the epileptics have normal EEG This complicates the issue and the confusion more confounded if the history is vague. In o study, 81.4% of total subjects had a vague histor whom we classified as "Suspected". The EE results in patients who had fainting or syncope generally normal. The exception are the patient with structural and metabolic abnormalities.

In the study of 524 subjects there were 8 syncope and remainder consisted of 165 suspect and 353 clinical epileptics (table 4). Out of uncomplicated episodic unconsciousness subject (syncope) 4 (66.7%) had normal EEG while (33.3%) had borderline EEG. (Table 4). Our find ings are in general, consistent with those of oth workers like Hans Strauss[3] et al (1952) who al reported normal EEG records in patients of syncop 97 subjects (18.5%) showed abnormal EEG. (Tab 4) and all of these are interictal EEGs. The result could be affected by many variables such as duratio of recording, number of serial recordings, provoca tive methods used and physiological status dur recording. The duration of record should be lon enough to engulf intermittent electrical discharg and sometimes high scalp resistance attenuates t records. In such cases Electrocorticograms could b very useful but there are practical limitations for routine use.

In suspected epileptics 67.9% had normal, 13.3% had borderline and 18.8% had abnormal EEGs (table 4). Soloman[12] (1943) analysed 313 cases of suspected epilepsy and found 49% normal, 12% border line and 39% abnormal EEGs. The difference in our findings are only in percentage of abnormal records. This could be due to different methodology, other variables mentioned earlier and secondly the criteria of labelling these cases as abnormal are not known.

On analysis of the clinical epileptics it was found 72.5% had normal, 8.8% borderline and 18.7% had abnormal EEGs (table 4). The majority of them were dependants of serving personnel. Mani[10] (1973) found 28.9% abnormality in 1039 resting records of 621 patients and 31.5% abnormalities in 1447 records of all types included. His percentage is more because of longer and serial EEG recordings and his source of material is from a more pathological group than ours. The percentage of normal records in our series compares well with that of Gibbs and Gibbs (1952)[6] as they had 64% normal EEG's out of 1260 patients.

The main difficulty encountered was in classifying these subjects into two categories—suspected and clinical epileptics as this was mainly based on the history. The problem was due to lack of proper and reliable history and description of the fits given by a layman may lead to an erroneous diagnosis. Improper documentation also posed a great hurdle to us. Out of the 97 abnormal cases 31.9% had a vague history. It is quite possible that many of these patients may have been in the clinical category and this would have, increased the percentage of abnormals in the clinical category.

The family history is of paramount importance. Out of the total 524 subjects, only 7.8% had a positive family history and out of the 97 abnormals, only 2.1% had a positive family history. It is obvious that some subjects with positive family history would have shown abnormal records if serial EEGs were taken. It is well documented that with possible exception of spike and sharp wave pattern and its fast and slow variant, there is no other EEG change which is pathognomonic of epilepsy. Any electrical dysrhythmia may underline epilepsy, psychoneurosis, psychosis, psychopathy and other clinical condition. Thus it follows that an abnormal EEG can be interpreted only in relation to the clinical history of the patient. Gibbs et al (1935)[6], Jasper and Kershman (1941)[9], Finley, et al (1941[3] William (1941[14] Brazier, 1945[2], Soul IJ (1949)[13] Gastaut (1960)[4] and Richter (1971)[11]. The absence of history leads to a problem in diagnosis and this was also a limitation in our study. We agree with the opinion of most of the workers that the EEG is not infallible and it should be considered as a supplement and by no means a substitute for clinical judgement.

In our study, percentage of abnormal EEG is more in aged and children upto 15 years old. This could be due to possible effect of intracranial pathology current or past (table 1). As per diagnosis it was found that generalised discharges are most frequent and 3.1% were focal (table 6). Psychomotor seizures were nil. There were 8.3% petitmal cases in the present study out of which 3 were pure and 5 were mixed with grandmal epilepsy. This is quite comparable to a statistical survey of 2000 patients (Harrison 1983)[5] in which 51% had generalised, 8% had petitmal and other minor seizure 1% psychomotor and remaining 40% mixed type.

The study aimed to examine the efficacy of routine provocative techniques. Our findings of statistically high significant difference ($P < 0.001$) under hyperventilation and photic stimulation are consistent with other workers in this field. Further it was found that most frequent abnormality is non-specific and generalised in nature 75.2% (table 6). The occurrence of slow waves is the commonest abnormality. The morphology of abnormal patterns in our study (table 5) compares well with those of Gibbs and Gibbs (1952).

Conclusion

The simple uncomplicated episodic unconsciousness (syncope) subjects did not reveal any EEG abnormality. The majority of the conditions which produce transient loss of consciousness were found in a relatively young population in our study. EEG abnormality were found to be 18.7% in clinical epileptics, and 18.8% in suspected epileptics which has been at variance with other workers. This could have been due to various factors the nature of which at present is only speculative. It is possible

Table-I

Distribution as per Age (n=524)

Age group years	No.	N-EEG No.	N-EEG %	BL-EEG No.	BL-EEG %	AB-EEG No.	AB-EEG %
5. &<	22	15	68.2	—	—	7	31.8
6-10	13	10	77.0	—	—	3	23.0
11-15	28	17	60.7	3	10.7	8	28.6
16-20	156	107	68.6	18	11.5	31	19.9
21-25	136	98	72.0	15	11.0	23	17.0
26-30	85	64	75.3	9	10.6	12	14.1
31-35	47	38	80.9	5	10.6	4	8.5
36-40	18	10	55.5	5	27.8	3	16.7
40 & >	19	13	68.4	—	—	6	31.6
Total	525	372	17.0	55	10.5	97	18.5

Table-II

Distribution as per Sex (n=524)

Sex group	No	N-EEG No	N-EEG %	BL-EEG No	BL-EEG %	ABN-EEG No	ABN-EEG %
Male	465	333	71.6	53	11.4	79	17.0
Female	59	39	66.1	2	3.4	18	30.5
Total	524	375	71.0	55	10.5	97	18.5

Table III (A)

Distribution of subjects with and without provocation

Test group	No.	N-EEG		BL-EEG		ABN-EEG	
		No.	%	No.	%	No	%
Resting	524	440	84.0	70	13.3	14	2.7
Under Hyperventilation	524	365	69.7	52	9.9	107	20.4

$X^2 = 49.8$ $P < 0.001$ (Highly significant)

Table III (B)

Test group	No	N-EEG		BL-EEG		ABN-EEG	
		No	%	No	%	No	%
Resting	524	440	84.0	70	13.3	14	2.7
Under Photic stimulation	524	446	85.1	31	5.9	47	9.0

$X^2 = 32.95$ $P < 0.001$ (Highly significant)

TABLE IV

Distribution of subjects as per history

History group	No	N-EEG		BL-EEG		ABN-EEG	
		No	%	No	%	No	%
Syncope	6	4	66.7	2	33.3	—	—
Suspected Epilepsy	165	112	67.9	22	13.3	31	18.8
Clinical Epilepsy	353	256	72.5	31	8.8	66	18.7
Total	524	372	71.0	55	10.5	97	18.5

Table-V

% Distribution of morphology of 97 abnormal EEGs

Morphology	BETA	THETA	DELTA	SHARP WAVES	SPIKE & DOME WAVE	SPIKE
No.	10	73	39	15	12	1
%	6.2	45.3	24.2	9.3	7.5	7.

Table-VI

% Distribution of type of electrical discharge in abnormal EEGs in relation to history

	No.	Generalised	Lateralised	Focal
Suspected Epilepsy	31	61.3	32.3	6.4
Clinical Epilepsy	66	81.8	16.7	1.5
	97	75.2	21.7	3.1

that these factors were beyond the control of electroencephalographic technique employed in this study. The significance of a proper history with description of the fit, serial interictal EEGs and provocative techniques for correct diagnoses and disposal need special emphasis. Clinical diagnosis seems to have clear cut edge over EEG diagnosis, However a combination of the two will prove to be of greater help in the proper diagnosis, management and disposal, especially of air crew.

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