AN EEG AND CLINICAL STUDY OF MURDERERS

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"AND CAIN said to Abel his brother: 'Let us go forth abroad.' And when they were in the field, Cain rose up against his brother, Abel, and slew him.

"And the Lord said to Cain: 'where is thy brother Abel?' And he answered: 'I know not. Am I my brother's keeper?'

"And He said to him: 'What hast thou done?' The voice of thy brother's blood crieth to me from the earth.

" 'Now, therefore, cursed shalt thou be upon the earth."

(The Book of Genesis, iv. 8-11)
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INTRODUCTION

Ever since Cain slew Abel mankind has debated the subject of murder. The act of murder occupies a unique place in the feelings of man, which is understandable because of its link with death which causes primitive anxiety and therefore provokes an intense social reaction. Nevertheless for a long time society has accepted the distinction between the 'sane' and 'insane' murderers. The sane murderer is thought of as acting upon rational motives that can be understood and the insane one as being irrational senseless and motiveless. In practice, the distinction is not so simple. At one end lies the psychotic murderer who is McNaughton-mad and can be objectively measured on this yardstick, while at the other end is the murderer who kills for obvious reasons of profit. It is the middle ground where boundaries become blurred.

Scottish Law for a long time has recognised three categories of murderers. Those who are completely responsible and those who are completely irresponsible and a third category comprising those who, while they may not merit the description of being insane, are none the less in such a condition as to reduce the quality of their act from murder to culpable homicide.

The incidence of murder in this country is remarkably consistant. The Home Office Research Unit Report on Murder (Statistics based on England and Wales) clearly establishes an unchanging pattern/
/pattern with minor fluctuations over the years. About 150 homicides are known to the police in a year. A third of the murderers commit suicide before charged and another third show sufficient mental abnormality for the law to concede that they are either insane or of diminished responsibility, whereas the remaining third commit murder because of "wickedness".

The present study includes murderers whose unlawful act was the product of mental disease or mental defect. The courts as a result of finding no criminal responsibility for their unlawful acts because of mental illness regarded these persons as mental patients and not as prisoners and therefore they were committed to the State Mental Hospital instead of prisons.

Since Berger's pioneering work in the electroencephalography innumerable articles have been written on the subject of cerebral electrical activity in mental illness. Unfortunately there are few investigations done on individuals who commit murder while mentally ill. The last study in this area appeared in 1955. During the past few years there has been considerable refinement of EEG recording techniques. It is the purpose of the present investigation to study the patterns of electrical activity of psychiatric patients who commit murder, utilising modern apparatus for recording.

In the previous reports on this subject assessment of EEGs is primarily qualitative designating the records as 'normal' or 'abnormal'. It is intended that the present investigation will take into account both qualitative and quantitative aspects of the EEGs/
EEGs. The quantitative assessment will be done by manual and automatic methods of analysis in control and experimental populations.

Finally, an attempt will be made to relate the psychiatric and other clinical attributes of the patients to specific types of electrical patterns.

Perhaps it would be appropriate to point out at the outset that the control population in this study consists of 'normal' individuals matched for age. Since at this stage the objective is to find out if there is a higher incidence of abnormal EEGs among the experimental group. Once this point has been elucidated the next step in the systematic study would be a comparison between a population of matched psychiatrically ill patients who have not committed murder and a population of murderers who are not psychiatrically ill in the eyes of the law.

The bibliography includes a number of publications which have not been referred to in the text of this dissertation. These publications are not essentially pertinent to the subject of electrophysiology of homicide. However, the author has attempted to compile a comprehensive list of literature broadly related to the subject of psychiatric aspects of murder.
REVIEW OF THE LITERATURE

So far as is known there are four studies which deal exclusively with the EEG investigation of murderers. In several other publications murderers are included as a sub-group of a population consisting of psychopaths and criminal offenders. The association between murder, criminality and epilepsy has been a subject of numerous communications, some of which date back to the middle of last century. The valuable contribution of electroencephalography in the understanding of epilepsy not only renewed the interest in this area, but emerged as a useful objective technique to establish responsibility in criminal cases. During the past decade the 14 and 6 positive spike phenomenon and its association with murder attracted the interest of various workers, initiating one of the most controversial subjects in electroencephalography.

In the following pages the literature in the above mentioned areas will be surveyed according to the following scheme:

(A) The survey of studies devoted to the electroencephalog- graphic investigation of the murderers.

(B) Epilepsy and criminality.

(C) 14 and 6 positive spike phenomenon.

(D) Electroencephalographic studies of psychopaths and criminals.
Perhaps the first significant indicator of forensic possibilities of the EEG in murder cases came from Hill and Sargent\textsuperscript{45} in 1943. They reported a case of matricide, in which a boy of twenty who had a tendency to hypoglycaemia had a quarrel with his mother. Following this he was pushed out of his mother's room, which involved some struggle. Subsequently he went to the kitchen where he saw a kitchen knife with which he stabbed his mother. He stated "something came over me so that I was a homicidal maniac". His mother died of multiple wounds. EEG examinations of the accused revealed that whenever his blood sugar was below about 100 mg/c.cm., his tracings were abnormal and showed marked instability of hyperventilation response, which was associated with some impairment of judgement and clouding of consciousness. The history indicated that his dietary intake prior to the murder had been poor and presumably his blood glucose level was below 100 mg/c.cm., hyperventilation was induced as a result of struggle and emotional stress and this combination of various factors resulted in an abnormal behaviour. EEG examination under laboratory conditions as nearly comparable to those at the time of the crime provided the supportive evidence for this assumption. It was concluded that from the legal point of view: the accused knew what he was doing at the time when he killed his mother and knew what he was doing was wrong. But at that time, his brain was functioning abnormally which impaired
his judgement and rendered him incapable of appreciating the nature of the act. The prisoner was pronounced guilty but insane.

Session Hodge cited a case of an eighteen year old girl who was accused of "grievously bodily wounding" a woman without apparent motive. It happened at about 2 a.m. when the accused got up from her bed, descended to the floor below and inflicted severe head injuries with a horse-shoe on a woman asleep there. Subsequently she had a complete amnesia about the events of the crime and remained emotionally unperturbed. Her resting record showed a peak at 7 c.p.s., the fasting record with blood sugar of 84 mg/c.cm. showed increased energy in the 7 c.p.s. frequency band, with bursts of lower frequencies. Hyperventilation resulted in a regular bilateral delta. Ingestion of glucose did not produce any change. Imitation of conditions preceding the crime under laboratory conditions led to the assumption that the accused probably had a low blood sugar level at the time she committed the crime. It was concluded that an EEG which was abnormal at 84 mg/c.cm. would be at least as abnormal, if not more so, at lower levels of blood sugar. This inference was accepted and the accused was bound over, with a condition to undergo psychiatric treatment.

Stafford-Clark and Taylor examined sixty-four prisoners awaiting trial on charge of murder. The group included fifty-eight men and six women, whose age ranged from 14 to 60. After trial, three were found not guilty and in fifteen cases the charge was reduced to manslaughter. Fourteen of the sixty-four
prisoners were executed and fifteen were found guilty but insane after a statutory enquiry. The investigators obtained full personal and family histories together with the details of the charge and trial proceedings and examination of mental state. On the basis of clinical evaluation the prisoners were placed in one of five groups:

1. Eleven cases in which killing had been incidental to the commission of another crime or in self defence.
2. Sixteen cases having clear motive for killing or in which death resulted as a consequence of violence used during the commission of another crime.
3. Fifteen cases in which there was apparently little or no motive.
4. Eight cases in which some kind of sexual activity occurred at the time of the murder.
5. Fourteen persons who were found unfit to plead, or guilty but insane, at their trials or later at a statutory enquiry.

Most of the EEGs were recorded on a standard three channel apparatus, using the bipolar technique and electrodes placed in conventional patterns. All subjects were examined after twelve hours of fasting and each record lasted for at least thirty minutes. Three minutes of hyperventilation was the only provocative technique used and if this procedure resulted in a significant change, a serum glucose estimation was performed and hyperventilation repeated thirty minutes after 100 g. of glucose had
been given by mouth.

Records were divided into four categories as follows:

(1) Normal:— Records characterised by the presence of symmetrical, reactive alpha activity (8-13 c.p.s.) as a dominant rhythm in the post-central regions persisting from 5-100 per cent of the time in the entire record. The presence of symmetrical, low amplitude 14-30 c.p.s. activity in the pre-central regions and a small amount of 4-7 c.p.s. activity in the central and temporal areas, unless it had a paroxysmal occurrence or its voltage exceeded that of the alpha rhythm, was regarded within normal limits.

(2) Mild unspecific:— Records showing 4-7 c.p.s. activity but in excess of that seen in the normal group; or tracings with theta as the dominant rhythm in frontal, central or temporal regions. High amplitude slow activity appearing as a result of hyperventilation was also included in this group.

(3) Specific abnormalities:— Only definite focal or epileptic disturbances were included in this group.

Stafford-Clark and Taylor found an incidence of 10% and 25% EEG abnormality in prisoners included in Group I and Group II respectively. Only one subject in Group I had an abnormal record; four of the Group II subjects had abnormal records. Two of these were specific in character, one patient having a history
of head injury and the other of traumatic epilepsy. The other two had mildly abnormal unspecific EEGs and it is worth noting that each had a very poor motive for murder. The authors pointed out the parallel between the 10% incidence of EEG abnormality among the accidental murder group to that of normal population and the 25% abnormal EEGs of the motivated group corresponding to Gibb's findings in sane murderers and Silverman's study of a normal prison population (31% and 26.7% respectively).

The highest incidence of abnormality (86%) was demonstrated in subjects included in Group V. Of the cases comprising the insane group, five were epileptic, three schizophrenics, three depressives, two psychopaths and one "paranoid". A high proportion of abnormal records in this group is of course due to preponderance of an epileptic population in the group. However, of only two of the fourteen subjects who had normal records, one was a schizophrenic and the other was suffering from a depressive illness. The incidence of abnormality in the group of sex murderers was 73% but the sample was too small for a meaningful statistical analysis.

The most important observation in this study was the greater than 70% incidence of abnormal EEGs in Group III. This finding led the authors to the conclusion that a significant correlation between motiveless murders and EEG abnormality needs further investigation. They commented that the unsatisfactory nature of psychiatric criteria commonly admissible in criminal justice to assess an
individual's culpability is well known. With the EEG there is a possibility that an objective indication of innate propensities affecting responsibility may be obtained. It is, moreover, an indication likely to be of maximal value in those cases where clinical observations and the operation of reason are least able to elucidate the individual's actions. It seems highly unlikely that the correlation between an apparently motiveless murder and an abnormal EEG is purely coincidence.

Hill and Pond extended the study and reported on the 105 capital cases which included the 64 subjects reviewed by Stafford-Clark previously. The age range was 12 to 54 years but the majority of prisoners were men in the 20-30 age range (55 cases). The prisoners were classified into five groups according to the crimes, and EEG records were evaluated on the same criteria as Stafford-Clark and Taylor. They found 50 subjects with abnormal records. The incidence of abnormality decreased with the age of prisoners, until the age of 40 when there was again a slight rise, probably because of the onset of cerebral degenerative disorders. Without giving details, they claimed that their findings underlined those of Stafford-Clark and Taylor. 18 subjects included in the study were undoubtedly epileptics. In the non-epileptic population, 13 had a severe EEG abnormality (five with localized and eight with diffuse abnormality) and their histories were loaded with morbidity of all types. 27 patients revealed only mild non-specific "constitutional" abnormality in contrast to the thirteen subjects with severe abnormality who were considered to have acquired brain damage at some period.
of their lives.

Mundy-Castle reported on a mixed racial group of twenty-two subjects which included 15 Africans and 7 Europeans, involved in twenty murders and two attempted murders. Four subjects were found to be mentally disordered or defective before arraignment and twelve on arraignment. Three were found guilty but insane and one committed his crime when in a mental hospital. These subjects committed murders with little or no motive. Of the remaining two subjects, one attempted murder under extreme provocation and one killed in self defence.

EEGs were recorded on a six channel standard apparatus in conjunction with B.N.I. analyser. Electrodes were placed in conventional patterns and the bipolar technique of recording was used. In addition to standard hyperventilation, intermittent photic stimulation was affected by a Scophony stroboscope. The EEG records were evaluated on the same criteria as Stafford-Clark and Taylor and classified as normal or abnormal, the latter being graded in three categories: A1, mild non-specific abnormalities; A2, severe non-specific abnormalities; A3, focal and epileptic abnormalities. The EEG components were classified as delta rhythm (.5 - 3.5 c.p.s.); theta (4 - 7 c.p.s.); alpha (8 - 13 c.p.s.) of occipital or parieto-occipital origin; alphoid (8 - 13 c.p.s.) of frontal or fronto-temporal origin; beta rhythm (14 - 30 c.p.s.) and slow alpha variants (2nd subharmonic of the alpha rhythm of the same origin).
The authors' finding of an incidence of 73% of EEG abnormality supported the results of two previous studies and confirmed the high incidence of EEG abnormality among psychotics or motiveless murderers. Of the sixteen abnormal records, three were in A1 category (Mild unspecific), one in A2 category (Severe unspecific) and twelve in A3 category (Epileptic or focal). Six subjects (27%) were confirmed epileptics. 27% of the records showed unilateral temporal lobe foci, which points in particular to the importance of the temporal lobes in criminal behaviour. Hyperventilation did not result in a significant change except in one subject whose resting record was normal but hyperventilation tended to change it towards abnormal. Photic stimulation accentuated existing abnormalities in 3 out of 20 cases. In two subjects abnormalities appeared which were not seen in the resting record.

Apart from slow alpha variant abnormalities, three cases were striking for harmonic relationships often present in the analysis of their EEG abnormalities. A particularly high incidence of alphoid rhythm in frontal or fronto-temporal regions was noted. Analysis revealed that 14 cases showed this type of activity, five of these sometimes in anterior or generalized paroxysmal burst form. These alphoid rhythms were spatially and temporally independent of alpha rhythms. This activity has previously been reported as common among normal Africans, but its incidence in the group of Africans included in this study was greater than normal. The significance of this activity is not certain but Mundy-Castle is of the opinion that
"there are indications of its relationship both to emotional instability and to racial or cultural differences".

The author emphasized that an abnormal EEG provides strong presumptive evidence of cerebral dysfunction; therefore, he stated that on the basis of EEG alone it would be quite sufficient evidence for diminished culpability, particularly if unusual stresses prevailed at the time of the crime. The validity of this view, the author points out, is substantiated by the conclusions of courts concerning the final disposal of the subjects included in this series.

Winkler and Kove reported 55 cases charged with homicidal actions (murder or manslaughter) admitted to the psychiatric prison for pre-sentence observation. This study lacks in the description of the criteria of EEG abnormality. The object of the study was described as understanding the psychological forces producing abnormal behaviour and their correlation with abnormal EEG. They found 13 patients (24%) showed abnormal records. The EEGs and psychodynamic factors were different in each case. However, it was possible to delineate them into the following broad categories:

1. EPILEPTICS: This group included two cases in which convulsive disorder was obvious. One of the patients cited was an epileptic of eight years' standing. He killed two women after approaching them sexually and being frustrated.
in his advances. He claimed complete amnesia for the events and apparently he was under the influence of alcohol when he committed the crime. He had a normal resting EEG but hyperventilation showed bursts of 3-4 c.p.s. delta waves in the temporal lobes. The dynamic factors considered to be contributory to the crime were his ambivalence, mother dependency and compulsion to assault women when his needs were frustrated.

The second case in this category is that of a 33 year old patient, who had a history of convulsions as a child and of fainting spells and seeing "dead people" in adolescence. His EEG was characterised by high voltage delta waves and a diffuse slowing in the anterior leads after hyperventilation. However it is doubtful whether his crime was related to epilepsy.

2. NON-CONVULSIVE PAROXYSMAL DISORDERS: Patients included in this group gave no history of seizures but EEG revealed paroxysmal disorders. Three patients demonstrated this phenomenon. One of them was a boy of 17 charged with matricide. He denied a history of grand mal seizures, amnesia or disorders of consciousness. During the EEG examination he disclosed that he saw coloured lights when he shut his eyes which was accompanied with spike discharges on the EEG. On further questioning, the phenomenon of deja vu was also elicited. His EEG was characterised by bursts of 3-4 c.p.s. high voltage, delta activity more marked anteriorly, and spike discharges in the right temporal
The second case is that of a girl aged 20, charged with infanticide. She became confused after suffering from "cramps" on the day of the delivery of her baby. She was hospitalized for "cramps" and subsequently her baby was found dead in the bathroom wrapped in the patient's pyjamas. She disclosed temporal lobe symptoms and a history of fainting spells as a child. Her EEG showed spike and sharp waves in the right posterior leads, which became accentuated on hyperventilation.

Another case is that of a 20 year old woman who was in the eighth month of her pregnancy. She became angry with her four year old son and hit his head against the wall. She could remember shaking him violently but had amnesia for subsequent events. This boy was conceived as a result of rape. Her EEG showed isolated spike activity on both sides and she admitted having black-outs during her last pregnancy.

These cases illustrate the pattern of reacting to frustration by losing control and the act appearing psychologically motivated. The patho-physiological basis of abnormal behaviour was reflected in their abnormal EEGs.

3. BRAIN INJURY: Three patients included in this group had histories characterized by long standing maladjustment and anti-social acts prior to committing homicide. They
had a definite history of head injury at some stage in their lives. This lowered their threshold of tolerance to frustration. Alcohol or minor provocations resulted in a drastic act like homicide. Their EEGs showed abnormalities as would be expected in post traumatic cases. The authors pointed out that in cases where head injury is responsible for behaviour disorder it is not essential that it is always accompanied by neurological signs or a clinical picture of chronic brain syndrome. The cerebral injury may not cause intellectual impairment or disturbances in cognitive functions, but still it may be sufficient to result in gross maladjustment and defective impulse control.

4. HEREDITY DYSRHYTHMIA: There was only one patient who showed fourteen per second positive spikes and a mild asymmetry. He committed multiple murders for which he was sentenced to 18 years imprisonment but was released from prison because of his good conduct. Immediately after his release he killed two men.

5. TOXIC, ATROPHIC AND DEGENERATIVE PROCESSES were considered to be responsible for brain dysfunction in three cases. One of the patients had pemphigus vulgaris of three years' duration. She became depressed while on steroid therapy and committed the crime. Another patient had Little's
disease. She had an abnormal EEG and the psychological motivation for murder was indicative of a hysterical reaction in response to a repressed wish to kill, which erupted and was acted out when she suffered from a black-out. Another two cases in this group were in their late seventies with symptoms of arteriosclerotic dementia, who killed without apparent motive.

The authors believe that an organic brain lesion of known or unknown origin is the agent which predisposes a person to defective impulse control and to react with aggressive behaviour to environmental pressure. There is no direct relationship between an abnormal EEG and behaviour disorder, but both phenomena are manifestations of the same underlying process - brain dysfunction. There are a variety of causes which may give rise to cerebral dysrhythmia; therefore to find out the agent responsible in each case, it needs individual evaluation. They emphasised that subjects with minor brain damage are as much prone to behaviour disorders as patients with gross brain damage. They cited Chapman, who used psychological tests in subjects with minor brain damage without clinical evidence of chronic brain syndrome. He found these patients had a significantly lowered frustration tolerance. In clinical practice a common example is that of convulsive disorders in which the most frequent ictal effect is anxiety, which in turn precipitates aggressive behaviour as a defence mechanism.
The authors are against using criteria like motive of the crime and amnesia regarding the events of the crime. They state that the crime may appear premeditated and purposeful but it is still possible that it might be the result of faulty resolution of inner conflicts and defective planning because of diseased cerebral function. The psychological and organic factors are equally important and are interdependent; therefore each case requires individual assessment. The general attitude of the legal profession, divorcing these two factors from each other, also needs re-orientation.
(B) **EPILEPSY AND CRIMINALITY**

The number of epileptics included in the preceding series reviewed is strikingly high. In Stafford-Clark & Taylor's\(^{113}\) fourteen insane murderers, five were definitely epileptic. Mundy-Castle\(^{86}\) reported that six out of twenty-two cases in his series were confirmed epileptics. Among the thirteen patients with abnormal EEG's in Winkler & Kove's\(^{125}\) series, two were considered to be epileptic and another three showed paroxysmal abnormalities unaccompanied by convulsive disorders. Hill & Pond\(^{44}\) in their 105 cases of murder found in 27 subjects that there was a possibility of epilepsy, which they were able to divide in three broad categories:

a) Nine subjects showed specific EEG abnormality out of which five were chronic epileptics but another three were not known to have epilepsy.

b) Nine subjects gave a definite history of epilepsy, six of them had abnormal EEG's although not of specific epileptic type.

c) Nine subjects gave a history raising the possibility of epilepsy in retrospect but it was not proven. Three subjects in this group had abnormal records.

Out of these 27 subjects, the authors considered at least 18 were undoubtedly epileptics. The type of EEG abnormality demonstrated by half the epileptic group was of generalised type (a typical spike and wave) and of the remainder, five showed temporal foci and one subject revealed non-temporal focal dysfunction.
In the murderers included in Hill & Pond's series, the incidence of epilepsy is 32 times that of general population in which the incidence is 0.5%. This is no doubt suggestive of a relationship between murder and epilepsy but it was stressed by the authors that this does not necessarily mean that murders are committed during epileptic seizures or post-epileptic automatisms. They discussed the conditions under which dangerous behaviour can occur in relation to the seizure. The purposive, co-ordinated and sustained behaviour necessary for spontaneous assault is out of the question during the cortical discharge of major or minor epileptic seizure. In true petit mal recovery is immediate and without automatism, but in the recovery process following a grand mal seizure, there is a variable period of confusion accompanied by a considerable disorganisation of electrical activity of brain and it is during this phase that there is the possibility of dangerous aggressive behaviour. Certainly some murders have been committed in this state but none of their own series was in this category. Psychomotor seizure is yet another type of attack to which antisocial behaviour is often ascribed because of partial preservation of consciousness. Hill was able to take EEGs during such attacks which showed slow activity from one side of head, but the accompanying behaviour consisted of semi-purposive movements which lacked enough co-ordination to pose an aggressive threat. Therefore, if one adheres to Hughlings Jackson's definition of the epileptic phenomenon as a sudden excessive discharge of the grey matter, there are insufficient grounds to assume that aggressive acts
of epileptics are narrowly related to their actual fits, never the less, it would appear that some relationship exists.

Rey, Pond and Evans\(^9\) showed that epileptic and non-epileptic temporal lobe foci are often associated with behaviour disorders. This is understandable because fronto-temporal areas represent a higher level of autonomic function and it is possible that temporal lobe foci cause autonomic disturbances by discharging at a higher level. However, all episodic phenomena cannot be explained on the basis of recurrent temporal discharges or as post-ictal states. Recurrent catatonic episodes followed by seizure, showing cyclic EEG changes, is one example. This makes one think that the epileptic fit is not a disease but a train of physiological events which can be set in motion in "normal" brains given sufficient provocation. When epilepsy is associated with brain damage obviously the behaviour disorder in such cases is not a manifestation of epilepsy but both are dependant on the common factor of brain damage.

Podolsky\(^1\) describes the features which characterise the murders committed by epileptics. There is lack of motive, behaviour is extremely violent, the acts are repetitive and subsequent amnesia may be partial or complete but rarely amnesia does not occur. There is usually no attempt to escape or if an attempt is made it is confused. A case of a 12 year old boy cited by Davidson\(^1\) exemplifies these features. The boy had a left temporal lobe discharging focus; he killed a 4 year old girl while playing with her. He had some memory of the event and stated "The next thing I know I was choking her. I seemed to be under a spell - just run like a piece of machinery - I
couldn't help it when I was doing it. I shook my head and I was O.K. again. Neilson reported a case of a 21 year old epileptic who committed a relatively motiveless murder; it was unprovoked, his behaviour was extremely aggressive and continued long after the victim was dead. He made a poor attempt to escape.

Urechia reported several cases in which several homicidal and suicidal tendencies developed during the attacks of "diencephalic epilepsy". The first case was that of a man of 41. Three years after a febrile illness he started having attacks of impulsive behaviour which he had great difficulty in controlling. His family became afraid of him and demanded his hospitalisation. These attacks appeared unexpectedly lasting for 15-20 minutes. During the attacks his face was congested and he had tachycardia; he became agitated; he appeared furious and aggressive. He also had an irresistible desire to kill someone - a desire he would try hard to master. This was a terrifying experience for the patient because he thought he might lose control over himself and actually kill someone. These attacks would terminate suddenly and there would be no subsequent amnesia. During the interval between the seizures he was perfectly normal.

The second case was that of a man of 34 who had had encephalitis at the age of 28 and since the age of 31 he developed parkinsonian symptoms. He had diencephalic attacks which were at times accompanied by oculogyric crises. The affective components of his attacks consisted of sadness, feelings of fatigue and he seemed apathetic. In addition to tachycardia and profuse sweating, he showed a tendency towards impulsive behaviour and a desire to commit suicide which
he found difficult to control. The whole attack lasted for 10-15 minutes and he finally did kill himself in one of these attacks. The author described three other cases where attacks were accompanied with feelings of anxiety or depression. All these cases of diencephalic crisis had some common factors; the attacks appeared suddenly involving the affectivity and autonomic system. In the interval between the attacks the patients were completely normal. These features would qualify this condition to be called epilepsy but such a phenomenon fails to explain another essential component of the clinical picture - the parkinsonian symptoms. Therefore, the author concluded that the epilepsy and the parkinsonism are dual affections caused by a single agent.

Walker described a patient with a temporal lobe lesion who killed his wife. A 48 year old man who was well adjusted till the age of 22 when he became depressed and restless. He was diagnosed as thyrotoxic and a thyroidectomy was performed. Following the operation he remained well for six or seven years, but then he became nervous and complained about attacks of pain round the heart. During these attacks it was noted that his head would turn to the right and he would sweat. These episodes became more frequent. At this stage the diagnosis of idiopathic epilepsy and of infiltrating tumour of the left temporal lobe was made. The patient refused operation. Because of his aggressive and hostile behaviour he was admitted to a mental hospital but after a few months was discharged. He started having frequent "spells" occurring three or four times a day. During one of these
spells he killed his wife. When the police officer called, the patient
admitted him to the house and pointed to an adjacent room where the
partly clothed body of his wife lay with thirty stab wounds in her
chest. The patient showed no emotional response and at times sat
staring into space, muttering that he had been framed. He had complete
amnesia and next day when newspaper accounts of the murder were shown
to him he could not believe it had happened. A court psychiatrist
verified that the patient killed his wife in post-ictal automatism.
Because of the nature of his attacks and left temporal spikes, left
temporal lobectomy was performed, following which his attacks became
less frequent and he was less aggressive. During the next eight years
he gradually deteriorated and he demonstrated some paranoid features.
He died of complications following a fracture and his autopsy revealed
right temporal angioma. Perhaps it is not unjustified to assume that
his epilepsy was associated with this tumour but except for the right
sided temporal slow waves on the EEG, the clinical picture never
suggested such a lesion.

Lennox \(^6\) emphasises in his monograph the paucity of criminal
action among the thousands of epileptic patients and states "The
muddy medical literature of the last century, based on bromide
saturated and mentally defective patients has discoloured the concepts
of doctors even to the present day". In his thirty-five years of
experience he came across only two cases of murder by epileptics and
in both instances the relationship with epilepsy was doubtful.
The first case was that of a 22 year old veteran who, while visiting an aunt, beat her to death, took her money, placed her body in the back of a car and drove from a suburb of Boston to Maine where he was arrested. This young man had had convulsive seizures at least twice in his life, once at the age of 2 and again when 11. Once while on Military Service he found himself in a different State without knowing how he got there. The patient had a complete amnesia regarding the events of the murder; his EEG revealed a normal resting record but hyperventilation resulted in an abnormal build-up with high voltage spiky waves which were marked on the left anterior temporal zone. Metrazol caused a similar increase in voltage but did not induce seizure discharges or a seizure. A jury found the patient guilty of murder and he was sentenced to death, but the sentence was commuted to life imprisonment. The second case is that of a 52 year old man and his wife who while driving back from a cocktail party, had a minor quarrel. She struck her husband on the head with a handbag. That was his last memory - his next memory was picking up his wife's badly mutilated body which he carried to his car and drove away. After driving about for the rest of the night, he put the body on the side of the road and covered it with her coat. Following this, he drove home, changed his clothes, went to his office, wrote some letters and drove back to the place where he had deposited the body, meanwhile informing the police to meet him there. The defence pleaded that amnesia was of epileptic origin but on continued questioning the prisoner recalled details of his actions after the murder which at the beginning of the enquiry, he claimed he could not remember. Several EEGs under varying conditions revealed no abnormality.
A neurologist testified that although he had episodes in the past which appeared to be of an epileptic nature, one could not be sure that the patient had been in an epileptic attack at the time of the murder.

Lennox also cited four cases in which epilepsy was blamed or might have been blamed. One of the patients who had occasional seizures planned and carried out a brutal murder of a girl schoolmate. The defence raised the possibility of epilepsy but failed to find a doctor who would testify for him. Another case is one of twins one of whom was a confirmed epileptic. He became involved with a group of homosexuals and shot their leader dead. He was fully conscious at that time. The defence did not bring the question of epilepsy into the proceedings. The third case is that of a teenage boy who stabbed a schoolmate 42 times. His memory of murder was complete. He had a normal EEG and his personal and family history gave no suggestion of epilepsy. The defence heavily leaned on the diagnosis of schizophrenia and epilepsy. A death sentence was passed, but was later commuted to life imprisonment. This last case was also included in Stearns' series of motiveless murders by adolescents. He attempted to discover common elements in order to establish a clinical syndrome. He found all were adolescent males with uniformly good records. Their victims were all females, two of them scantily clad and the crimes had been motiveless, wanton and ferocious.
Walker laid down criteria to be considered when attributing a crime of violence to an epileptic state:

i) That the patient was subject to genuine epileptic attacks. The assumption can be proved by accurate description from the subject and witnesses. Ideally a physician should be able to observe a spontaneous attack himself but at times it might be necessary to induce an attack with a minimal dose of Metrazol.

ii) That the spontaneous attacks of the individuals are similar to the one which allegedly occurred at the time of the crime.

iii) That the amnesia claimed by the subject is compatible with the type of epileptic attack. Obviously there is no disturbance of consciousness with a Jacksonian fit during local jerking and one may not lose consciousness until the entire body is affected by the seizure. During the petit mal attacks patients do not lose contact. The difficulty arises in cases of temporal lobe seizures when consciousness can be affected to various degrees.

iv) That the degree of confusion and loss of consciousness claimed by the subject at the time of committing the crime is compatible with the confusion and unconsciousness of previous attacks.
v) That the EEG signs are compatible with the clinical disorder. Thus if the individual is considered to have temporal lobe epilepsy he should reveal a discharging focus on the EEG. One should not trust a single EEG but perform the examination repeatedly. However, it should be remembered that a negative EEG does not necessarily exclude the dysfunction but repeated examinations diminish the probability of having such a disorder. An abnormal EEG is not a complete diagnosis but it should be interpreted in the context of the entire clinical picture.

vi) The motive of the crime; the degree of violence and attempt to escape are other factors which should be weighed carefully.

These criteria cover a wide ground and sometimes it may not be possible to evaluate the crime on all of these criteria, but if several do not appear to be met, one should look with suspicion upon the epileptic basis of the homicide.

Although gruesome murders committed by epileptics have been reported in the literature from time to time, Alström\(^1\) failed to find evidence in his study of 897 epileptics. He compared the male adult epileptics with the records of a control series of 42,000 Swedish males. Of the latter group, 46% had been entered in the penal register compared with 7.0% of all epileptics. However for the mentally affected epileptics there was an increase from an expected percentage of about 5 to an observed percentage of 10 per cent.
The incidence of criminality in the group with symptomatic epilepsy was 9.7%. Mentally unchanged patients affected with epilepsy due to unknown cause showed an incidence of 5.1% which is not much different from the rate of criminality in general population. The number of habitual criminals among the epileptics was nearly the same as in the control population. As to the nature of the offences, of those in the penal register, in a ten year period, crimes of violence were recorded for 11% of the control group and for the 17% of patient series. However all crimes of aggression were minor and, usually, a complication of abuse of alcohol. This finding is in agreement with Berbeau who reported on admissions to a hospital for the criminally insane; 1.6% of all admissions over a period of 20 years were epileptic. He pointed out that in the majority of the cases crimes were of a minor nature and there appeared to be no particular type of crime associated with epilepsy. The group consisted of a high percentage of cases with mental symptoms or brain injury and many of the crimes were committed or attempted under the influence of alcohol.

None of the patients in Alström's study committed crimes during psychomotor seizure; there were no instances of murder or manslaughter. The author stated: "In a modern text book of neurology published in 1946 we read that impulsive equivalents in epileptics have many crimes to their account such as arson, unmotivated homicide, theft and exhibitionism". This gloomy view of the criminality of epileptics by no means conforms with the conditions that have been found to obtain in his present investigations. The so-called impulsive equivalents in psychically
unchanged epileptics are greatly exaggerated in respect of their dangerousness for the general public.

One recent example of arousing the general public's feeling on the subject of murder and epilepsy was that of the trial of Jack Ruby. During the trial, news headlines and the glaring publicity on T.V. once again brought back the repressed superstitious attitude towards epilepsy. The experts testified at the trial that there was no clinical or EEG evidence suggesting that Jack Ruby was an epileptic. They contended that epileptic seizures are never associated with complicated and planned behaviour. This brought an inspired editorial in JAMA from Dr. Samuel Livingstone, his remarks are worth quoting "...... I certainly would not question the fact that an epileptic might kill, not because he has epilepsy, but because he is a human being. However, I would like to state emphatically that the violation of the sixth commandment is not endogenous to epilepsy and that an epileptic is no more a potential murderer than the so-called 'normal' individual".
FOURTEEN AND SIX PER SECOND POSITIVE SPIKES

Gibbs & Gibbs\textsuperscript{26} were the first investigators to describe one of the most controversial EEG findings designated the 14 and 6/sec positive spike phenomenon. This pattern of electrical activity can be seen during light sleep only. 14/sec positive spikes resemble typical sleep spindles except that they are distorted and the negative phase of each wave is cut off and the slope of the positive phase is more abrupt than normal. 6/sec spikes are morphologically similar to 14/sec spikes. Both types are usually associated but occasionally one abnormality may exist independent of the other. This phenomenon occurs diffusely on both sides at one time or another, but is most marked on the occipital and temporal areas. The Gibbs's found this pattern in 2\% of a group of 300 normal control subjects and 6\% of a group of 5,000 epileptics or suspected epileptics. The highest incidence was found among adolescents or young adults. 14/sec spikes were common below 20 years of age and 6/sec spikes were common in the above 40 age group. Most of the patients demonstrating one or both of these patterns had otherwise normal waking records.

This report by Gibbs & Gibbs had extensive acclamation but the earlier experiments by Grossman\textsuperscript{36 & 37} appear to have been overlooked by other workers. He verified his earlier finding of depressed evoked activity in sleep on the side of the lesion and noted a characteristic distortion of both spontaneous and evoked activity from posterior head regions in patients with episodic aggressive behaviour. The distortion occurred as a marked reduction of the negative half of the fast 14/sec activity and exaggeration of the positive phase.
There are various types of symptoms which have been reported to be associated with positive spikes, namely autonomic dysfunction, convulsive phenomena and behaviour disorder. Since the first two categories are not relevant to the subject of this dissertation, these will be touched on only briefly.

The incidence of autonomic symptoms reported in patients with these positive spikes varies from 64.7% (Kellaway56) to 19% (Poser96). Gibbs and Gibbs26 & 27 stated that in patients with positive spikes autonomic symptoms occurred in attacks (for example, flushing, sweating, shivering, palpitations, gagging etc.) and some symptoms tended to recur in each attack. Some investigators describe headaches as the commonest symptom in these patients. The next most common symptoms were abdominal pain, vertigo and syncope. In the majority of cases more than one of these autonomic symptoms might occur at the same time. Walter et al120 disclaimed any significant association of positive spikes with autonomic symptoms. The predominance of autonomic symptoms was one of the reasons which lead the workers in this field to believe that the focus of positive spikes must be situated in the thalamus or hypothalamus.

The incidence of convulsions reported in the patients with 14 and 6/sec. positive spikes varies from 20.7% (Walter et al120) to 67% (Gibbs & Gibbs26). Niedermeyer & Knott89 pointed out that a majority of patients with positive spike phenomenon who had convulsions had additional EEG abnormality. Gibbs & Gibbs26 claimed that 87% of their patients with positive spikes had ictal types of symptoms including fainting attacks, attacks of loss of consciousness, crying spells etc. which they preferred to call "epileptoid" and later
designated as "the borderland of epilepsy". The episodic nature of the symptoms was another reason which made many authors speculate that the positive spikes were related to epileptic phenomena which originated from a deep lying epileptogenic focus.

Gibbs & Gibbs in their original study reported an association between behaviour disorder and the positive spike phenomenon. Four of the subjects in the study were murderers. Presumably because of the repeated failure of electroencephalographic approaches which yielded little useful information in psychiatric disorders, the discovery of positive spikes came as a gift from heaven and it gave a new lease of life to the entire field. Disillusioned investigators of human behaviour chasing the wild goose of EEG correlates were suddenly thrilled to find the association between 14 and 6/sec. positive spikes and behaviour disorders. The overwhelming enthusiasm of various workers to establish this relationship is reflected in the number of publications.

The incidence of behaviour sufficiently disturbed to present as a major complaint in patients with positive spikes, varies from 12% (Metcalf) to 19.8% (Millen and White). Over 60% of patients with positive spikes showed disturbance to a lesser degree, presenting not as a major symptom but merely as histories suggestive of behaviour disorder.

Walter et al. denied any significant relationship between positive spikes and behaviour disorder. Their excellent study was carefully controlled and investigators were unaware of the diagnosis when recording their observations. They stated that when one is
dealing with such poorly defined terms as "epileptic equivalents" or "autonomic epilepsy", it is difficult to get two electroencephalographers to agree on the clinical application of these terms. It is only by use of control groups and examiners who are unaware of the EEG findings that valid comparisons can be made. Indeed this statement very neatly sums up a major snag inherent in most studies in this area.

Behaviour disorders associated with the positive spike phenomenon have been claimed to be commonly characterised by disturbed aggressive outbursts. At least 17 cases of murder are cited in the literature and another wide variety of violent acts has also been attributed to the 14 and 6/sec positive phenomenon. Schwade & Geiger\(^99\) reported a case of matricide committed by a thirteen year old boy who was considered well adjusted in all respects. The presence of 14 and 6/sec positive spike discharges in his sleep EEG was regarded as indicative of thalamic or hypothalamic disorder. This report was soon followed by the publication of another case by Schwade & Otto\(^102\). A boy of high school age killed a younger playmate by stabbing him a number of times. His EEG showed the 14 and 6/sec positive spike phenomenon and homicide was considered to be a manifestation of thalamic or hypothalamic dysfunction.

Schwade & Geiger\(^101\) reported 623 patients with severe behaviour disorders, of which 453 showed 14 and 6/sec positive spikes. The group included five bizarre compulsive murders (two of these were reported above). The authors again emphasised the role of thalamic and hypothalamic dysfunction resulting in impulses which are released by trigger-like stimuli and acted out without cortical restraint.
Winfield & Ozturk\textsuperscript{124} presented a case report of a thirteen year old boy who shot and killed his mother. The authors agreed with other workers and interpreted 14 and 6/sec spikes in the patient's sleep EEG as thalamic or hypothalamic disorder. In this case other activation techniques including Metrazol, Pentothal and photic stimulation, yielded no useful information. Stehle\textsuperscript{116} studied thirty patients (7 to 18 years of age) with aggressive behaviour. Their EEGs demonstrated 14 and 6/sec positive spikes. Aggressive behaviour in two of the patients resulted in the death of a family member; two produced serious physical injury of others; several caused extreme property destruction by fire setting and others attempted serious homicidal acts.

Mills\textsuperscript{81} confirmed 14 and 6/sec positive discharges in cases of behaviour disorder, including a teenage murder. He stressed the role of organic factors in these cases and stated that to ignore the organic factors by considering only the psychodynamics is to create a "sin of omission" in diagnosis, treatment and prognosis. Woods\textsuperscript{129} reported two cases of adolescent homicide of family members. Both patients had 14 and 6 positive spikes in their sleep records.

A recent study by Friedlander\textsuperscript{20} was based on the sleep EEGs of 20 late teenage prisoners. None of the prisoners exhibited 14 and 6/sec spike discharges in their EEGs. They noted a large number of prisoners were on anticonvulsant medication and suggested this variable might well have been responsible for their essentially negative results. This certainly raises an interesting point which has not been clarified in the literature.

The relationship between 14 and 6/sec positive spikes and psychopathology has been repeatedly emphasised by authors\textsuperscript{68,101 & 120}. 
One really wonders how justified this marriage is between psychoanalysis and electroencephalography. Perhaps it is the distressingly wide gap between neuro-physiology and psychodynamics which forces some workers into wishful thinking of having discovered that missing link.

Unfortunately the controversy is not entirely confined to the clinical correlates of positive spikes, there are many workers who completely deny the existence of such a phenomenon. The British experts are among the foremost who regard 14 and 6/sec. discharges as nothing more than a figment of the imagination of the American workers. The proponents of the 14 and 6/sec. phenomenon counter attacked and questioned the techniques of investigators who were unable to recognise the 14 and 6/sec. pattern. They stressed that there were three crucial requirements for eliciting this EEG sign. First, spike discharges appear only during light sleep and in laboratories where sleep recordings are not taken routinely the question of the positive spikes does not arise. Secondly, electroencephalographers relying almost exclusively on the bipolar techniques do not detect positive spikes because sleep patterns are jumbled in bipolar recordings. Nevertheless, Garneski et al.21 demonstrated that 14 and 6/sec. spikes are observable in bipolar recordings but when registered this way the pattern appears to be upside down or may be cancelled, depending on where the electrodes are placed. The third objection is to the placement of electrodes conforming to the "Ten-Twenty International System", because it does not focus sharply on the primary areas where monopolar recordings show distinctive patterns of 14 and 6/sec. positive spikes.
Those clearly laid down requirements for the registration of 14 and 6/sec spikes are rarely met in British laboratories. Nonetheless, British experts have repeatedly refuted the existence of 14 and 6/sec positive spike phenomenon. Perhaps this extremely controversial EEG sign deserves further systematic investigation on the same lines as suggested by the American investigators, in an effort to clarify this progressively confusing issue.
Since Jasper, Solomon and Bradley\textsuperscript{54} reported a high incidence of abnormal EEGs among the behaviour problem children, a large number of articles on the subject of behaviour disorders in adults has been published. Keeping in the tradition of the main stream of psychiatry, the data are empirical and naturally a variety of opinion has been expressed. The majority of the investigators agree that among persons exhibiting behaviour disorders, a greater proportion of abnormal EEGs is found than among normals but beyond this point, opinions are in conflict. Some of the inconsistencies in the findings of various workers are explicable on the basis of variability of criteria adopted for EEG abnormality. Other sources of discrepancies among the reports are the difference in the clinical populations studied and variability of diagnostic criteria from one centre to another.

In the following pages the existing data will be surveyed in relation to the following specific questions:-

(i) Is there a high percentage of abnormal EEGs among psychopathic personalities and criminals?

(ii) What are the criteria of EEG abnormality accepted by the various investigators?

(iii) What are the electroencephalographic characteristics of psychopathic personalities and criminals?

(iv) Is there any specific type of abnormal behaviour which is associated with abnormal EEGs and are there other clinical correlates of EEGs?

(v) Are there any factors contributory towards EEG abnormality?
(vi) An abnormal EEG is usually indicative of brain dysfunction. In cases of psychopathic personalities and criminals with abnormal EEGs, is brain dysfunction manifested by abnormal neurological signs?

(vii) Can EEG findings be translated into the etiology of psychopathy and criminal behaviour?

Since much of this material is controversial, the reviewer has attempted to present the opinions expressed by various authors either by direct quotation or by summarization without too much criticism. The incidence of EEG abnormality and the type of subjects included in the various studies is listed in chronological order in Table I.

(i) General Findings Derived from Statistical Surveys of Groups of Criminals and Psychopaths

The earliest data relating EEG abnormalities to the category of psychopathic personalities were contributed by Hill & Watterson in 1942. The group consisted of 151 cases diagnosed as psychopathic personalities; EEGs were abnormal in 48 of the cases. Gibbs et al in the same year reported their findings based on EEG studies of 339 inmates of a state prison. A sample of 100 men selected at random from this prison population revealed an incidence of 34% EEG abnormality compared with a 15% rate of EEG abnormality in a control series of 1000 adults. Harty et al obtained EEGs from 274 candidates for military service and found that 30% had abnormal records. 8 subjects included in the sample were considered psychopathic personalities, with the incidence of abnormal EEGs among these subjects being 88%.
<table>
<thead>
<tr>
<th>Author</th>
<th>Date</th>
<th>Type of subjects included in the sample</th>
<th>No. of Cases</th>
<th>% Abnormal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hill &amp; Watterson</td>
<td>1942</td>
<td>Psychopaths</td>
<td>151</td>
<td>48%</td>
</tr>
<tr>
<td>Gibbs, Bloomberg &amp; Bagchi</td>
<td>1942</td>
<td>Criminals</td>
<td>339</td>
<td>34%</td>
</tr>
<tr>
<td>Silverman</td>
<td>1943</td>
<td>Criminal Psychopaths</td>
<td>75</td>
<td>80%</td>
</tr>
<tr>
<td>Knott &amp; Gottlieb</td>
<td>1943</td>
<td>Psychopaths</td>
<td>44</td>
<td>52%</td>
</tr>
<tr>
<td>Greenblatt</td>
<td>1944</td>
<td>Psychopaths and cases of behaviour disorders</td>
<td>295</td>
<td>31%</td>
</tr>
<tr>
<td>Silverman</td>
<td>1944</td>
<td>Prisoners diagnosed as psychopaths</td>
<td>208</td>
<td>75%</td>
</tr>
<tr>
<td>Knott &amp; Gottlieb</td>
<td>1944</td>
<td>Psychopaths</td>
<td>68</td>
<td>54%</td>
</tr>
<tr>
<td>Session Hodge</td>
<td>1945</td>
<td>Patients including children adolescents and adults referred for anti-social or criminal behaviour</td>
<td>70</td>
<td>85.5%</td>
</tr>
<tr>
<td>Silverman</td>
<td>1945</td>
<td>Homosexuals</td>
<td>55</td>
<td>75%</td>
</tr>
<tr>
<td>Gibbs, Bagchi &amp; Bloomberg</td>
<td>1946</td>
<td>Criminals</td>
<td>452</td>
<td>23%</td>
</tr>
<tr>
<td>Simon &amp; Dietholm</td>
<td>1946</td>
<td>Psychopaths</td>
<td>69</td>
<td>53%</td>
</tr>
<tr>
<td>Gottlieb &amp; Knott</td>
<td>1946</td>
<td>Psychopaths</td>
<td>100</td>
<td>58%</td>
</tr>
<tr>
<td>Simon O'Leary &amp; Ryan</td>
<td>1946</td>
<td>Psychopaths</td>
<td>96</td>
<td>15%</td>
</tr>
<tr>
<td>Stafford Clark &amp; Taylor</td>
<td>1949</td>
<td>Prisoners charged with murder</td>
<td>64</td>
<td>50%</td>
</tr>
<tr>
<td>Hill &amp; Pond</td>
<td>1952</td>
<td>Prisoners charged with murder</td>
<td>105</td>
<td>47.6%</td>
</tr>
<tr>
<td>Kennard &amp; Levy</td>
<td>1953</td>
<td>Inmates of State Penitentiary</td>
<td>100</td>
<td>30%</td>
</tr>
<tr>
<td>Mundy—Castle</td>
<td>1955</td>
<td>Cases of murder drawn from mental hospitals</td>
<td>22</td>
<td>73%</td>
</tr>
<tr>
<td>Bagchi et al</td>
<td>1959</td>
<td>Adult prisoners</td>
<td>466</td>
<td>38%</td>
</tr>
<tr>
<td>Winkler &amp; Kove</td>
<td>1962</td>
<td>Cases admitted to a psychiatric prison</td>
<td>55</td>
<td>24%</td>
</tr>
<tr>
<td>Kozaczewska &amp; Kaczanowska</td>
<td>1962</td>
<td>Psychopaths</td>
<td>71</td>
<td>22.5%</td>
</tr>
<tr>
<td>Small</td>
<td>1966</td>
<td>Criminals</td>
<td>100</td>
<td>33%</td>
</tr>
</tbody>
</table>
Obviously this is too small a number on which to base any conclusion but curiously, the incidence of abnormal EEGs in the entire group was extraordinarily high for a so-called "normal" population.

There are three contributions from Silverman \(^{104,106,109}\) which are worth mentioning: the first study was of 75 criminal psychopaths and the incidence of abnormal EEGs was 80%. The second report was based on the EEG findings in 411 prisoners which included 208 psychopathic personalities. The rate of EEG abnormality among the psychopathic group was 75%. The third study by Silverman & Rosanoff \(^{109}\) was a report on 55 homosexuals with an incidence of 75% abnormal EEGs. It is indeed very surprising that the authors should have found an enormously high incidence of abnormal EEGs among the homosexuals.

In the series reported by Knott & Gottlieb \(^{64,65}\) the first communication was based on the examination of a group of 44 psychopathic personalities. The total incidence of EEG abnormality in the group was 52%. In the second report the authors extended the number of patients and were able to confirm their earlier findings. In a still later study, Gottlieb & Knott \(^{33}\) examined 200 subjects with behaviour disorders and classified them into two groups of 100 each: patients of 16 years or over were considered as psychopathic personalities and disorders in patients of 15 years or under as primary behaviour disorders. The incidence of abnormal EEGs among the former was 58% and in the latter 56%.
Hodge examined 70 cases including children, adolescents and adults referred for antisocial and criminal behaviour. The overall extent of EEG abnormality which he reported was 85.8%. Simon & Diethelm noted that 69 psychopaths included in their series had an incidence of 53% abnormal EEGs.

The findings of Kenard & Levy, Bagchi et al and Stafford-Clark et al are also suggestive of a higher incidence of abnormal EEGs among prison populations. On the contrary Revitch, in a brief communication, reported on 16 aggressive female inmates of a State Reformatory all of whom were late adolescents or young adults. In spite of the small number of subjects included in the study, this author denied the existence of a higher incidence of EEG abnormality among aggressive criminals.

From the foregoing account it would appear established that a significantly high proportion of patients with psychopathic personalities and criminals have abnormal EEGs - the range of incidence of abnormality varies but the majority of studies demonstrate that nearly one half of the subjects included in the samples have abnormal EEGs. However, the following three studies are not in agreement with the others.

Simon, O'Leary and Ryan examined 96 patients diagnosed as "constitutional psychopathic states" in a military setting and found that 15% had abnormal EEGs, an incidence which is not significantly different from the normal controls. Gibbs, Bagchi and Bloomberg
enlarged their earlier study\textsuperscript{24} and based their second report on 452 prisoners including 148 cases of robbery, 79 cases of murder, 114 sex offenders, 33 cases of assault and battery and 77 subjects imprisoned for various repeated offences. The rate of abnormality was 23\% which they concluded was not significantly different from normal controls. This contradiction of their earlier findings, they believed, was due to the age factor being omitted in their previous study.

Kozaczewska and Kaczanowska\textsuperscript{67} recently studied 71 psychopaths. The diagnosis was based on Schneider's\textsuperscript{103} classification. The EEG records were normal in 55 cases, slightly altered in eleven and distinctly abnormal in only four cases. The authors concluded that when patients with organic brain lesions are carefully excluded from the sample, the incidence of abnormal EEGs is not significantly different in psychopathic personalities and normal populations.

(ii) \textbf{CRITERIA OF EEG ABNORMALITY}

One of the major sources of discrepancy among the reports of various investigations is the lack of universally acceptable criteria of EEG abnormality. Gross abnormalities like focal disorders, paroxysmal phenomena and delta activity (slower than 4 c.p.s.) are accepted by all investigators as abnormal. It is with respect to moderately slow activity (5 - 8 c.p.s.) and low voltage fast activity and other slight to moderate deviations from normal that dispute arises. Another source of confusion is the hyperventilation response.
Some authors give no consideration to slight to moderate changes occurring after overbreathing, while others disregard it unless the resting record also shows abnormal findings and designate these phenomena as borderline, questionable abnormality, or abnormal.

Hill & Watterson considered resting records as abnormal if the dominant rhythm was less than 8 c.p.s., if the record showed bursts of two or more waves having a frequency of less than 7 c.p.s., or if isolated random waves at 6 c.p.s. or less with an amplitude equal to or more than that of dominant rhythm were present provided these were repeated from the same cortical area. Wave and spike complexes and series of waves with a frequency of 14 or more rising in voltage to over half that of the dominant rhythm were other features regarded as pathological. After 3 minutes hyperventilation if sub-clinical discharges appeared or high amplitude slow waves persisted continuously for more than 20 seconds after discontinuing hyperventilation or recurrence of 2-3 c.p.s. waves 8-15 seconds after cessation of hyperventilation when the record had started to return to the normal, it was rated as an abnormal record.

Silverman classified his records into three categories. He considered those records as abnormal which showed presence of definite rhythm of a frequency below 7.5 c.p.s. or occurrence of definite high voltage spike activity in roughly more than 10% of record. Borderline records were characterised by arhythmia, random slow and spike waves might appear in these records but did not form a definite rhythm. Records presenting abnormalities only on hyperventilation were classed as borderline.
Knott & Gottlieb evaluated the records upon a three point scale - normal, questionable or abnormal. A questionable EEG was considered to be one in which there were repetitive slow waves, 8 per second or slower appearing infrequently in short sequences and of low voltage. These might appear in the absence of any alpha or might be superimposed or interposed between alpha activity. Abnormal records were characterised by presence of frequent bursts of rhythmic activity slower than 8 c.p.s. with an amplitude greater than the average voltage of the records.

Greenblatt arbitrarily regarded "abnormality" as activity with a predominant frequency outside the range of 8-12 c.p.s. or tendency to change with overbreathing and applied this criterion to all groups of his patients irrespective of age and diagnosis. Records outside this range were arbitrarily considered abnormal and classified as slow, fast, and mixed slow and fast on the basis of predominant rhythm.

Gibbs, Gibbs & Lennox classification which is based primarily on frequency and wave form has been used by several investigators (Gottlieb & Knott, Gibbs & Bagchi, Simon et al) EEGs containing a moderate amount of 5-8 c.p.s. waves are classified as slightly slow (S₁), those containing a moderate amount of fast waves as slightly fast (F₁) and those with much fast activity as fast (F₂). Paroxysmal disorders are considered individually, they are placed at fast or the slow extreme depending upon the frequency of waves that form paroxysm.
Focal abnormalities are not included in the scheme but considered under a separate heading. There is some disagreement among the authorities utilising this scheme for the analysis of their records. Simon & Diethelm\textsuperscript{111} regarded low voltage, fast records as abnormal. Simon et al\textsuperscript{110} considered $S_1$ and $F_1$ records in the series as normal.

(iii) ELECTROENCEPHALOGRAPHIC CHARACTERISTICS

Hill & Watterson\textsuperscript{46} noted that the most characteristic abnormality seen in the records was the presence of 4-6 c.p.s. rhythm with an amplitude slightly less than the dominant rhythm and the commonest abnormal hyperventilation response was the appearance of 2-3 c.p.s. waves recurring 8-15 seconds after cessation of hyperventilation. Eleven out of 51 records showed delta foci. Silverman\textsuperscript{106} & 109 reported diffuse abnormalities; a tendency to change after hyperventilation; the presence of 6 c.p.s. activity in anterior regions and posterior spikes in the records classed as borderline. Half of his abnormal records showed a definite 6 c.p.s. rhythm originating diffusely from frontal and prefrontal areas. Posterior spikes, spike and wave complexes and focal abnormalities were also seen in some of his abnormal records.

Greenblatt\textsuperscript{35} found a high incidence of abnormal slow activity in cases of psychopathic personality and behaviour disorders. Knott & Gottlieb\textsuperscript{65} using Gibbs' scale found 75\% of abnormal EEGs showed moderate amounts of activity slower than 8.5 c.p.s. in any lead ($S_1$ - Gibb’s terminology). In the series reported by Hodge\textsuperscript{49} nearly
half the abnormal records were characterised by the presence of persistent slower rhythms; and in twenty out of seventy EEGs, hyperventilation resulted in the appearance of delta activity although the resting records revealed no abnormal features.

Ostow and Ostow\textsuperscript{90} reported a high incidence of bilaterally synchronous paroxysmal slow activity in the EEGs of a prison population. They define the phenomena as "sudden bursts of regular activity at frequencies of 2-7 per second, the amplitude of which is at least as great as the amplitude of the alpha activity and usually greater, which forms patterns clearly distinct from the immediately preceding record, and which appears synchronously from homologous areas of the right and left sides of the head". This activity was most frequently seen in the frontal areas and was relatively infrequent in occipital leads. The authors cited the incidence of bilateral slow activity in the normal population and epileptics as 5\% and 39\% respectively, and if criminality was associated with epilepsy the incidence was 86\% Nearly one third of the criminal psychopaths exhibited paroxysmal phenomena in their tracings.

Simons & Diethelm\textsuperscript{111} were able to classify their abnormal records into two types namely, those of average amplitude, 5 to 7/ second activity, and those of low voltage slower than 5 to 7/ second activity. The majority of the abnormal records showed the presence of 5 to 7/ second activity of low average amplitude in the frontal and parietal leads.
Although Simon et al.\textsuperscript{110} could not find a significant correlation between EEG abnormality and psychopathy, they did obtain a definitely higher percentage of records falling in $F_2$ and $S_2$, categories on Gibbs' scale as compared to the normal control.

Hill\textsuperscript{42} has classified the EEG patterns found in adult neuro-psychiatric patients demonstrating episodic psychopathic or psychotic behaviour. The various groups studied were psychopaths, schizophrenics and a prison population serving sentences for various offences, persons charged with murder and patients with undiagnosed episodic behaviour disorders. This study failed to reveal any EEG characteristics which could be related specifically with any class of patient included in the study. However, he was able to group the EEG patterns into two classes, namely maturation abnormalities and paroxysmal abnormalities. Maturation defect possesses the following features:

1. Excess of theta activity particularly in temporal and central areas with an amplitude equal to or greater than that of the alpha rhythm.

2. Alpha variant, first described by Goodwin\textsuperscript{32} is defined by Hill as "alpha in 8-13 c.p.s. frequency band associated with a multiple or submultiple of its frequency". The latter is named slow alpha variant and is more commonly associated with behaviour disorders and is seen symmetrically in both occipital regions.
3. Unilateral or bilateral posterior temporal slow wave foci usually within the frequency band of 3-5 c.p.s. and having an amplitude greater than alpha. This phenomenon, unlike alpha variant, is accentuated on hyperventilation.

4. Record dominated with theta activity.

The reason Hill regards these phenomena as fundamentally representing defective maturation in the central nervous system is that such features are quite common in the EEGs of normal children, and are sensitive to the age factor. Maturation defects to some extent were seen in all the subgroups studied but by far the highest incidence was among the subjects with psychopathic personality.

Paroxysmal abnormalities are characterised by paroxysmal fast and slow activity appearing symmetrically on both sides of head, or bilateral low voltage spikes occurring in groups, or 4-6/sec. spike and wave complexes. A spike focus situated in the anterior or posterior lobe is also considered one of the paroxysmal disorders. These phenomena, contrary to maturation defects, are not noticed in records of children and bear no relationship to age. Hill regards these abnormalities as "homeostatic" because of their relationship to the recovery process rather than to the illnesses themselves. Paroxysmal abnormalities were more common in the schizophrenic group. The author states that anterior temporal lobe foci are usually associated with a history of epileptic seizures and episodes of disturbed consciousness, but posterior temporal foci are more common in patients with attacks of psychopathic aggressive anti-social behaviour and a history of nocturnal enuresis in childhood. Lastly, it should be mentioned that these two sets of EEG phenomena, maturation defect and
and paroxysmal disturbance, are far from being mutually exclusive; in fact they are often found together.

Stafford-Clark et al\textsuperscript{114} confirmed the presence of excessive theta activity in the abnormal records of psychopathic prisoners. In addition to excessive slow activity some of the records also showed an abundance of fast activity in the beta range. The records had a tendency to change significantly with overbreathing.

Of four records considered abnormal by Revitch\textsuperscript{94} in his series of sixteen patients, one showed delta bursts and 20 per second fast activity, one was S\textsubscript{1} (Gibb's scale), another showed delta activity in the posterior leads, while the fourth record had focal delta bursts in the right hemisphere. Obviously, the number of records is too small upon which to formulate an opinion as regards the type of EEG in behaviour disorders.

From the foregone account one could draw the following conclusions:-

1. Almost every type of EEG abnormality has been mentioned in cases of psychopathic personality and behaviour disorders. This is understandable because this is a heterogenous diagnostic group and no specific EEG pattern can be expected.

2. The majority of the records are characterised by the presence of diffuse slow activity. Slower rhythms are a common feature in EEGs of normal children but subsequently decline with age. The persistence of such activity in tracings of psychopaths therefore reflects a maturation defect.

3. Earlier workers claimed that the site of origin of slower rhythms
is in the anterior regions of the brain, but in more recent studies it has been pointed out that the activity is more prominent in temporal and occipital zones. This discrepancy is perhaps explicable on the basis of different recording techniques and the utilisation of two or three channel recording apparatus by early investigators.

(iv) **CLINICAL CORRELATES OF EEG's**

Hill & Watterson adopted Henderson's classification and divided the psychopaths into two groups, namely aggressive and inadequate. The incidence of abnormal EEGs was nearly twice as high among the aggressive psychopaths as the inadequate group. When epilepsy was associated with psychopathy the rate of abnormality was even greater.

They observed that the more aggressive the patient, the more likely is the EEG to be abnormal.

Among the various studies reported by Knott & Gottlieb an attempt to classify the subjects into sub-groups was made only in the first series. It included 7 patients with "pathological sexuality", 7 patients who had "asocial and amoral trends", 17 psychopaths who had "pathological emotionality", and 13 patients were unspecified. The incidence of abnormality was 72%, 71%, 41% and 46% respectively. The authors rightly pointed out that the number of cases in each sub-group was too small to permit meaningful statistical analysis.

In Silverman's study of criminals, prisoners with evidence of organic brain lesions had an incidence of 82.5% EEG abnormality;
45% of psychotic prisoners exhibited abnormal records, but prisoners with normal mental states had a rate of 26.7% abnormality. He classified the criminal psychopaths into four sub-groups (diagnostic criteria were not mentioned) and found "hostile" and "hedonistic" groups of psychopaths had an incidence of 80% EEG abnormality. "Inadequate" and "homosexual" groups revealed an incidence of 67.4% and 67.7% abnormality respectively.

Ostow and Ostow obtained EEG records from 440 patients and other inmates at the Medical Centre for Federal Prisons. 27% of 64 criminal psychopaths with no homosexual proclivities showed bisynchronous paroxysmal slow activity. Criminal psychopaths who were facultative homosexuals and psychopaths with obligatory homosexuality, exhibited paroxysmal abnormality in 28% and 39% of their records respectively. When mental defect was associated with psychopathy this phenomenon was seen in 38% of records.

Simon et al. in their series studied records with a view to determining whether or not an increased incidence of abnormality in the EEG was associated with various manifestations of extreme degrees of psychopathy and failed to find any significant correlation. Five out of twenty-five severely inadequate psychopaths had abnormal records. Twenty three patients who were considered to have severe emotional instability yielded six abnormal records including three borderline tracings. They could not find any differences in the incidence of abnormality between patients with positive or negative history of aggression, overt antisocial behaviour, drug addiction, alcoholism or homosexuality.
Simons & Diethelm attempted to correlate psychopathological features with EEG abnormality. Twelve patients with psychopathic personalities associated with psychoneurotic symptoms revealed no abnormalities in their EEGs. Similarly, seven patients who had pronounced and easily provoked mood swings showed normal EEGs. A group of 11 patients was characterised by poor ethical standards, lack of persistence of emotional relationship and lack of emotional depth. Symptoms of their maladjustment included stealing, untruthfulness, truancy and irresponsibility as regards social and financial obligations. All the patients in this group were considered aggressive and had abnormal EEGs showing a prominent, moderately slow, activity. Thirty-one patients possessed prominent traits like unsatisfactory emotional control, contradictory strivings, poor self description and a rebellious attitude towards authority and society. They were considered to be aggressive as well as passive types with loose organisation of personality and immaturity. Records of patients in this category demonstrated a mixed group of EEG patterns in which normal, moderately slow, very slow and fast types of activity were seen. Eight patients were evaluated as psychopathic personalities with a generally inadequate type of personality and vague thinking. In this group of aggressive as well as passive patients minor neurotic reactions with anxiety and resentment as a prominent emotional response occurred readily. Their level of intelligence ranged between average and superior grades but still
they had difficulty in concept formation and logical thinking. They lacked persistence in pursuing goals which were poorly defined. All patients had abnormal EEGs five of which showed low voltage activity slower than 5-7/ second.

Stafford-Clark et al\textsuperscript{114} reported an incidence of 85\% EEG abnormality among criminal psychopaths who were considered to be severely aggressive on a clinical basis, but it dropped to less than 50\% in those without overt aggressiveness. The authors were unable to find any relationship between family situation in childhood, enuresis or history of amnesia and EEG signs.

Kennard \& Levy\textsuperscript{59} could not find a difference in the rate of EEG abnormality among violent and non-violent criminals. They investigated the relationship between type of EEG record and personality structure evaluated on the basis of M.M.P.I. Prisoners who appeared more stable and had relatively fewer convictions 11\% showed high peak in the Pd scale indicating psychopathic deviation and 17\% of EEG's in this group were abnormal, while those who were repeated offenders 39\% had typical Pd profiles and 34\% of their EEGs were abnormal. The normal records in the series were characterised by an unusually high percentage of pure alpha activity, half of such alpha dominant records occurred in individuals with a high psychopathic deviation. The authors, on the basis of their findings, concluded that there are two types of criminals. One type is characterised by high Pd peak on the M.M.P.I. and a high incidence of alpha dominated normal records.
The second group contains those of normal or neurotic profiles on the M.M.P.I. and have a higher incidence of abnormal EEGs showing excess of 4-6 c.p.s. They regard the former group as having a more or less fixed personality pattern and the latter possessing underlying instability of personality which is usually associated with any one of several dynamic factors.

Cohn & Nardini report the occurrence of bilateral occipital slow activity in the EEGs of children and adults with aggressive antisocial behaviour. They described the activity as usually bilaterally synchronous and the individual wave forms having a complex contour. They cited several cases to support their hypothesis and concluded that the said pattern of activity between the ages of 17 to 25 was often associated with overt aggressive attitudes which were reflected in the case histories of patients as truancy from school, unauthorised liberty from Naval duties, need of excessive stimulation such as drugs and highway speeding, sexual hyperactivity and general rebellion against authority figures. However, a fifth of the patients included in their series who had evidence of similar aggressive attitudes did not show this type of electrical activity. In their opinion the latter group of patients had acquired faulty patterns of responses during some stages of their personality development but in the former group of patients with EEG evidence of bilateral occipital slow activity, the underlying brain dysfunction was responsible for their maladjustment. Nevertheless, some of the individuals included as normal controls also exhibited this phenomenon but on further exploration it emerged that
most of them with bi-occipital slow activity were in fact basically aggressive, but in a socially accepted way and presented high level qualities of leadership in their own environments.

Bagchi et al\textsuperscript{2} selected forty variables and correlated them against each other on an I.B.M. computer. Some of the significant correlations at the 1\% level were found between any seizure including "temporal lobe" seizure and temporal focus; early behaviour disorder and recidivism, aggressive behaviour, stealing, mental retardation; crime of extreme violence and aggressive behaviour; first degree murder, alcoholism, manslaughter, recidivism, sex crime and higher age group. Trends of association not statistically significant were found between bi-occipital focus and early behaviour disorder, manslaughter; temporal focus and murder.

Collomb et al\textsuperscript{10} noted in a group of forty three psychopathic personalities that alpha activity had a characteristic tendency to be better synchronised and of higher amplitude. The frequency of 8 per second was encountered more than were faster alpha frequencies.

Kennard, Rabinovitch & Fisher\textsuperscript{61} attempted to correlate EEG frequency pattern as measured by a frequency analyser and psychological factors. The study comprised of comparing three groups, namely patients in a mental hospital, inmates of a prison farm and normal controls. They confirmed that theta activity was present to a higher degree in subjects considered psychopathic. Analysis of psychological elements obtained from the Rorschach test showed a
positive correlation between the incidence of factors related to anxiety and fast activity in the EEG. The investigators also reported a strong correlation using the Bender Gestalt test of aggression and poor Gestalt performance with high theta activity.

In conclusion one could say that there is no evidence to suggest that a relationship exists between EEG abnormality and any particular type of psychopathy. Many workers point out correlation between aggressiveness and EEG abnormality and it appears reasonable to believe the more aggressive a patient, the more likely is the EEG to be abnormal.

(v) FACTORS CONTRIBUTORY TO ELECTROENCEPHALOGRAPHIC ABNORMALITY

Hill & Watterson observed that 10% of inadequates included in the series who gave a history of head injury had normal EEGs compared with 39% of the aggressive group with a history of head injury who had normal EEG records. The differential incidence of head injury in inadequate psychopaths and aggressive psychopaths with abnormal tracings, clearly suggests that among inadequate psychopaths past head injury is probably a contributory factor to dysrhythmia; but EEG abnormality in aggressive psychopaths is independent of previous head injury.

The authors could not find a greater incidence of EEG abnormality in either the aggressive or the inadequate psychopaths whose family history revealed presence of "epileptoid" conditions such as fits and
bad temper. Another interesting observation was a significantly high incidence of "aggressive bad temper" among the first degree relatives of aggressive psychopaths. Patients who had had fits in childhood but subsequently recovered showed a high rate of EEG abnormality. Eleven patients considered to be epileptics had $81\%$ of their records abnormal. Obviously this is too small a number on which to claim significance but the authors suggested that, when epilepsy and psychopathy are associated, there is a tendency for the EEG to be more frequently abnormal than when either condition occurs alone.

There are three studies by Gottlieb & Knott$^{33,65,66}$ devoted to the problems of exploring the correlation of EEG abnormality, antecedent illness or injury, and family history of general morbidity. They investigated their patients for family history of psychosis, personality disorder, alcoholism and epilepsy, and personal history for birth injury, febrile convulsions and head injury. A significantly greater number of abnormal EEGs were found in cases with both a positive family history and personal history of head injury or severe illness than when either of these factors was present alone.

Further investigations in a large group of children diagnosed as primary behaviour disorders and in an equally large group of young adults diagnosed as psychopathic personalities—to find which particular factors were most important—revealed that abnormal EEGs had a higher incidence when there was a positive family history
of epilepsy and maladjusted personality than in those with a negative family history and personal history. The sex of the parent involved also appeared to be related to the abnormality of patient's EEG which was greater when mothers were alcoholic or maladjusted than when fathers had such a disorder and it was still greater when both parents were affected. Of the various factors in the personal history, there seemed to be a relationship between EEG abnormality and history of convulsions, head injury, loss of consciousness or severe illnesses. The age at which illnesses occurred appeared to be relevant to the abnormality and it emerged that the younger the patient at the time of illness and the more severe the illness, the greater the probability of his having an abnormal EEG.

Stafford-Clark et al.\(^{114}\) found a greater incidence of EEG abnormality among psychopathic prisoners who had a history of head injury or those who were epileptic. Kennard & Levy\(^{60}\) in their series of one hundred psychopathic criminals, considered only those cases of head injury where there was definite loss of consciousness following the trauma; 26% of their subjects had a positive history. The percentage of EEG abnormality in these patients was 37% compared with 28% abnormality in patients with a negative history.

It has been observed by various workers that the incidence of EEG abnormality declines with age. This is true in most of the psychiatric illnesses and epileptics but such a relationship
is especially evident in the behaviour problem and psychopathic groups. Stafford-Clark et al confirmed the negative relationship between EEG abnormality and age in their study of 149 psychopathic prisoners. Knott & Gottlieb failed to find any association between age and abnormal brain activity.

Gibbs et al on the contrary reported that normal EEGs were common in the younger than in the older groups. They found an incidence of 26% EEG abnormality among criminals in 41-60 age group as compared with 18% in normal controls of the same age group. There was no appreciable difference in percentage of abnormal records in criminals and normal controls falling in age groups 21 to 30, and 31 to 40. In their preliminary report authors found a greater number of abnormal records in criminal as compared with control group which was apparently unmatched for age. This observation was not borne out when age factor was controlled and it led them to the conclusion that there was no significant difference in the incidence of abnormality in the EEGs of prisoner population and general population. Authors emphasised that studies by other workers where a high incidence of EEG abnormality was noted included a higher proportion of subjects with epilepsy and organic brain lesions. Therefore there is no justification in attributing EEG abnormality to criminality but its true correlation is with epileptic or organic factors. Hill's criticism to their findings - the correlation between EEG and aggressive psychopathy
would have been different if the 70 murderers who were included in that study had been examined at the time of their crimes because incidence of epileptic and psychopathic EEG abnormality diminishes with age.

Kennard & Levy\textsuperscript{60} noticed decline of EEG abnormality with age in group of 100 convicts. 18-20 age group had 35\% of the records as abnormal but only 26\% of those in age range of 21-55 showed abnormal records. They reported a positive correlation between abnormal EEG and the age at time of first offence. Those who had difficulties prior to the age of 15 had 42\% of their records as abnormal but the incidence of EEG abnormality among the patients who had first conviction between ages of 16 to 20 was 30\% when first offence was committed after the age of 20 the rate of EEG abnormality was 25\%.

(vi) **NEUROLOGICAL SIGNS IN PSYCHOPATHY AND CRIMINALS**

A major source of confliction in the results, is the selection of subjects included in various studies. The term psychopathy itself is notoriously difficult to define but in electrophysiological studies it becomes more confusing when some investigators scrupulously exclude subjects from their clinical material where there is the slightest hint of structural damage, whilst other authors select their experimental group regardless of the presence or absence of organic pathology.
The contamination of samples with cases of organic deficit is, to a large extent, responsible for inconsistency of results. In this connection there are four studies worth mentioning.

Neurological examination of 75 criminals included in Silverman's series\textsuperscript{106} revealed that 39\% of subjects had neurological signs and/or histories suggestive of cerebral lesion. The signs were uniformly confined to the motor and extrapyramidal system and included inequality of deep tendon reflexes and superficial reflexes, inequality of pupils, unilateral facial palsy, marked tremor, dysarthria and inco-ordination of movements. Silverman claimed the organic lesions were not responsible for behaviour disorders because the incidence of EEG abnormality was no more frequent in the patients with evidence of "organic" disease than in patients without such evidence.

76\% of psychopaths studied by Hodge\textsuperscript{49} showed a curious neurological sign. The finding, which has never since been observed by any other worker, consisted of non-phasic flickering movements of the fingers seen when the eyes are closed, and arms and hands extended. It was more prominent on the left side commonly accompanied by an equivocal plantar response on the same side and accentuated by hyperpnoea following which it may appear on both sides.

Stafford-Clark et al\textsuperscript{114} noted neurological anomalies in 25\% of non-psychopathic prisoners used as control group, 52\% in psychopaths without head injury or epilepsy, 46\% in psychopaths with head injury and 36\% in those with epilepsy. It is indeed very surprising that the highest incidence of neurological anomalies was found among the group without head injury or epilepsy.
In a recent study Small\textsuperscript{112} investigated 100 criminals for the evidence of CNS dysfunction. In addition to the neurological signs he regarded a history of severe head injury or seizures, chronic alcoholism, psychiatric symptoms suggestive of organic involvement, abnormal EEGs and psychological tests suggestive of organicity as indicative of CNS disorder. Strong evidence for the presence of CNS lesion was designated in cases with three or more of the criteria but in cases with one or two of these items the involvement was considered to be doubtful. Of the sample 33\% demonstrated strong evidence of brain damage.

(vii) **SIGNIFICANCE OF ABNORMAL EEG AND ITS RELATIONSHIP WITH ETIOLOGY OF PSYCHOPATHIC AND CRIMINAL BEHAVIOUR**

There have been many speculations as regards the significance of the abnormal EEG and its relationship to the etiology of psychopathy and criminal behaviour. Silverman\textsuperscript{104} noted the similarity between type of EEG abnormality presented by psychopaths and children with behaviour disorder. This led him to the conclusion that a psychopath possesses a brain which has been malfunctioning since early childhood. He stated that nearly all psychopaths in his study were born with or acquired in infancy a defective cerebrum particularly in the frontal areas of the brain and concluded "cerebral dysfunction increases the sensitivity to emotional trauma of childhood. Unstable reaction to
these traumas and inability to integrate new experiences into growing personality enhances the development of psychopathic modes of behaviour". Hill\textsuperscript{41} found bilateral theta rhythm in post central regions and criticised Silverman's technique, which he believed was responsible for the abnormal rhythms being seen in anterior regions. He believed that abnormal rhythms in aggressive behaviour originated from the hypothalamus. He based this view on the evidence gained from animal studies and cases of suprasellar tumours. He concluded "..... there is strong presumptive evidence that bilateral rhythmic disturbance in the EEGs of dysrhythmic aggressive behaviour is associated with dysfunction of basal grey matter, probably thalamus or hypothalamus or in circuits of neuronal chains which connect the two". He cited clinical observations relating hypothalamic dysfunction with behaviour disorder. He regarded aggressive outburst as a release phenomenon from an acute failure of cortical inhibition over hypothalamic activity which is associated with vegetative responses to emotional stimulation. Unstable hyperventilation response indicates the readiness with which cortical inhibition can become ineffective. He considered the dysrhythmia of aggressive behaviour and convulsive disorders is similar except in their site of origin and the degree of spread to other areas. Hodge\textsuperscript{49} put forward similar views emphasising that behaviour disorders are due to a failure in the establishing of normal cortico-thalamic association patterns in early childhood.
In their classic study, Hill and Watterson\(^4^6\) considered three factors which could possibly give rise to the dysrhythmia in psychopaths, namely biochemical changes in blood, cerebral trauma and constitutional defects. Hypoglycaemia, oxygen lack and other biochemical changes in the blood were excluded because dysrhythmia in those conditions is associated with impairment of consciousness. Head injury, although resulting in abnormal electrical activity, in their opinion was not responsible for EEG abnormality in psychopathy. This formulation was based on the observation that abnormal EEGs were more common in aggressive psychopaths with negative history of cerebral trauma than in those with positive history. In addition, they dismissed the factor of head injury on the basis of the type of EEG abnormality encountered among post-traumatic cases since it is different from the one noticed in aggressive psychopaths. In the inadequate group, however, the authors concluded that trauma probably accounted for some of the abnormal EEG records.

Hill and Watterson favoured the idea of "cortical immaturity" on the basis of the similarity between EEGs of aggressive psychopaths and those of young children, the authors stated: "In view of the similarity between the aggressive behaviour of psychopaths and the normal bad temper response to frustration in young children on the one hand, and the similarity between the EEGs of aggressive psychopaths and those of young children on the other, the suggestion that the abnormality in the EEGs in these cases is produced by a failure of development in the
central nervous system is very tempting. It fits in with our psychiatric, biological and social conception of psychopathic personalities. Hill & Watterson concluded: "On the evidence gathered by us from control material and patients and on that obtained by other workers, one can have little doubt that an abnormal EEG constitutes for its possessor a handicap in the business of biological adaptation, failure of which may show itself as in our present series, in undesirable asocial behaviour".

Gottlieb et al33 have similar views about normal subjects with abnormal EEGs. They stated that the abnormal EEG is merely an organism's susceptibility to difficulties in behavioural adjustment. Such persons may possess less elasticity in their neural limits for withstanding the stresses and strains of the adjustment process, their neural limits varying but being dependent on genogenic, histogenic and/or chemogenic factors.

Hill42 designated four types of phenomenon, excess theta, alpha variant, posterior temporal slow wave focus, and dominant post-central theta rhythm, as maturation defects and discussed their relationship to defects in maturation processes of the nervous system. The rationale of grouping those phenomena is that all four are sensitive to age and decline with advancing age up to 50 and all four phenomena can occur in the EEGs of normal children. Hill cited the observation of Lindsley,71,72 and Henry40 on the developmental aspects of the
EEG. The spontaneous rhythmic activity appears first in two discreet areas of the cerebral cortex namely central area and occipital area. The occipital activity finally takes the form of the alpha rhythm of the adult. Between ages 5 - 10, theta activity is the dominant rhythm in central regions and throughout childhood temporal areas show predominantly slower rhythms. In the normal process of development, the central theta gradually increases in frequency and declines in percent time. This tendency for slower rhythms to disappear with age results in adult EEGs showing only traces of central theta. Posterior temporal and inferior parietal are the areas where the slowest activity persists longest; therefore the failure of maturation in this zone accounts for posterior temporal slow wave foci associated with behaviour disorder. Hill also pointed out the facilitation of the theta rhythm in acute experiments in which young subjects have been exposed to frustration or made aggressive thereby suggesting that theta dominated records of psychopaths are related to aggressive behaviour.

There is some supportive evidence in the eight year follow up study of criminal psychopaths by Gibbons et al 22. They found in inadequate psychopaths and those over 25 years old an abnormal EEG was a favourable sign because there appeared to be better prognosis as regards subsequent criminal behaviour, indicating the possibility of change by maturation.
Cohn & Nardini\(^9\) reported the presence of occipital "complex" waves in young psychopaths and related these abnormalities to the high incidence of the initial occipital dysrhythmia in childhood including those with an overt epileptic disorder. They regarded this phenomenon as representative of an "unmatured" brain physiology in the individuals with clinical disorders of behaviour and concluded that "the aggressive behaviour, in selected patients, appears to be the conditional response of a disordered brain to the exigencies of interpersonal experience".

Knott & Gottlieb\(^{65}\) divide psychopathic personalities into two categories, namely psychopaths with normal EEGs and psychopaths with abnormal EEGs, the latter being further divisible into a type where EEG abnormality is of genetic origin and a type in which it is acquired in early life as a result of cortical damage due to illness or injury. One of their studies\(^{66}\) is completely devoted to verifying empirically the assumptions pertaining to heredity determinants of EEG abnormality in psychopathy. It was based on a group of parents of both children and adults, diagnosed either as primary behaviour disorder or psychopathic personality. A group of patients and foster parents was utilised as the control. The distribution of parents on Gibbs' scale did not show the distribution as would be expected if they were drawn from a normal control population. Knott et al classified the EEGs of true parents as follows:-
(1) those cases where both parents showed normal EEGs (2) those where one parent showed a normal EEG and the other one an abnormal EEG (3) those very few cases where both parents had abnormal EEGs. As EEG abnormalities of parents increased, there was a progressively increasing incidence of abnormal EEGs compared with normal children. They noted a statistically significant trend indicating that patients with slow EEGs had parents with slow EEGs and patients with fast EEGs had parents with fast EEGs but they did not see such a relationship in the tracings of foster-parents and foster children.
MATERIAL

A. Patients

(i) Selection

The clinical material comprises thirty-two male patients all being inmates of a State Mental Hospital. All patients had committed murder except one patient who had attempted to murder. The legal status of the patients was as follows: Three of the patients were found 'guilty but insane', one 'certified insane after trial', one 'convicted of manslaughter on the grounds of diminished responsibility' and the remaining twenty-seven were considered 'insane in bar of trial'. No attempt was made to exclude the patients with below average intelligence or those who were treated with drugs at the time of EEG examination.

(ii) Age

The age range was from 20-58. The mean age of the group was 37.7 (S.D. 3.5). The age distribution of the patients is shown in Fig. 1.

B. Controls

The control population consisted of thirty-two male subjects matched for age. Only those subjects without a previous history of psychiatric or neurological illness were included. Control material was drawn from two sources. 24 male nurses and three members of the medical staff at the State Mental Hospital, the remaining five were members of the medical staff at the Royal Infirmary.
Fig.1. AGE DISTRIBUTION OF PATIENTS
METHOD OF INVESTIGATION

1. Clinical Data

Clinical information was obtained from the case notes prepared by the medical staff of the hospital. Psychiatric reports submitted at the time of the trial, by two independent psychiatrists in each case, were also utilised in obtaining relevant clinical material. Observations and comments of the nursing staff recorded in the hospital journal were taken into consideration for evaluation of behaviour after admission.

Prior to collection of the data a check list was prepared (see appendix). The relative findings were noted as 'positive', 'negative' or 'not known'. Finally, forty-four items were selected for Kendall's tau analysis. Each item was selected on the criterion that it was known in every case and it could be clearly dichotomised or ranked. The following is the set of forty-four items selected for analysis:

(1) Age on admission. This corresponded to the age of the patient when he committed the capital offence.

Diagnosis: Patients were classified into one of the three categories.

(2) Psychopathy
(3) Schizophrenia
(4) Psychotic depression

Diagnosis in each case was based on the psychiatric reports submitted to the court at the time of conviction. In few cases where diagnosis was uncertain it was clarified with the help of the psychiatrist treating the patient since admission.

(5) The presence of neurotic traits in childhood included nail biting, thumb sucking, stammering, nocturnal enuresis and night terrors.
(6) School performance was categorised as either average or below average.

(7) Did the patient attend an approved school?

(8) Marital status: whether single or married. There was only one patient who was divorced and he was considered single.

(9) Homosexual experience, fetishism, transvestism were recorded as sexual deviations.

(10) History of alcohol intake prior to admission was rated as following:
(a) Patients who did not take alcohol or those who took only a moderate amount.
(b) Patients who consumed excessive quantities but at no stage in their lives were treated for chronic alcoholism.
(c) Patients who at some time or other were treated for chronic alcoholism.

(11) History of head injury: Minor bumps or other trivial cases of injuries were excluded. Only those patients who lost consciousness for at least one hour following trauma were considered to have a positive history.

(12) A definite history of convulsions, loss of consciousness or alteration of consciousness was classed as epilepsy. Isolated attacks of syncope or fainting were carefully excluded.

(13) Patients who had a positive Wasserman or those treated for gonorrhreal infection at any period of their lives were considered to have a positive history of venereal disease.

(14) History of previous psychiatric illness: only those illnesses for which a patient was treated in a mental hospital were taken
Family history of psychiatric illness was considered positive only in those cases where a member of the family was treated in a mental hospital for one of the following illnesses:

(15) Neurosis.
(16) Alcoholism.
(17) Psychopathy.
(18) Psychosis.
(19) Mental deficiency.

The next set of four items dealt with the relationship of the patient to the family members who suffered from a psychiatric illness.

(20) Mother.
(21) Father.
(22) Siblings.
(23) A relative other than the first degree relatives.

History of previous convictions was classified into one of the following three groups.

(24) Petty crimes: - Thefts, burglary, house breaking and frauds, i.e. crimes against property.
(25) Aggressive crimes: - Arson, assault, physical injury of the others and property destruction.
(26) Sex crimes included rape, homosexual assault and attacking females with an intent to assault.
(27) The age of the patient at the time of the first conviction for any of the above mentioned crimes.
(28) Whether the patient committed a single or multiple murder.

The type of murder was classified into one of the following categories:
When killing occurred as a result of extreme provocation and the patient's exaggerated response resulted in the death of his victim. The intention to kill or the death of the victim occurring accidentally was not taken into account but the main criteria used were a sudden provocation and the patient's inability to restrain himself.

When killing was apparently motiveless or in which the motive was very slight. Although in some cases the motive of killing was explicable on the basis of psychopathology, such factors were disregarded and the murder was classed as motiveless. Patients who killed as a result of delusions were also included in this group.

Sexual murders: Murders with strong sexual motives were included in this group. However, only those cases qualified to be included in which sexual activities of some kind occurred actually at the time of the murder.

The fourth class included those cases where an element of premeditation was present. There was a chronic conflict between the murderer and his victim and forced by the difficult circumstances killed his victim. Provocation of some kind immediately before murder was not taken into account but the main criterion used was an understandable, persistent and conscious desire of the patient to get rid of the victim by killing.

Whether the victim was a relative of the patient.

Was the victim male or female?

Age of the victim: Whether the victim was above or below the age of 16.
(36) Degree of violence: When the murderer continued to strike his victim even after death and bizarre killings were included in the category of "severe violence".

(37) If the killer was unable to recall the events which took place at the time of murder he was considered to have had amnesia.

Items (38) and (44) concern the type of EEG abnormality and severity of the dysfunction which will be dealt with in the next section.

2. Electroencephalographic Recording

The study was conducted as part of the routine electroencephalographic examinations of patients in the hospital. The patients were aware that EEG examination was voluntary and optional and knew that its purpose was to give possible help in diagnosis and treatment.

Before starting the project the recordists were given written instructions to which they could refer when required (see appendix). An Offner, eight channel type TC electroencephalograph was used. All recordings were done in a supine position with the head supported by a hard pillow. Patients lay on a couch in a semi-darkened room separated from the recording apparatus by a viewing hatch. Silver-silver chloride pad electrodes were placed according to the "Ten-twenty system" utilising the bipolar method of recording. (Fig. 2). Controls were adjusted as following:-

- Gain Control - Deflection of 1 cm. representing 100 uv.
- Time Constant - 0.3 seconds
- High Frequency Filter - Frequency response to 50 c.p.s.
- Recording Speed - 3 cm/second.
Fig 2. The placement of electrodes for EEG recording.
Each patient was recorded once during morning and had a second record after lunch on another occasion. The first record lasted 28 to 37 minutes, including three minutes over-breathing recorded in the lateral longitudinal position. The total recording time for the second record was never less than 30 minutes and overbreathing was recorded in the medial longitudinal position. The effect of eye opening and closure was noted in all records. Intermittent photic stimulation was effected by a Schophony stroboscope with eyes closed for a duration of two minutes. Flash-frequency was gradually built up to 30 flashes per second. It was returned to 10 FPS and again slowly increased to 20 FPS. Finally stimulation was affected at the same frequency as resting alpha. However, in some cases other frequencies which proved evocative were also employed. B.N.I. analysis was used when recording the hyperventilation response in the first record but was utilised throughout the second record. All subjects included in the control group except five of them were recorded at the same centre as the experimental group. Similar techniques were employed for controls and patients except that the former had only one record and intermittent photic stimulation was not utilised.

Each record, whether experimental or control, was designated by a different code number. Therefore at the time of evaluation the electroencephalographer was unaware of the name, diagnosis and clinical history of the patient. The following information was registered at the beginning of each record:

1) Code number
2) Date and time of recording
3) Time of patient's last meal
4) Handedness
5) Whether the patient was on any drugs or not
6) Condition of the patient during EEG examination

3. Evaluation of EEGs

(i) Criteria of normality

1. An EEG was classified as normal at rest during hyperventilation and intermittent photic stimulation when a dominant frequency of 8 to 13 c.p.s. was recorded in the post-central areas of the head. Such rhythm should be present symmetrically on both hemispheres in respect to frequency, amplitude and spread. It had a tendency to block on visual attention. A slight asymmetry of amplitude and forward spread on the dominant hemisphere was accepted as normal in young subjects. 14-30 c.p.s. activity with an amplitude not exceeding 20 uv. having a symmetrical distribution in the frontal and central areas was regarded as normal. A small amount of 4-7 c.p.s. activity in central and temporal regions was considered normal provided such activity did not constitute the dominant rhythm in the record and was not seen to have a paroxysmal occurrence and its amplitude did not exceed that of 8-13 c.p.s. activity. Activity less than 4 c.p.s. appearing as a discrete rhythm was regarded as abnormal. The normal EEG response to hyperventilation showed no significant change in the record. However, a slight change in amplitude or frequency, provided it occurred symmetrically, was accepted as normal.

2. EEGs with minor irregularities but within normal limits:— This category included records which showed the features of normal records but alpha activity extended symmetrically forward into anterior regions. Some of the records showed only partial blocking on
eye opening. The records which showed a slight excess of 4-7 c.p.s. activity unrelated to the age were included in this category. Occasionally resting EEGs exhibited no abnormality but hyperventilation evoked few bursts of theta activity of an amplitude higher than that of 8-13 c.p.s. activity, such records were designated as EEGs with minor irregularities.

(ii) Type of Abnormality

The records which did not meet the above mentioned criteria were considered abnormal and were classified into one of the following categories:

(1) Diffuse abnormality:— Records in which, in addition to the characteristics of normal records, other activity was present. The minimal abnormal activity appeared diffusely as random irregular slow waves. In severe cases occasional runs of 4-7 c.p.s resulted in disorganisation of the dominant frequency. The voltage of the abnormal activity was either the same or greater than that of the dominant rhythm.

(2) Paroxysmal abnormalities:— The mildest form of paroxysmal abnormality occurred as generalised short runs of slow waves in the resting record. The extreme type of such abnormalities existed as subclinical discharges including 3 per sec. wave and spike pattern and its variants, paroxysms of fast waves (12-30 c.p.s.) of increasing amplitude and widely separated, single or multiple, positive or negative spikes. In most cases paroxysmal abnormalities appeared in records previously relatively free from abnormal activity.
(3) Focal abnormalities:-- The records with slow, fast, transient spikes or sharp waves located to a focal area by method of phase reversal were included in this group. In the majority of the cases localised dysfunction existed as irregular 1-4 c.p.s. waves varying from wave to wave in form, duration and amplitude at the site of disturbance. The severity of the dysfunction was judged on the basis of the amplitude and distribution of the abnormal waves.

(4) Generalised Abnormality:-- Records dominated with rhythm less than 8 c.p.s. appearing bilaterally and spreading forward to the anterior zones. The dominant rhythm showed very little response to eye opening. The severity of the abnormality was rated on the basis of the degree of slowing.

(iii) Effect of Photic Stimulation

The following observations were made in respect of reaction to photic stimulation:

1) Response of abnormalities already existing in the resting record.

2) Occurrence of any abnormal responses: namely subclinical discharges, frontal spikes, runs of irregular slow waves, and focal abnormalities.

(iv) The Degree of Electrical Disturbance

All records were rated on a five point scale:

Rating I  - Normal type of records.

Rating II  - Records with minor irregularities but within normal limits.

Rating III  - Mild diffuse. Mild generalised or mild paroxysmal.
Rating IV - Any kind of disturbance of a moderate severity.
Rating V - Records indicating a severe dysfunction.

(v) Quantification of EEGs

Other characteristics of EEGs were studied by considering the following components:

1. Dominant frequency: which is defined here as the frequency in cycles per second of the majority of the waves present.

2. Amplitude of the dominant rhythm: The maximum voltage of the potentials was taken as the amplitude of the dominant rhythm. It was measured by a pair of calipers adjusted to the calibration for voltage at the beginning of the record.

3. Alpha factor is defined as length of the record occupied by waves of a given frequency (8-13 c.p.s. band) determined in an arbitrary unit of 100 mm. of record.

The following technique was used in the measurement of this Alpha factor: - Preliminary to beginning the analysis of the record a survey of the entire record was made and three meters of artifact-free record were selected. Each meter being taken from three different positions representing medial longitudinal, lateral longitudinal and a sample of record taken during overbreathing. The alpha waves were counted by moving a piece of transparent celluloid marked with longest and shortest alpha wave durations, along each meter of record. Any waves which fell outside the alpha range were omitted. No effort was made to count incomplete waves or waves with a voltage below 20 µv. Two posterior channels from each hemisphere were analysed. Therefore the measurements
in the first record represented the activity of occipito-temporal and posterior regions but in the second record the potentials from occipito-parietal and posterior parietal regions were taken into consideration.

Alpha factor is determined as follows:-

(i) The space occupied by a single arbitrary wave is determined by dividing 30 by the mean frequency per second of alpha activity (30 represents 30 mm of record taken at a standard paper speed of 3 cm/second).

(ii) This is multiplied by the total number of alpha waves giving the space occupied by alpha activity in one meter of the record.

(iii) Divided by 1000, it represents the quantity of the factor in 1 mm. of record.

(iv) Multiplied by 100 to obtain the quantity of alpha in a unit of 100 mm., which is the arbitrary unit utilised for standardisation.

The above mentioned four steps can be represented by the following formula:-

\[ 30 \times \text{total number of alpha waves in one meter length of record} \times 100 \]
\[ \text{Mean frequency of alpha/second} \times 1000. \]

4. A sub-alpha factor is defined as the amount of space occupied by waves of less than 8 c.p.s. determined in an arbitrary unit of 100 mm. of record. It is measured by sliding a ruler along one meter sample and adding up the length of the record occupied by waves slower than 8 c.p.s. The length of record thus determined is divided by 1000 to obtain the amount in 1 mm. This is multiplied by 100 to ascertain the quantity of sub-alpha factor in an
arbitrary unit of 100 mm. Obviously, there is a possibility of some error arising in measuring; therefore a sub-alpha factor less than 2 was not taken into account and was altogether omitted.

(5) Low voltage amorphous activity (LVA factor) is defined as the amount of space occupied by activity below 20 uv. appearing as waves which cannot be counted, determined in an arbitrary unit of 100 mm. record. Quantity of LVA is determined by adding alpha factor and sub-alpha factor and subtracting it from 100.

(vi) Classification of EEGs according to the pattern of electrical activity

Records were also evaluated from a different angle. This constituted the study of a record in detail as a complex of inseparable and variable relationships. The method of evaluation described by Davis was modified and records were classified into one of the following patterns of electrical activity. Pattern I (PI) records show predominantly a stable, regular and clearly countable alpha rhythm and is not interfered with other frequencies. The pattern of each area remains consistent throughout the record. Pattern II (P2) records have a mixture of alpha and fast frequencies in 14-20 c.p.s. range. Pattern III (P3) covers records with a mixture of alpha and slow frequencies (4-7 c.p.s.). Slow activity may be superimposed or interposed between the runs of alpha activity. EEGs without alpha activity
but presenting slower rhythms as dominant activity were also included in this pattern.

Pattern IV (P4) includes records with polyrhythmic background activity consisting of alpha, slow and fast rhythms.

Pattern V (P5) records have very little organised activity, a poorly developed alpha and the amplitude seldom exceeds 20 uv.

The examples of various patterns of electrical activity are illustrated in Fig. 3.
Electroencephalographic Observations

The incidence of abnormal EEGs in the experimental group was 65.6% (21 cases). Three records with minor irregularities were considered normal. In the control group the incidence of abnormal records was 15.6% (5 cases). The percentage of the abnormal EEGs in three diagnostic categories is shown in Table 2. The number of patients with psychotic depression is too small for any conclusions to be drawn. Therefore, it would be appropriate to combine the schizophrenics and depressives and classify the population into psychotic and non-psychotic groups. In such instance the incidence of EEG abnormality in the former group would be 77.8% and in the latter 50%.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. of patients</th>
<th>Normal EEGs</th>
<th>Abnormal EEGs</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
<td>Per Cent</td>
<td>Number</td>
</tr>
<tr>
<td>Psychopathy</td>
<td>14</td>
<td>7</td>
<td>50%</td>
</tr>
<tr>
<td>Schizophrenia</td>
<td>15</td>
<td>4</td>
<td>26.6%</td>
</tr>
<tr>
<td>Depression</td>
<td>3</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Table 2: The diagnosis and incidence of abnormality
The percentage of records classified according to the type of abnormality is shown in Table 3. The majority of the records (43%) showed diffuse abnormalities. Three patients demonstrating focal involvement, one had a right fronto-parietal focus, one showed left fronto-temporal focus and in one patient a left posterior temporal focus became marked only after hyperventilation and photic stimulation.

<table>
<thead>
<tr>
<th>Type of EEG Abnormality</th>
<th>No. of records</th>
<th>Per Cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diffuse</td>
<td>9</td>
<td>43.0</td>
</tr>
<tr>
<td>Generalised</td>
<td>5</td>
<td>23.4</td>
</tr>
<tr>
<td>Paroxysmal</td>
<td>4</td>
<td>19.3</td>
</tr>
<tr>
<td>Focal</td>
<td>3</td>
<td>14.3</td>
</tr>
</tbody>
</table>

Table 3: EEGs classified according to the type of abnormality.

The type and severity of the disturbance and its relationship to the diagnosis of patients is shown in Table 4. There was only one EEG which was severely disturbed (Grade V) showing a generalised abnormality. 8 out of 9 records considered to be diffusely abnormal demonstrated only a mild dysfunction; in the remaining record the disturbance was more pronounced.

In the psychopathic group seven patients had abnormal EEGs which included 5 EEGs showing a mild diffuse abnormality, one had a mild generalised abnormality and one demonstrated a paroxysmal abnormality. Psychotic patients showed a greater variety and severity of dysfunction.
<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Diffuse</th>
<th>Generalised</th>
<th>Paroxysmal</th>
<th>Focal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Psyhopathy</td>
<td>5</td>
<td>1</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Schizophrenia</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Psychotic Depression</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Total</td>
<td>8</td>
<td>2</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

Table 4: The type and severity of the electrical disturbance and its relationship to the diagnosis of patients.
Fig. 4. The distribution of normal and abnormal EEGs in various age groups.

(Each column represents the total number of cases in the age group.)
The type and severity of the disturbance in the EEGs of the control group is shown in Table 5. Four out of five subjects with abnormal EEGs had diffusely abnormal records and one showed generalised abnormality.

<table>
<thead>
<tr>
<th>Type of EEG Abnormality</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<tr>
<td></td>
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<tr>
<td></td>
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<td></td>
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<td></td>
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<tr>
<td></td>
</tr>
</tbody>
</table>

Table 5: The type and severity of the electrical disturbance in the EEGs of control group

The distribution of normal and abnormal EEGs in various age groups is shown in Fig. 4. Chi-Square test demonstrated no significant correlation between age and incidence of EEG abnormality ($X^2$ values for the age groups 20-25 and 26-30; 20-25 and 30-40; 31-35 and 50+; and 36-40 and 50+ was 0.015, 0.74, 0.18 and 0.097 respectively).

The relationship of age, diagnosis and type of EEG abnormality is shown in Table 6. The incidence of normal and abnormal EEGs in relation to the age and diagnosis of the patients is shown in Fig. 5. Schizophrenic and patients with psychotic depression are combined and designated as 'psychotics'. Three patients above 50 years of age (2 psychopaths with normal EEG and one schizophrenic with abnormal EEG) are not included in the figure. No significant correlation was demonstrated between age, diagnosis and type of abnormality.
Fig. 5. The incidence of normal and abnormal EEGs in relation to the age and diagnosis of the patients.
(Three patients above 50 years of age are not included.)
Table 6: The relationship of age diagnosis and type of EEG abnormality
(Psych: Psychopath; Sch: Schizophrenia; Dep: Psychotic Depression; Diff: Diffuse Gen: Generalised; Foc: Focal; Parox: Paroxysmal)

A comparison of the dominant frequencies in controls and patients is shown in Fig. 6. It is apparent that the distribution is similar in the two groups except that two records in the experimental group were dominated by frequencies which were below alpha range. A four-way analysis of variance demonstrated that the variance within the experimental group did not differ significantly from the variance of the control group.

In 17 records (53.2%) no significant change was noted in response to photic stimulation. In 5 records stimulation effected at the rate of 5 flashes per second (f.p.s.) resulted in increased activity, 2 of which showed increased activity below alpha frequencies and the remaining 3 records, augmentation was in the alpha range. A stimulation effect at 8-18 f.p.s. was apparent in 10 records, half of which showed increased activity below alpha frequencies and the other half had increased activity in the alpha range.
Fig. 6. DOMINANT FREQUENCY IN THE PATIENTS AND CONTROLS
Existing abnormalities were augmented in one record but photic stimulation failed to evoke any abnormalities which were not present already in the resting or hyperventilation record.

The results of analysis in terms of alpha factors and amount of low voltage amorphous activity (LVA) is shown in Tables 7 and 8 in experimental and control groups respectively. P4 and P5 types of records were considered to be unsuited for visual analysis and therefore excluded for the purposes of quantification. In Fig. 7 and Fig. 8 alpha factor and LVA is represented graphically as mean activity of the posterior four channels (two from each hemisphere) in three different situations, namely medial longitudinal position, lateral longitudinal position and activity recorded during overbreathing in the lateral longitudinal position. The posterior channels in the medial longitudinal position represent the activity of occipito-parietal and posterior parietal regions of each hemisphere, while posterior channels in the lateral longitudinal position record the potentials from occipito-temporal and posterior temporal zones of each hemisphere. It is obvious from figures 7 and 8 that in resting EEGs the amount of mean alpha factor and LVA is not significantly different in two groups. Indeed statistical analysis gave the following values of t for the difference between the t's in two groups:

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean LVA</td>
<td></td>
</tr>
<tr>
<td>(Medial)</td>
<td>0.42</td>
</tr>
<tr>
<td>(Lateral)</td>
<td>0.20</td>
</tr>
<tr>
<td>(Hyperventilation)</td>
<td>0.54</td>
</tr>
<tr>
<td>Alpha factor</td>
<td></td>
</tr>
<tr>
<td>(Medial)</td>
<td>0.48</td>
</tr>
<tr>
<td>(Lateral)</td>
<td>0.53</td>
</tr>
<tr>
<td>(Hyperventilation)</td>
<td>0.77</td>
</tr>
<tr>
<td>Serial No.</td>
<td>Pattern of electrical activity</td>
</tr>
<tr>
<td>-----------</td>
<td>--------------------------------</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>P5</td>
</tr>
<tr>
<td>2</td>
<td>P1</td>
</tr>
<tr>
<td>3</td>
<td>P3</td>
</tr>
<tr>
<td>4</td>
<td>P5</td>
</tr>
<tr>
<td>5</td>
<td>P2</td>
</tr>
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<td>6</td>
<td>P3</td>
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<td>7</td>
<td>P1</td>
</tr>
<tr>
<td>8</td>
<td>P1</td>
</tr>
<tr>
<td>9</td>
<td>P3</td>
</tr>
<tr>
<td>10</td>
<td>P3</td>
</tr>
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<td>11</td>
<td>P3</td>
</tr>
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<td>12</td>
<td>P3</td>
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<td>13</td>
<td>P4</td>
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<td>14</td>
<td>P3</td>
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<td>15</td>
<td>P3</td>
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<td>16</td>
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<td>17</td>
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<td>18</td>
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<td>19</td>
<td>P4</td>
</tr>
<tr>
<td>20</td>
<td>P4</td>
</tr>
<tr>
<td>21</td>
<td>P3</td>
</tr>
<tr>
<td>22</td>
<td>P4</td>
</tr>
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<td>23</td>
<td>P3</td>
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<td>24</td>
<td>P3</td>
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<td>25</td>
<td>P3</td>
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<td>26</td>
<td>P3</td>
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<td>27</td>
<td>P3</td>
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<tr>
<td>28</td>
<td>P3</td>
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<td>29</td>
<td>P3</td>
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<td>30</td>
<td>P3</td>
</tr>
<tr>
<td>31</td>
<td>P3</td>
</tr>
<tr>
<td>32</td>
<td>P3</td>
</tr>
</tbody>
</table>

Table 7: Mean activity of posterior two channels from each hemisphere in medial longitudinal position and lateral longitudinal position recorded under resting conditions, and potentials recorded during hyperventilation in lateral longitudinal position (EXPERIMENTAL GROUP).
Table 8: Mean activity of posterior 2 channels from each hemisphere in medial longitudinal position and lateral longitudinal position recorded under resting conditions, and potentials recorded during hyperventilation in lateral longitudinal position (CONTROL GROUP)
Fig. 7. THE DISTRIBUTION OF MEAN ALPHA FACTOR IN THE TWO GROUPS.

DURING HYPERVENTILATION
- **PATIENTS**
- **CONTROLS**

LATERAL POSITION

MEDIAL POSITION

NUMBER OF CASES

MEAN ALPHA FACTOR
Fig. 8. THE DISTRIBUTION OF MEAN L.V.A. FACTOR IN THE TWO GROUPS

During Hyperventilation

Lateral Position

Medial Position

Number of Cases

Mean L.V.A.
It was interesting to note that during hyperventilation in the control group, 15 out of 25 records (60%) demonstrated an increase of alpha factor in the 80-90 range but still it did not reach fiducial level. Such an augmentation of alpha factor on hyperventilation was not demonstrated in the EEGs of patients and only 29.1% of the experimental group presented maximum activity in 60-70 range.

Only one of the subjects included in the control group demonstrated sub-alpha factor (SAF) but in the remainder it was either completely absent or less than 2. In the experimental group 11 patients in their resting EEGs showed measurable SAF in the medial position and in 14 patients it was evident in the lateral position either in resting or hyperventilation records. Hyperventilation effects were by no means consistent in all records. 2 patients demonstrated SAF only during overbreathing; in two cases it became less marked on hyperventilation. In 2 records it was relatively diminished but in 8 records its quantity was appreciably increased.

The number of EEGs in various categories classified according to the pattern of electrical activity and the incidence in various diagnostic groups is shown in Table 9. A comparison of the distribution of various patterns of electrical activity in the experimental and control groups is shown in Fig. 9. It will be seen that 21 EEGs of patients (85.6%) were classified as P^ compared with only 4 records (12.5%) of the control group which were regarded as P type \( (X^2 = 18.95 p < 0.01) \). On the contrary the majority of the EEGs (53%) in control population were of P type and the corresponding figure in the experimental group was 9.4% \( (X^2 = 44.86 p < 0.01) \).
Fig. 9. CLASSIFICATION OF RECORDS ACCORDING TO THE PATTERN OF ELECTRICAL ACTIVITY IN PATIENTS AND CONTROLS.
In both groups $P_2$ EEGs were rare, number of $P_4$ was nearly the same, but controls had twice as many $P_5$ records as the patients.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>$P_1$</th>
<th>$P_2$</th>
<th>$P_3$</th>
<th>$P_4$</th>
<th>$P_5$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenia</td>
<td>1</td>
<td>-</td>
<td>11</td>
<td>3</td>
<td>-</td>
</tr>
<tr>
<td>Psychopathy</td>
<td>2</td>
<td>1</td>
<td>7</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Psychotic depression</td>
<td>-</td>
<td>-</td>
<td>3</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>3</strong></td>
<td><strong>1</strong></td>
<td><strong>21</strong></td>
<td><strong>5</strong></td>
<td><strong>2</strong></td>
</tr>
</tbody>
</table>

*Table 9: The diagnosis and pattern of electrical activity*
<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Neurotic traits &amp; schooling</th>
<th>Marital status</th>
<th>Alcohol intake</th>
<th>Premorbid personality</th>
<th>Sexual deviations</th>
<th>Previous psychiatric history</th>
<th>Head injury</th>
<th>Family History</th>
<th>Present Mental State</th>
<th>Number of previous convictions &amp; age on 1st conviction</th>
<th>Type of murder</th>
<th>FFG</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>39</td>
<td>Psychopathy</td>
<td>NT† Average Scholar</td>
<td>Single</td>
<td>+</td>
<td>None of significance</td>
<td>+</td>
<td>+</td>
<td>—</td>
<td>Psychosis</td>
<td>Non-psychotic</td>
<td>None</td>
<td>Sex</td>
<td>Normal P5, Grade I</td>
</tr>
<tr>
<td>2</td>
<td>47</td>
<td>Schizophrenia</td>
<td>NT Average Scholar</td>
<td>Single</td>
<td>+</td>
<td>None of significance</td>
<td>Schizophrenic since the age of 24</td>
<td>—</td>
<td>ISolate</td>
<td>Psychosis</td>
<td>Psychotic</td>
<td>None</td>
<td>Motiveless</td>
<td>Normal P1, Grade I</td>
</tr>
<tr>
<td>3</td>
<td>22</td>
<td>Schizophrenia</td>
<td>NT† Average Scholar</td>
<td>Single</td>
<td>+</td>
<td>Schizoid</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Psychosis</td>
<td>Flat ened effect</td>
<td>None</td>
<td>Motiveless</td>
<td>Normal P3, Grade II</td>
</tr>
<tr>
<td>4</td>
<td>43</td>
<td>Psychopathy</td>
<td>NT† Attended Approved School</td>
<td>Single</td>
<td>++</td>
<td>None of significance</td>
<td>+</td>
<td>+</td>
<td>—</td>
<td>Psychosis</td>
<td>Non-psychotic</td>
<td>Petty crime</td>
<td>1</td>
<td>Multiple sex murders</td>
</tr>
<tr>
<td>5</td>
<td>51</td>
<td>Psychopathy</td>
<td>NT Poor Scholar</td>
<td>Single</td>
<td>+</td>
<td>Maladjusted Sexual perversion</td>
<td>+</td>
<td>— Sustained at 11 Sustained at 17</td>
<td>—</td>
<td>Attempted suicide &amp; alcoholism</td>
<td>Non-psychotic</td>
<td>Petty crimes</td>
<td>4</td>
<td>Violence</td>
</tr>
<tr>
<td>6</td>
<td>47</td>
<td>Psychopathy</td>
<td>NT Poor Scholar</td>
<td>Married</td>
<td>+</td>
<td>None of significance</td>
<td>+</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Non-psychotic</td>
<td>Petty crimes</td>
<td>2</td>
</tr>
<tr>
<td>7</td>
<td>55</td>
<td>Psychopathy</td>
<td>NT Poor Scholar</td>
<td>Single</td>
<td>+</td>
<td>Unstable</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Non-psychotic</td>
<td>Petty crimes</td>
<td>8</td>
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</tr>
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<td>8</td>
<td>24</td>
<td>Psychopathy</td>
<td>NT Poor Scholar</td>
<td>Single</td>
<td>++</td>
<td>Unstable Sexual perversion</td>
<td>+</td>
<td>—</td>
<td>—</td>
<td>Alcoholism</td>
<td>Aggressive</td>
<td>Petty crimes</td>
<td>2</td>
<td>Violence</td>
</tr>
<tr>
<td>9</td>
<td>23</td>
<td>Schizophrenia</td>
<td>NT† Average Scholar</td>
<td>Single</td>
<td>++</td>
<td>Aggressive</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Psychosis</td>
<td>Psychotic</td>
<td>Violence</td>
<td>1</td>
<td>Motiveless</td>
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<tr>
<td>10</td>
<td>31</td>
<td>Schizophrenia</td>
<td>NT Poor Scholar</td>
<td>Single</td>
<td>+</td>
<td>Unstable Schizoid</td>
<td>Schizophrenic since the age of 17</td>
<td>—</td>
<td>Left frontal-parietal focus</td>
<td>Mental deficiency</td>
<td>Psychotic</td>
<td>None</td>
<td>Provoked</td>
<td>Right frontal-parietal focus</td>
</tr>
<tr>
<td>11</td>
<td>32</td>
<td>Schizophrenia</td>
<td>NT Average Scholar</td>
<td>Married</td>
<td>++</td>
<td>Unstable Schizoid</td>
<td>Schizophrenic since the age of 29 Suicidal attempts</td>
<td>—</td>
<td>Neurosis</td>
<td>Psychopath Alcoholism</td>
<td>Psychotic</td>
<td>Violence</td>
<td>1</td>
<td>Provoked</td>
</tr>
<tr>
<td>12</td>
<td>37</td>
<td>Schizophrenia</td>
<td>NT† Average Scholar</td>
<td>Single</td>
<td>+</td>
<td>Impulsive</td>
<td>First illness at 15</td>
<td>—</td>
<td>—</td>
<td>Psychotic</td>
<td>Sex crimes</td>
<td>3</td>
<td>Sex</td>
<td>A mild paranoid abnormality</td>
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</table>

**Table 10**
<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Neurotic traits &amp; schoolings</th>
<th>Marital status</th>
<th>Alcohol intake</th>
<th>Premarital personality</th>
<th>Sexual deviations</th>
<th>Previous psychiatric history</th>
<th>Head injury</th>
<th>Epilepsy</th>
<th>Family History</th>
<th>Present Mental Status</th>
<th>Number of previous convictions &amp; age on 1st conviction</th>
<th>Type of Murder</th>
<th>EEG</th>
</tr>
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<tbody>
<tr>
<td>13</td>
<td>20</td>
<td>Psychopathy</td>
<td>NT - Average Scholar</td>
<td>Single</td>
<td>+</td>
<td>Withdrawn</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>None</td>
<td>Premediated</td>
<td>Diffuse abnormality (P4, Grade III)</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>42</td>
<td>Psychotic depression</td>
<td>NT + Average Scholar</td>
<td>Married</td>
<td>+</td>
<td>None of significance</td>
<td>Depressive</td>
<td>Illness at 33</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Episodes of depression</td>
<td>None</td>
<td>Multiple</td>
<td>Bilateral generalized runs of high voltage &amp; slow waves (P3, Grade IV)</td>
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<tr>
<td>15</td>
<td>58</td>
<td>Schizophrenia</td>
<td>NT - Poor Scholar</td>
<td>Single</td>
<td>++</td>
<td>Eratic &amp; unstable</td>
<td>+</td>
<td>Leucotomized at 40</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Marked personality deterioration</td>
<td>Petty crimes 7</td>
<td>Attempted murder</td>
<td>Diffuse abnormality &amp; runs of theta and delta in frontal regions (P3, Grade III)</td>
</tr>
<tr>
<td>16</td>
<td>50</td>
<td>Psychopathy</td>
<td>NT + Average Scholar</td>
<td>Single</td>
<td>++</td>
<td>Unstable &amp; maladjusted</td>
<td>Attempted</td>
<td>Suicide</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Non-psychotic</td>
<td>Violence 7 (11)</td>
<td>Motiveless</td>
<td>Diffuse abnormality with a tendency to appear in runs (P3, Grade III)</td>
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<tr>
<td>17</td>
<td>38</td>
<td>Schizophrenia</td>
<td>NT - Average Scholar</td>
<td>Single</td>
<td>+</td>
<td>Schizoid</td>
<td>+</td>
<td>Onset of schizophrenia at 26</td>
<td>Sustained at 13</td>
<td>–</td>
<td>–</td>
<td>Psychotic &amp; aggressive</td>
<td>Violence 2 Sexual 2 (13)</td>
<td>Sex</td>
<td>Diffuse abnormality P3, Grade III</td>
</tr>
<tr>
<td>18</td>
<td>38</td>
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<td>Divorced</td>
<td>++</td>
<td>Aggressive</td>
<td>+</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Non-psychotic</td>
<td>Violence 2 Petty crime 1 (20)</td>
<td>Motiveless</td>
<td>Only minor irregularities but within normal limits (P3, Grade III)</td>
</tr>
<tr>
<td>19</td>
<td>32</td>
<td>Schizophrenia</td>
<td>NT + Approved School</td>
<td>Married</td>
<td>+</td>
<td>Sensitive shy and suspicious</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Neurosis</td>
<td>Paranoic delusions</td>
<td>Motiveless</td>
<td>Generalized abnormality P4, Grade III</td>
</tr>
<tr>
<td>20</td>
<td>39</td>
<td>Schizophrenia</td>
<td>NT - Average Scholar</td>
<td>Married</td>
<td>++</td>
<td>Maladjusted</td>
<td>First breakdown 22</td>
<td>Mild head injury at 27</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Suspicious</td>
<td>Petty crimes 4 (15)</td>
<td>Motiveless</td>
<td>Diffuse abnormality P4, Grade IV</td>
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<tr>
<td>21</td>
<td>35</td>
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<td>NT - Average Scholar</td>
<td>Single</td>
<td>+</td>
<td>Quiet sociable</td>
<td>Onset of schizophrenia at 24</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Psychotic</td>
<td>Motiveless</td>
<td>Minor irregularities but within normal limits P3, Grade III</td>
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<tr>
<td>22</td>
<td>32</td>
<td>Schizophrenia</td>
<td>NT + Average Scholar</td>
<td>Married</td>
<td>+</td>
<td>None of significance</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Psychotic</td>
<td>Multiple</td>
<td>Generalized abnormality P4, Grade V</td>
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<tr>
<td>23</td>
<td>31</td>
<td>Psychotic depression</td>
<td>NT + Poor Scholar</td>
<td>Married</td>
<td>++</td>
<td>Anxious &amp; suspicious</td>
<td>Depressive</td>
<td>Illness at 26</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Psychosis</td>
<td>Episodes of depression</td>
<td>Multiple</td>
<td>Bursts of generalized 3-4 cps activity P3, Grade III</td>
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<tr>
<td>24</td>
<td>27</td>
<td>Psychopathy</td>
<td>NT - Average Scholar</td>
<td>Single</td>
<td>+</td>
<td>Maladjusted</td>
<td>Attempted suicide</td>
<td>Head injury at 27</td>
<td>Convulsions at 8</td>
<td>–</td>
<td>–</td>
<td>Psychosis</td>
<td>Non-psychotic</td>
<td>Violence 1 (22)</td>
<td>Provoked</td>
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<tr>
<td>25</td>
<td>47</td>
<td>Schizophrenia</td>
<td>NT - Average Scholar</td>
<td>Single</td>
<td>++</td>
<td>None of significance</td>
<td>Onset of Illness at 35</td>
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<td>–</td>
<td>–</td>
<td>–</td>
<td>Psychotic</td>
<td>Motiveless</td>
<td>Slow wave abnormality arising from left posterior temporal zone P3, Grade III</td>
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<tr>
<td>Case No.</td>
<td>Age</td>
<td>Diagnosis</td>
<td>Neurotic traits &amp; schooling</td>
<td>Marital status</td>
<td>Alcohol intake</td>
<td>Premorbid personality</td>
<td>Sexual deviations</td>
<td>Previous psychiatric history</td>
<td>Head injury</td>
<td>Epilepsy</td>
<td>Family History</td>
<td>Present Mental State</td>
<td>Number of previous convictions &amp; age on 1st conviction</td>
<td>Type of Murder</td>
<td>EEG</td>
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<td>26</td>
<td>43</td>
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<td>NT – Average Scholar</td>
<td>Single</td>
<td>+</td>
<td>Obsessional &amp; anxious</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Psychosis Episodes of depression</td>
<td>– Motiveless Diffuse abnormality P3, Grade III</td>
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<td>27</td>
<td>34</td>
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<td>NT + Approved School</td>
<td>Single</td>
<td>+</td>
<td>Unstable Destructive</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Psychopathy Non-psychotic Violence 3 Motiveless</td>
<td>Generalized abnormality P3, Grade III</td>
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<tr>
<td>28</td>
<td>36</td>
<td>Schizophrenia</td>
<td>NT –</td>
<td>Single</td>
<td>+</td>
<td>Maladjusted</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Neurosis Psychotic</td>
<td>– Motiveless          Record dominated with theta activity. Occasional delta activity seen in anterior zones, P3, Grade IV</td>
<td></td>
<td></td>
</tr>
<tr>
<td>29</td>
<td>27</td>
<td>Schizophrenia</td>
<td>NT + Poor Scholar</td>
<td>Single</td>
<td>++</td>
<td>Aggressive &amp; unpredictable</td>
<td>First admission at 20</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Neurosis Delusions Violence 1 Motiveless</td>
<td>Generalized abnormality P3, Grade IV</td>
<td></td>
<td></td>
</tr>
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<td>30</td>
<td>30</td>
<td>Psychopathy</td>
<td>NT + Approved School</td>
<td>Single</td>
<td>+</td>
<td>Sexual perversions, Aggressive</td>
<td>Attempted suicide</td>
<td>Head injury at 8</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Neurosis Psychotic Violence 5 (8) Premeditated Multiple murders</td>
<td>Diffuse abnormality P3, Grade III</td>
<td></td>
<td></td>
</tr>
<tr>
<td>31</td>
<td>48</td>
<td>Psychopathy</td>
<td>NT – Average scholar</td>
<td>Married</td>
<td>++</td>
<td>Inadequate Aggressive</td>
<td>Attempted suicide</td>
<td>Head injury at 34</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Mental deficiency and psychosis Violence 1 (20) Provoked</td>
<td>Diffuse abnormality P3, Grade III</td>
<td></td>
<td></td>
</tr>
<tr>
<td>32</td>
<td>42</td>
<td>Psychopathy</td>
<td>NT – Average scholar</td>
<td>Married</td>
<td>+</td>
<td>Sensitive unsociable</td>
<td>Attempted suicide</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Aggressive outbursts Provoked</td>
<td>Diffuse abnormality P4, Grade III</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**TABLE 10** shows details of Clinical & Electroencephalographic findings.

* Alcohol Intake:
  + None or moderate
  ++ Excessive
  + + Chronic alcoholic

* Previous convictions: The number following the type of offence indicates the number of offences committed. The number in the brackets represents the age when first crime was committed:
  (e.g. Petty Crime 2 patient was convicted twice and first conviction was at the age of 20).
CLINICAL FINDINGS

Table 10 shows details of clinical and EEG findings. Some of the clinical findings and their relationship to the normal and abnormal records are shown in Fig. 10 (A and B). The mean age when the crime was committed was 31 (standard deviation + 8.8). The age ranged from 16 to 49. Nearly a third of the patients were in their third decade and a similar number of patients were in fourth decade when the offence was committed. The age distribution of the group is represented graphically in Figure 11.

When patients are divided into psychotic and non-psychotic groups the mean age of committing the capital offence is 31 + S.D. 7.9 and 31 + S.D. 9.9 respectively. The diagnostic categories of the patients included in the study are as follows:-

- Schizophrenia - 15 cases (46.8%)
- Psychopathy - 14 cases (43.8%)
- Psychotic depression - 3 cases (9.4%)

As mentioned earlier the term psychopathy is used loosely covering personality defects, sociopathy and neurosis; indeed all those patients who did not show signs and symptoms of psychosis either before or after the admission were included in this category.

14 patients (43.1%) had definite neurotic traits during childhood. Neurotic traits included nail biting, thumb sucking, stammering and nocturnal enuresis. Bed-wetting was the commonest of all traits. 3 patients attended approved schools, of the remaining 29 patients who were educated in ordinary schools, 8 (27.1%) were considered poor scholars.
Fig. 10 (A)
The black columns show the number of cases with certain clinical findings and these are contrasted with the number of abnormal (white columns) and normal (hatched columns) EEG's in the group of patients characterized by such clinical findings.
The black columns show the number of cases with certain clinical findings and these are contrasted with the number of abnormal (white columns) and normal (hatched columns) EEG's in the group of patients characterized by such clinical findings.
Fig. 11. AGE DISTRIBUTION OF THE PATIENTS ON ADMISSION
There were 9 patients (28%) who were married when they committed the offence. One patient was divorced but for the purpose of analysis he was included in the unmarried group. Adopting the criterion that only those patients who were treated for alcoholism in the past would be classified as chronic alcoholics, there were two patients who met this criterion but 10 patients (31%) revealed history of excessive drinking.

Homosexual experience, fetishism, transvestism and indecent exposure were the examples of sexual deviations found among the histories of 10 patients (31%).

7 patients had head injuries. None of the patients had severe head injury. One patient sustained a moderately severe head injury, losing consciousness for a period of longer than 12 hours. Another patient had a trauma of similar severity but it happened after committing the crime; 5 patients had only minor injuries. It was interesting to note that one of the patients developed a psychotic illness immediately after an accident in which he suffered cerebral trauma.

Unexpectedly, none of the patients included in the series was suffering from epilepsy. There were 4 patients (12.5%) who had demonstrated epileptic phenomenon at some stage in their lives. 2 patients had seizures at the age of 8 and 11 respectively. One patient had a grand mal seizure after admission, the only instance in his life of having a fit, and, despite not being treated with anti-epileptic drugs, has had none since. One patient complained that he had had brief spells of loss of consciousness when he was 8 years old.
Since the sample included some patients who were hospitalised at the time when venereal disease was a common affliction, it was decided to scrutinise the case histories for the evidence of positive W.Rs or gonorrhoeal infections. None of the patients had a positive W.R. at any time in their lives. There were however two patients who had been treated for gonorrhoea before admission.

There were 25 patients (78%) who had positive histories for psychiatric illnesses before admission. A patient was considered to have had a psychiatric illness in the past only if he was treated in a mental hospital. Of the 7 patients who had negative histories, 2 were psychopathic, 1 had a psychotic depression and 4 were schizophrenics. Past histories suggested that patients were suffering from a similar illness at the time of committing the crime for which they had been treated previously, or to put it another way the diagnostic labels did not change a great deal over the years and remained the same before and after committing the crime. Six patients attempted suicides before admission, 5 of those patients were psychopaths and one had schizophrenia. Although it is not accurate to regard the age of the first admission as the time of the onset of illness because it can be argued that a patient could remain ill for a number of years before seeking treatment. However, in a retrospective study like this, the age at which a patient had his first admission to a psychiatric institution could be taken as the period of onset of illness. Comparing the psychotic and non-psychotic groups for the mean of number of years between the first admission and commission of crime was 6.4 (S.D. ± 3.8) in the former group and 7.3 (S.D. ± 5.8) in the latter group.
These are not significantly different \((t = 0.28)\).

Family history of psychiatric illness was considered positive only in those cases where a member of the family was admitted to a mental hospital for neurosis, alcoholism, psychopathy, psychosis or mental deficiency. It was further analysed whether it was a first degree relative or some other member of the family who was treated for mental illness. The incidence of psychiatric illnesses in the family histories was 59.4\%. Psychosis was the most common illness which occurred in the relatives of the patients.

A fairly accurate history of any previous criminal record was available in each case because it was presented by prosecution at the trial proceedings. Indictable offences in the past were classified into three categories. 18 patients (56.2\%) had committed offences prior to the capital offence. Total number of offences committed was 58. This included 27 crimes involving stealing, fraud, house-breaking etc.; 25 cases involving violence and the nature of the remaining 6 was sexual. The mean age at first conviction was 18 (S.D. ± 6.9).

Five patients committed multiple murders, including two patients who were diagnosed psychopaths and the remaining three were psychotics. One of the psychotics was a schizophrenic and two had psychotic depression. It was noted that all three psychotic patients were grossly deluded and killed their families. The main theme of delusions in the cases of schizophrenic patients was of a persecutory nature but in cases of psychotic depression the main content was based on 'guilt' and self depreciation. Half the patients committed motiveless murders.
As mentioned earlier, crimes resulting from delusional thinking were included in this category. In 3 cases the pattern of delusions conformed to psychotic depression and in 9 cases delusions were clearly of a schizophrenic nature, in the remaining 4 cases the murders with little or no apparent motive were committed by patients diagnosed as psychopaths. In seven cases (21.8%) there was clear evidence of provocation preceding the act of murder. In such cases, the typical pattern was that of a quarrel between husband and wife followed by an extreme rage of the patient culminating in the death of the victim.

In six cases (18.8%) the murder was preceded by some kind of sexual activity. 3 cases (9.4%) were classified as premeditated murders. One of the patients in this group was a non-psychotic, There was a long history of difficult interpersonal relationships between the patient and his victim and patient considered he would be much happier if he could remove from his life situation the person with whom he was in conflict. It finally ended in his killing the victim. The remaining two cases were those of psychopaths who killed their victims after planning in order to achieve the furtherance of another crime.

Victims, in 16 cases, were relatives, including 5 matricides, 1 grandmother, 1 father, 1 brother, 1 sister-in-law, 5 wives, and 4 offsprings (2 cases were multiple murders involving wives and children). In the remaining 16 cases, 10 were strangers and 6 were acquaintances. The victims of all married patients included in the study were relatives (except in the one case who was divorced at the time of the offence). In 11 cases the victims were below/
the age of 16.

6 patients were considered to have resorted to severe violence and 8 patients claimed complete amnesia regarding the events of killing.
DISCUSSION

Before discussing the main findings in the study it would be appropriate to point out some of the problems inherent in the field of electroencephalography. First there is reliability of recording instruments, secondly there is the problem of reliability of the reading procedure itself and finally assuming that a reliable EEG recording has been produced, there remains the question of reliability of interpretation. The clinical interpretation is, to a large extent, still a subjective procedure.

In order to overcome these difficulties, it was decided that every patient should have EEG examinations on two different occasions and all records were designated by a different code number. The author was kept ignorant as to which records came from the same patient. After completing the evaluation, the EEGs were decoded and the result of the examination compared.

The findings in both first and second record were consistent in 84.4% of the EEGs. The inconsistency of results arose in records classified as demonstrating a 'mild diffuse abnormality', particularly in borderline records. In some cases abnormality was demonstrated only in one of the two records whereas the other record was considered to be normal. These observations merely emphasise Houfek and Ellingson's views stressing the value of repeated EEG examination in doubtful cases.

Quantification of EEGs by means of manual methods has been/
been undertaken by many investigators. In the present study the main purpose of quantification of EEGs was to test the hypothesis of previous workers that there was an excess of slow activity in the EEGs of patients with behaviour disorders. Needless to say the method not only proved to be laborious, tedious and time consuming but also was an inaccurate measure of the electrical activity of the brain. The major criticism of such a procedure is that the brain activity is a dynamic process which alters from one moment to another. To consider the measurements of such a phenomenon on a strip of record one meter in length is undoubtedly a gross over-simplification and at best can be only a very crude approximation.

An experimental procedure, to be useful, must be reliable — that is, there must be no significant difference in the result regardless of where, when, or by whom the operations are performed. It must be admitted that the technique of visual analysis is largely a subjective measure. There is a wide margin of error in counting the number of waves in a strip of EEG record. It is unlikely that quantification of the same record by two investigators utilising naked eye analysis would yield similar results. Therefore it is fair to say that this method is an unreliable measure. By and large the classification of records according to the pattern of electrical activity appears to be more reliable. It entails forming a total impression of the tracings but certainly it does not eliminate the element of subjectivity. Nevertheless the/
the proportion of agreement between two experienced electroencephalographers is usually remarkably high.

The complexity of EEGs and also the limitations of visual analysis have been emphasised by Dawson and Grey Walter. EEGs are essentially a graph of the averaged sum of the potential differences with respect to time. It is an established fact that the rhythms in the EEG are rarely simple harmonic oscillations and often have a number of components. Visual examination of an EEG record and direct measurement of the waves in it, is reliable so long as the rhythms under investigation are significantly longer than any others present. However, when the rhythm which is being studied becomes comparable in size to or smaller than others which are present, a visual examination of the tracings may be completely misleading. On the basis of these arguments it is explicable that the slow activity which is uncountable in some of the records of the experimental group became prominent on hyperventilation and similarly the mean alpha factor in records of the control group increased simply because, on hyperventilation, the amplitude of the rhythm was augmented and it became distinct and easy to count.

A four-way analysis of variance demonstrated that the variance within the experimental group did not differ significantly from the variance of the control group. This is most likely a sampling error rather than a finding of significance because the strips of record for quantification were selected on the criteria that it should be free of abnormal rhythms as far as possible/
possible and component waves should be easily countable. This resulted in measurements based in each case on essentially normal tracings.

In spite of such a crude technique, it is interesting to note that a significantly higher proportion of EEGs from the experimental group exhibited countable slow waves. The presence of excess of slow activity in patients with behaviour disorders confirms the finding of other investigators. The results from the B.N.I. analyser are not included in the study because, due to unavoidable circumstances, automatic analysis was not carried out on the EEGs of the control group. It is proposed in any future study to analyse the records of controls by this technique to enable a comparison with the experimental group.

The overall incidence of 65.6% abnormal EEGs in the experimental group compared with 15% in the normal controls is statistically significant ( \( \chi^2 = 55.3 \ p < 0.001 \)). The findings are in agreement with Mundy-Castle but show a higher proportion of abnormal EEGs than reported by Stafford-Clark and Taylor and Hill and Pond (50% and 47.6% respectively). This is understandable because of difference between the populations in the present series and two of the above mentioned studies, Stafford-Clark and Taylor and Hill and Pond basing their investigations on heterogenous group of murderers including some who were not psychiatrically ill, whereas the present study is exclusively based on murderers who were/
/were considered insane at the trial proceedings and therefore hospitalized. The murderers studied by Mundy-Castle were drawn from mental hospital; hence the present sample and his sample are of similar composition and findings are essentially identical. Gibbs et al.\textsuperscript{24} reported an incidence of 31\% abnormal EEGs among the murderers; again the disagreement in the results can be attributed to the fact that murderers included in his study were legally sane and not considered psychiatrically ill clinically.

Stafford-Clark on the basis of clinical assessment classified his subjects into five groups. It would be legitimate to compare his findings in two of his groups namely sex murderers and insane murderers with the present series because of the similarities of the populations. Four out of eight sex murderers included in his series had abnormal EEGs. Fourteen cases included in Stafford-Clark's series were considered to be insane, demonstrating an incidence of 85.7\% abnormal EEGs, which is somewhat higher than the observed incidence in the present series. Apparently there were five confirmed epileptics who were included in Stafford-Clark's series whereas none of the subjects included in the present study was epileptic - which explains the inconsistancy of results.

The present study confirms the incidence of abnormal EEGs among insane murderers reported by Hill and Pond.\textsuperscript{44} The authors extended Stafford-Clark's series\textsuperscript{113}, which included 22 insane murderers and demonstrated an incidence of 68\% EEG abnormality in this group./
On the basis of clinical assessment it was possible to classify the patients into two groups namely 'psychotic' and 'psychopaths'. The incidence of abnormal EEGs among the two groups was 77.8% and 50% respectively. These incidences are significantly different ($\chi^2 = 16.75 \ p<0.001$).

It appears established in view of the observations by many investigators that nearly half of all psychopathic personalities have abnormal EEGs. The 50% incidence of abnormal EEGs among the psychopaths in the present study merely underlines the observation of other investigators.

One could assume that psychopaths who kill should be more aggressive than other psychopaths and therefore it would be justified to compare the present findings with Hill's findings in aggressive psychopaths. Such an assumption would be misleading since the psychopaths included in the study were a heterogeneous group which included personality disorders, sociopaths etc.; indeed all patients who were not psychotic were included in this category.

Silverman's observations are surprisingly different from those of other investigators. He reported that the incidence of abnormal EEGs among the psychotic and psychopathic prisoners was 45% and 75% respectively. This observation is in sharp contrast to our series which showed that psychotics had a greater proportion of abnormal EEGs than psychopaths. In his study the diagnostic/
/diagnostic criteria are not reported. It is possible that the discrepancy in results is due to difference in type of populations.

The reports of EEG findings among schizophrenic patients in ordinary mental hospitals vary considerably. The highest incidence of abnormality was 60% reported by Kennard and Levy\textsuperscript{59}, but in other reports it varies from 44% to 23%. It is apparent from the investigations of Stafford-Clark, Hill and Pond, Mundy-Castle and the present series that the incidence of abnormal EEGs among schizophrenics who have killed is strikingly high compared with schizophrenics who have not killed.

This observation puts forward a strong case for further investigation based on electroencephalographic studies of psychotic murderers compared with matched controls drawn from an ordinary mental hospital population.

The collected EEG findings with clinical histories were subjected to Kendall's tau analysis and a synopsis of the limits of the unit normal deviate at the two acceptable levels of probability (5% and 1%) are shown in Table 11. The significant relationships are shown in Table 12.

Synopsis of Statistical Technique/
Synopsis of Statistical Technique

Kendall's Tau Analysis* was used to give unit normal deviates.

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<tr>
<th>Probability</th>
<th>Limits of Area under N(0,1) p.d.f.</th>
<th>Limits of unit Normal deviate</th>
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<td>0.01</td>
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* Dr. K Hope kindly supplied the computer program for this analysis.

The attempt to demonstrate a relationship between the nosological status of the patients and EEG patterning revealed that psychopathy was unrelated to any specific type of electrical activity. This finding is in contradiction to previous investigations. As mentioned earlier patients included in the present study who were diagnosed as psychopathic personalities constituted a heterogenous group. This category embraced all patients who were non-psychotic. Therefore, the failure to demonstrate an association between EEG and psychopathy reaching an acceptable level of statistical significance is not surprising. On the other hand, most of the psychotics included/
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<td>Sibs psychiatrically ill</td>
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included in this study were grossly disturbed, presenting a well defined clinical picture and there was hardly any doubt in establishing a diagnosis of psychotic depression or schizophrenia. This resulted in fairly homogenous sub-groups both of which demonstrated a relationship with certain types of EEG abnormalities.

A focal disturbance in an individual who is clinically schizophrenic and who had never been known to have had a seizure is well documented in the literature and the present study provides further evidence of this. Paroxysmal abnormalities demonstrated a positive relationship with psychotic depression. No doubt transient disturbance of this nature has been described in psychiatric patients by a number of investigators but there seems to be no study exclusively designed to establish such an inter-relationship between psychotic depression and EEGs with paroxysmal outbursts. It should be added that paroxysmal abnormalities are by no means specific and many investigators would agree that it is not unusual to find paroxysmal outbursts in the EEGs of patients with behaviour disorder. Although this type of disturbance is most commonly seen in the records of epileptics it does not necessarily imply that the pathophysiological mechanisms involved in epilepsy and behaviour disorders are essentially similar. Williams regards transient disturbances as representative of inborn constitutional abnormality of the C.N.S. This may manifest itself in the subject or his off-spring as behaviour disturbances which may be psychoneurotic, psychopathic, psychotic or epileptic in nature. However,
However, the existence of paroxysmal dysfunction in the EEGs of murderers with psychotic depression requires further investigation.

The negative relationship between paroxysmal disturbance and single murder is explicable on the basis that this type of electrical phenomenon was observed predominantly among severely disturbed psychotics who had committed multiple murders involving members of their own family. The results of the statistical analysis also indicated that the victims of patients with focal abnormalities were also members of their own families. Thus one might suggest that severely disturbed psychotics demonstrating focal or paroxysmal abnormalities in their EEGs are likely to commit multiple murders involving their family members. Of course this hypothesis warrants further investigations and if validated could have important practical implications in the sense that the EEG could be utilised as an indicator for assessing the risk of violence in psychotics. Comparison of the EEG patterns of psychotic murderers with a matched group of non-murderer psychotics could clarify this issue.

For the purpose of this study, background rhythm activity disorders were classified as generalised or diffuse abnormalities. Some electroencephalographers believe that there is no real distinction between these types of abnormality and that the terms/
/terms have mainly descriptive usefulness. On the other hand, some workers are of the opinion that such a distinction is more than merely an academic exercise because, in recognising these types of dysfunctions, one can infer the probable site of disturbance. They believe that generalised abnormalities originate from deeply placed structure and effect the brain as a whole, while diffuse abnormalities are caused by widely scattered groups of dysfunctionsing neuronal populations at a relatively more superficial level. The presence of diffuse abnormalities in cases of head injury is therefore understandable and the present study confirms this relationship.

The records with generalised dysfunction were dominated by rhythms less than 8 c.p.s. Hill considers theta dominated records as one of the phenomena representative of maturation defect. Hence the EEGs in the present series which were designated as demonstrating generalised dysfunction can be regarded as indicative of maturation defect.

Such an assumption can be supported further with the existence of a relationship between generalised EEG abnormality and neurotic traits, which positive relationship emerged as a result of statistical analysis. Michaels and Secunda^79 found a significant correlation between abnormal EEGs and a history of bed-wetting in children with behaviour disorders. Admittedly no attempt was made to analyse the various traits separately,
/separately, but of the several traits investigated enuresis found to be the most common traits in the histories of patients. The presence of neurotic traits in childhood has been considered to be the result of generalised malintegration at the biological level. This would suggest that our patients with histories of neurotic traits in childhood had some degree of brain dysfunction from an early age. Demonstration of generalised abnormality in these individuals signifies the persistence of this defect, thereby reflecting a life long disability to adjust themselves to the environment.

It also emerged that generalised EEG abnormalities were positively related to a family history of neurosis but there was a negative relationship with a family history of psychosis. Inter-relationships between family history of neurosis and history of psychotic illnesses among first degree relatives would seem to imply that patients with generalised EEG dysfunction have a family history loaded with morbid heredity. It might be argued that generalised dysfunction may be inherited, especially when there is some evidence in favour of heredity or genetic determination of electrical patterns.66. At the same time it must be pointed out that the role of the environment should not be under-estimated. Although an EEG abnormality is a neurophysiological expression of underlying psychological disturbance, manifested as deviant personality, it is also a persistant handicap for its possessor, hindering his ability to adapt himself to the environment,
/environment, thereby making his environment hostile towards him, which, in turn, elicits defective responses from the individual.

The relationship between a past history of convictions for petty crimes, sex murders, family history of alcoholism and normal EEGs is explicable on two alternative hypotheses. Firstly, it might be that these factors are not associated with organic deficit; therefore, they are non-contributory to EEG abnormality. On this hypothesis, then, all these factors would be considered to be predominantly of psychological origin. The alternative hypothesis would be that these factors are dependent upon brain dysfunction but that EEG is a technique which is not subtle enough to detect this defect.

Without involving ourselves in the controversy of organic versus functional, one might suggest that there is some truth in both hypotheses. Probably deviant behaviour is a product of both psychological as well as organic factors but the available techniques are incompetent to discover the organic deficit. In the present state of our knowledge it would be profitable to follow the psychodynamic parameters but at the same time endeavour to involve new techniques which will enable the exploration of brain dysfunction.

According to the Home Office Research Unit Report on Murder\textsuperscript{31} the proportion of the insane murderers with previous convictions/
/convictions rose from 17% to 32% over the period from 1955 to 1960. These figures are based on statistics for England and Wales. Among our patients, 56% had previous convictions. This does not necessarily imply that a higher proportion of patients in Scotland have had previous convictions, because the present sample is too small on which to form any opinion.

The relationship between sex murder and previous convictions for sexual offences was confirmed on statistical analysis. None of the victims of sex murderers was a relative. Previous convictions for crimes against property had a positive relationship with normal EEGs, but other types of previous convictions has no significant relationship to the EEGs. The victims in cases where there were previous convictions against property were also non-relatives and were younger than the offenders. It is of interest to point out that as may be expected, psychopathy and previous convictions for violent crimes demonstrated a positive relationship.

Thus there are some patterns which are discernable on the basis of the present investigation. The victims of psychotic murderers are usually members of their family and a higher proportion of these patients have abnormal EEGs. At the same time victims of sex murderers and patients with previous convictions for crimes against property are non-relatives. These two groups of patients also demonstrated a positive relationship with normal EEGs. Perhaps one might postulate that different aetiological/
/aetiological factors contribute to the various groups. The organic deficit demonstratable by EEG might be an important contributory factor in the causation of murder by psychotics, whereas in non-psychotic patients with normal EEGs the aetiology might be altogether different. It is note-worthy that sex murders, family history of alcoholism and previous convictions for petty crimes are factors which are positively related to normal EEGs and therefore most probably not organically determined.

In the present series motive of murder was defined in the legal sense in which motive is used to describe what might be more accurately called the apparent precipitating cause. The cause may be no more than the most convenient social label which the community can apply. It must be emphasised that it is a hazardous process to infer motives from overt behaviour. The term motive has in fact two meanings: the reason for a particular action in the mind of the individual and the cause assigned to that action by other members of society.

However, despite the difficulties of inferring the motives of the murders in our patients, it is interesting to note a negative relationship between motiveless murders and normal EEGs. This finding is in complete agreement with the Stafford-Clark series. Nonetheless, it must be pointed out that the population included in the present series is not exactly comparable to Stafford-Clark's motiveless murders. In his sample the motiveless murders/
murders. In his sample the motiveless murders were apparently sane and showed no evidence of overt psychiatric illness but our series is based entirely on patients who were unquestionably psychiatrically ill. This implies that a significant proportion of motiveless murderers whether sane or insane have abnormal EEGs.

A negative relationship between average school performance and normal EEGs is difficult to understand. One might postulate that patients whose school performance was average were apparently well adjusted till a certain stage in their lives. Average school performance also had a negative relationship with a family history of neurosis, previous convictions for crimes against property and aggressive crimes. Again these inter-relationships would suggest that these individuals had stable home backgrounds and deviant behaviour was not evident, but at a later period in their lives these patients broke down. This illness probably resulted in an abnormal brain function and abnormal EEG. The tragedy of murdering another human being occurred because of an abnormally functioning brain. Whether they had a normal or abnormal EEG prior to the onset of illness is difficult to say. Probably these were representative of so called normal individuals with abnormal EEGs encountered by all electroencephalographers in their every day practice.

Certain factors which did not reveal inter-relationships are worth commenting on too. It would appear that the pattern of alcohol intake over the years prior to the capital offence/
/offence played an insignificant part in the pathogenesis of
of the murder. As pointed out earlier none of the patients
investigated were epileptics. Furthermore, a past history
of convulsions or other epileptic phenomena seemed to be
unrelated to abnormal EEGs or other clinical variables. It
should be stressed that the association between epilepsy and
murder has been over-emphasised and our study suggests that
this concept has to be abandoned.

The remaining inter-relationships are not associated
with EEG and are therefore outside the scope of this thesis.
However, some of them are interesting and reveal easily
discernable patterns and will be briefly touched upon.

Psychopaths, in contrast to the schizophrenics, attended approved
schools and, prior to committing a capital offence had previous
convictions for aggressive crimes. A higher proportion of these
patients committed motiveless murders.

Patients who were married had no history of sexual
deviations or previous convictions for aggressive crimes;
their murders were not sexually motivated; they killed the
members of their own families and had a significant amnesia
subsequently for the events of killing. In these individuals
it was apparent that murder was one of the symptoms of their
psychosis. From the point of saving life, further investigations
in this category of patient might be more than useful in
preventing the tragic outcome.
Family history of neurosis and alcoholism emerged as a relatively more important factor than a family history of psychopathy and/or psychosis. A significant proportion of patients with a positive family history of neurosis had father and mother and siblings who had been treated for psychiatric illnesses in mental hospital. It suggests that the entire family unit was ill adjusted. A family history of alcoholism revealed a positive relationship with a family history of psychopathy, treatment of father and second degree relatives for psychiatric illnesses and sexually motivated murders.

In conclusion therefore, it may be affirmed that the evidence of this investigation, in conjunction with that of other workers, strongly suggests that, compared with normal controls a significantly higher proportion of psychiatrically ill murderers have abnormal EEGs. It also demonstrates that the incidence of EEG dysfunction is higher among the psychotic than the non-psychotic group.

Another interesting observation is the association of various electrical patterns with some of the clinical attributes of this population. Focal EEG disturbance was associated with the diagnosis of schizophrenia, history of psychiatric illness among siblings and victims below the age of sixteen; paroxysmal abnormalities displayed a strong relationship with the diagnosis/
diagnosis of psychotic depression; diffuse EEG disturbance and a history of head injury were associated—generalised EEG abnormality showed association with a history of neurotic traits in childhood and a family history of neurosis.

Four factors, namely, diagnosis of schizophrenia, female victims, victims related to the murderer and married murderers demonstrated a positive relationship with severity of EEG disturbance.

A statistically significant correlation was found between a normal EEG and a past history of multiple convictions for petty crimes. Normal EEGs were also related to factors such as family history of alcoholism, sex murder and the victim being below the age of sixteen.

Probably the most noteworthy feature of the present study is that none of the patients included in the sample were suffering from epilepsy. It is striking in view of the number of epileptics appearing in other series of a similar nature. The present study re-emphasises the point which has been stressed by several investigators: murder and epilepsy are not necessarily associated.

In the clinical histories of 32 patients the most striking feature is that a majority had a history of previous psychiatric illness. This finding seems to indicate a failure on the part of psychiatrists to recognise the potential murderer. Another finding of equal importance is the fact that more than half/
half of the patients had a past history of single or multiple convictions for some sort of offence. Thus it is obvious that the presence of criminal behaviour in a psychiatrically ill patient should be considered as a 'dangerous symptom' and cognisance taken of it considering future management.

Finally, the value of electroencephalography in establishing criminal irresponsibility is worth commenting. In our study the severity of EEG disturbance seems to be directly proportional to the degree of psychiatric disturbance. The criteria of the McNaughton formula are easily fulfilled in severely disturbed patients on clinical grounds. Nevertheless, electroencephalography could be used as supportive evidence for the existence of brain dysfunction in this class of patient. However, in less severe cases EEG has very little to contribute in the way of establishing criminal irresponsibility.

It must be re-emphasised that a substantial number of psychotic murderers have abnormal EEGs. This finding strongly supports the observation of other investigators. Nevertheless, until there are studies of other samples with appropriate controls and follow up data, the clinical and forensic importance cannot be established. Needless to say extensive work must be done in this neglected field. Such investigations might be productive in evolving a cohesive and empirical system for recognition of psychotics who are potential killers and thus save many valuable lives.
SUMMARY

On surveying the literature it was found that the first study devoted to clinical and EEG investigations of murderers was reported by Stafford-Clark and Taylor in 1945. Later, Hill and Pond extended the same study and published their findings in 1952. The last paper on this subject appeared in 1955. In view of the scanty literature in this area the present investigations were undertaken.

The present study includes 32 murderers. In each case there were sufficient legal grounds for the patient to be regarded as mentally ill at the time of committing murder. All subjects were inmates of the State Mental Hospital.

18 patients were diagnosed as psychotic and the remaining 14 were considered as non-psychotic. The incidence of EEG abnormality in the former group was 77.8% and in the latter 50%. The overall incidence of EEG abnormality was 65.5%, which, compared with 15% in the normal control group, is statistically significant. This finding is in agreement with other workers indicating that a significantly higher proportion of psychiatrically ill murderers have abnormal EEGs.

The EEG records were classified according to the type of abnormality as diffuse, generalized, paroxysmal or focal disturbance and the percentage was 43%, 23.4%, 19.3% and 14.3%, respectively. The age of the patient showed no relationship with presence or absence of EEG dysfunction. Similarly, a comparison of/
of dominant frequencies in control and experimental groups failed to reveal a significant difference. However, 85% of the records in the experimental group demonstrated a characteristic pattern of electrical activity.

Clinical information was obtained from the case notes of the patients and the records of psychiatric evidence produced at the time of trial. Forty-four items were thus selected for Kendall's tau analysis. The apparent precipitating cause of murder was considered as the motive of murder. Half the patients committed motiveless murders. In 7 cases (21.8%) there was a clear evidence of provocation. In 6 cases (18.8%) murder was preceded by sexual activity of some kind. Motiveless murders demonstrated a significant relationship with abnormal EEGs.

A statistically significant correlation was found between normal EEGs, past history of multiple convictions for petty crimes, family history of alcoholism, sex murder and the victim being below the age of sixteen. Focal EEG abnormality was associated with the diagnosis of schizophrenia and a history of psychiatric illness among the siblings. Paroxysmal abnormalities, were related to the diagnosis of psychotic depression. Diffuse EEG disturbance and history of head injury were associated. The generalized electrical disturbance was related to neurotic traits in childhood and a family history of neurosis.

EEGs were rated according to the severity of disturbance. The degree of severity of dysfunction was positively correlated with four factors viz: diagnosis of schizophrenia, female victims, victims/
victims related to the murderer and married murderers. None of the patients included in the study were suffering from epilepsy. This finding re-emphasises the point which has been stressed by several investigators that murder and epilepsy are not necessarily associated.

78% of the patients had a history of psychiatric treatment prior to committing the capital offence. More than half the patients had previous convictions for some sort of offence. It is suggested that the combination of psychiatric illness and criminal behaviour are ominous signs and such patients should be treated with caution.

The incidence of EEG abnormality among psychotic murderers compared with the rate of EEG abnormality in psychotic patients reported by other workers indicates that the incidence is higher in the former. It is suggested that additional work must be done with similar aims with appropriate controls in order to elucidate the significance of these findings.
REFERENCES


DESCRIPTION OF EEGS AND SOME FINDINGS FROM CLINICAL DATA.

Case No: 1 Age 39, diagnosed as a psychopathic personality with sexual perversions. The patient had past history of episodes of loss of memory. The EEGs before admission showed no abnormality and the patient has a positive family history for psychosis. Since his admission he had behaved reasonably well and had shown no symptoms of a psychotic illness.

EEG: There is a poorly developed sparse alpha rhythm with frequency of 10 c.p.s. seen symmetrically in posterior regions which shows little response to eye opening. It is mixed with a considerable amount of fast activity.

Conclusion: A low voltage record but within normal limits.

Case No: 2 Age 47, diagnosed as a schizophrenic at the age of 24. He killed when he was 35. The murder was clearly a consequence of his delusions. One year after his admission he had a single grand mal seizure. He had no anti-convulsant medication and had no fits since.

EEG: There is a well developed reactive alpha dominant from 9-10 c.p.s. and amplitude of 30-40 µv. Fast activity is seen in anterior regions which is, at times, interrupted by an extension of the dominant rhythm from the posterior regions. Hyperventilation did not produce a significant change.

Conclusion: Normal.

Case No: 3 Age 22, diagnosed as simple schizophrenia at the age of 16 when he committed murder. His present mental state is characterised by a marked flattening of the affect.

EEG: There is a well developed reactive alpha rhythm with a frequency of 10 c.p.s. and an amplitude up to 50 µv. This is mixed with a considerable amount of theta activity. The amplitude of this slower rhythm at times exceeds that of the dominant rhythm. Slow activity became more marked after hyperventilation, particularly on the temporal lobes.
Conclusion: Slow activity is slightly in excess of what would be expected in a patient of this age; however, this record is within normal limits. (Grade II).

Case No: 4 Age 45, a borderline mentally deficient patient who had a history of maladjustment throughout his life. He committed multiple murders.

EEG: There is a poorly developed sparse alpha rhythm with a frequency of 11 c.p.s. and amplitude seldom exceeding 20 µv. There is very little response to eye opening or overbreathing.

Conclusion: A flat record but within normal limits.

Case No: 5 Age 51, a high grade mental defective with antisocial traits and pathological sexuality. Committed the offence at the age of 22. The patient had a minor head injury at the age of 11, following which he had convulsions for a couple of years but recovered without treatment.

EEG: There is a poorly developed reactive alpha rhythm with a frequency of 12 c.p.s. and an appreciable amount of fast activity having the same voltage as the dominant frequency. After hyperventilation, the amplitude is increased and becomes more pronounced.

Conclusion: Normal.

Case No: 6 Age 47, diagnosed as an inadequate psychopath. At the age of 36, when he was admitted, his EEG showed maturation defect.

EEG: There is a well developed partially reactive alpha rhythm dominant from 8-9 c.p.s. and amplitude up to 60 µv. The dominant rhythm is recorded symmetrically in posterior regions and also extends forwards into anterior regions. Some 3-6 sec. activity in the post-central areas is seen, which is mostly interposed between the runs of alpha. There is some increase in the amplitude of the dominant and slower rhythms after hyperventilation.

Conclusion: Normal.

Case No. 7 Age 55, a psychopathic personality with a past history of episodes of loss of memory. He has had fainting attacks but apparently during these spells consciousness was not lost. He developed an illness characterised by paranoid delusions at the age of 41.
EEG: There is a well developed reactive alpha rhythm seen symmetrically in posterior regions with a frequency of 9 c.p.s. and amplitude up to 40 μv. Fast activity is seen bilaterally in the anterior regions. Some slow activity, interposed between the runs of alpha, is particularly marked on temporal lobes. Occasional runs of theta activity are seen in the central regions. Overbreathing did not produce a significant change.

Conclusion: Normal.

Case No. 8: Age 24, diagnosed as psychopathic personality with sexual deviation and admitted at the age of 21. After admission he has demonstrated aggressive outbursts on several occasions.

EEG: There is a well developed reactive alpha rhythm dominant from 8-9 c.p.s. and amplitude up to 40 μv. Fast activity is recorded from the pre-central regions; hyperventilation did not produce a significant change.

Conclusion: Normal.

Case No. 9: Age 25, with onset of a schizophrenic illness at the age of 20. His illness has been characterised by delusions and hallucinations.

EEG: There is a well developed reactive alpha rhythm dominant from 8-9 c.p.s. and amplitude up to 50 μv. Some underlying slow activity is recorded from posterior regions. Hyperventilation did not result in a significant change.

Conclusion: Normal.

Case No. 10: Age 37, diagnosed as schizophrenic. His first psychiatric illness, at the age of 17, was characterised by aggression. He sustained a head injury after committing the crime which resulted in damage to the right frontal lobe. After admission he has demonstrated episodes of aggressive behaviour. He committed a murder as a result of provocation and was unable to control his temper.

EEG: There is a well developed reactive alpha rhythm dominant from 8-9 c.p.s. with an amplitude up to 50-60 μv. An appreciable amount
of interposed slow activity is seen which is accentuated on hyperventilation particularly over the temporal lobes. In the right fronto-parital some irregular fast and slow activity is seen consisting of waves which are fairly sharp and show consistent phase reversal at electrode F7. Fig. 13.

Conclusion: There is a focal involvement of the right fronto-parital zone and an excess of slow activity over temporal lobes. (Grade IV).

Case No: 11 A 32 year old schizophrenic who had a history of several admissions for psychotic illnesses which included three attempted suicides. Family history is loaded with morbidity. He killed as result of provocation.

EEG: There is a well developed partially reactive alpha rhythm dominant from 9-10 c.p.s. with an amplitude up to 50 µv. Some interposed slow activity is seen. Anterior regions show low voltage fast activity (period 300-400 m.sec., amplitude 10 µv.) are seen with occasional phase reversal at F7. Hyperventilation did not produce a significant change. (Fig. 14.)

Conclusion: There is a localised abnormality which appears to arise from the left fronto-temporal zone. (Grade IV).

Case No: 12 Age 37, possibly had a birth injury but definitely had a difficult childhood. All his life he has been impulsive and aggressive. He has had three admissions for psychotic illnesses and three previous convictions for sexual assaults. He committed a murder with an apparently sexual motive.

EEG: There is a fairly well developed partially reactive alpha rhythm which at times extends forwards to anterior regions. A considerable amount of slow activity is seen in all areas. Some brief episodes of bilaterally synchronous high amplitude slow waves (amplitude 150 µv, period 500-600 m.sec.) lasting \( \frac{1}{2} \) to 1 sec. are also seen. Hyperventilation did not produce a significant change.

Conclusion: A mild paroxysmal abnormality. (Grade III).
Case No: 13: A boy aged 18 with no previous convictions or psychiatric history. He was considered to be shy and sensitive. One of his parents had suffered from a depressive illness. This patient compelled by circumstance, killed his victim but showed no remorse or emotional response. Since his admission, apart from flattening of the affect, there are no apparent signs of psychotic illness.

**EEG:** A fairly well developed reactive alpha rhythm dominant from 9-10 c.p.s. and an amplitude up to 50 µv. There is a slight asymmetry which becomes prominent on hyperventilation. Irregular waves with a period of 250-300 m.sec. and of higher voltage than background rhythmic activity are seen on both hemispheres but are more prominent on the left side.

**Conclusion:** Mild diffuse abnormality which is slightly more pronounced on the left side, (Grade III).

Case No. 14: Age 42, episodes of psychotic depression. He was treated with ECT at the age of 32. He committed multiple murders as a result of delusion characteristic of psychotic depression.

**EEG:** The background rhythmic activity consists of well developed, reactive alpha and some slower activity in theta range. There are frequent bursts of bilateral high amplitude slow waves (period 400-500 m.sec. voltage 150-200 µv.) lasting 1 to 2 sec. The paroxysms become more marked on hyperventilation. (Fig. 15)

**Conclusion:** A paroxysmal abnormality. (Grade IV).

Case No: 15 Age 58, with schizophrenic illness of nearly 25 years duration. He was leucotomised at the age of 40. Chronic schizophrenia in this patient has resulted in obvious signs of personality disorganisation.

**EEG:** A well developed partially reactive alpha dominant from 9-10 c.p.s. and amplitude up to 50 µv. Occasionally, slower rhythms appear in anterior regions. Runs of irregular slow waves at times interrupt the background activity.

**Conclusion:** A mild diffuse abnormality; slower rhythms in the anterior regions are probably a sequel of his leucotomy. (Grade III).
Case No. 16: Patient aged 50, diagnosed as psychopathic personality with chronic alcoholism. No definite history of head injury.

EEG: The record shows a well developed reactive alpha dominant from 8-9 c.p.s. and amplitude seldom exceeding 60 μv., at times this activity extends further forward into anterior regions. Occasionally slower rhythm also appears on the anterior regions. The tracings are interrupted with runs of irregular slow waves with an amplitude up to 80 μv. Hyperventilation accentuated the slower rhythms particularly on temporal lobes. (Fig. 16.)

Conclusion: This record shows a mild diffuse abnormality. (Grade III).

Case No. 17: Age 58, sustained a moderately severe head injury at the age of 13. His first admission to a psychiatric hospital was at the age of 26 for a schizophrenic illness.

EEG: There is a well developed reactive alpha rhythm dominant from 9-10 c.p.s. The characteristic feature of the record is the presence of irregular theta activity particularly in the posterior regions and it has a tendency to appear in runs. (Fig. 17.)

Conclusion: Diffuse abnormality. (Grade III).

Case No. 18: Patient age 58, had a long history of antisocial and psychopathic behaviour. He drank excessively and was treated for chronic alcoholism. His crime was characterised by extreme violence and he claimed to have no memory of the events concerning the murder.

EEG: The tracings showed a well developed alpha rhythm dominant at 9 c.p.s. and amplitude up to 40 μv. The record demonstrated an excess of slow activity of the same voltage as the dominant rhythm which was more pronounced on both temporal lobes. Hyperventilation increased the voltage of slower rhythms.

Conclusion: There is an excess of slow activity which is more than would be expected in a patient of this age. However, this record is within the normal limits. (Grade II).
Case No. 19: The patient, aged 32, has been diagnosed as a schizophrenic since the age of 26. The illness has been characterised by delusions and hallucinations and a marked flattening of the affect.

EEG: There is a polyrhythmic background activity which shows a considerable amount of sharpish looking waves. On hyperventilation, some high amplitude spikes became apparent on both posterior temporal zones. At times generalised bursts of high amplitude sharp waves are seen in all leads.

Conclusion: Generalised abnormality. (Grade III).

Case No. 20: This patient, aged 39, had a mild head injury when 27. He was treated with ECT for a schizophrenic illness at the age of 34.

EEG: A rather poorly developed partially reactive alpha rhythm with a frequency of 11 c.p.s. and amplitude seldom exceeding 30 μV. Runs of irregular slow waves appear diffusely in all leads. Irregular slow waves (period 300 m.sec to 400 m.sec. amplitude 100 μV.) are seen on the left posterior temporal zone but adjacent leads, show no definite reversed polarity.

Conclusion: In addition to diffuse abnormality this record also shows a possible focal involvement of the left posterior temporal zone. (Grade IV).

Case No. 21: Age 35, a first psychotic episode occurred at the age of 24 which was diagnosed as schizophrenia and treated with ECT.

EEG: This patient's record shows a well developed reactive alpha dominant from 8-9 c.p.s. and amplitude up to 50 μV. This is mixed with a considerable amount of slow activity. Fast activity is seen in anterior regions. Occasional runs of slow waves appear diffusely in all areas. Hyperventilation did not result in a significant change.

Conclusion: There are only minor irregularities; however this record is within normal limits. (Grade II).
Case No. 22: Age 32, was diagnosed as schizophrenic when 30. No other previous history of psychotic illness is known. Apparently he was well adjusted till the onset of psychotic illness. There are no neurological signs.

EEG: In the posterior regions the record is dominated with regular 3 c.p.s. activity with an amplitude up to 50 µv. This activity is completely blocked and alpha appears when the eyes are open showing a paradoxical response. 3 per second activity is most marked in parieto-occipital, temporo-occipital and temporal zones. Fast activity is mostly seen superimposed on the slower rhythms. Hyperventilation did not produce a significant change. (Fig. 18)

Conclusion: Severe generalised abnormality. (Grade V).

Case No. 23: Age 31, since the age of 26 this patient has had several episodes of psychotic depression. Family history is positive for psychotic illnesses.

EEG: Fairly well developed reactive alpha rhythm dominant from 10-11 c.p.s. is seen symmetrically on both sides of the head. In addition to fast activity, runs of theta activity also appear in anterior regions. The record is frequently interrupted with generalised runs of high voltage theta activity which becomes more pronounced on hyperventilation. Irregular slow waves with a period up to 300 m.secs. and amplitude up to 80 µv. are occasionally seen on the left temporal lobe.

Conclusion: Paroxysmal abnormality. (Grade III).

Case No. 24: Age 27, he sustained a head injury when 21. The patient has always been aggressive and claims he cannot control his temper.

EEG: The present recording shows a well developed reactive alpha rhythm dominant from 8-9 c.p.s. and amplitude up to 50 µv. This activity extends forward to the anterior regions. The record is frequently interrupted by bursts of high amplitude (up to 150 µv.) slow (2-3c.p.s.) activity which is more pronounced anteriorly. Bursts of slow activity become more frequent after hyperventilation.

Conclusion: There is a paroxysmal abnormality. (Grade IV).
Case No. 25: Age 47, diagnosed as paranoid schizophrenic. There is a past history of excessive alcohol intake. He had 6 previous psychiatric admissions. His first illness was at 35. There have been periods after admission when he has been aggressive and unpredictable.

EEG: A well developed reactive alpha (9-10 c.p.s., 40 μv.) is seen symmetrically on both sides of the head. Fast activity is recorded in anterior regions. Only after hyperventilation do irregular slow waves (3-4 c.p.s., 80 μv.) appear in the left occipito-temporal region and occasionally phase reversal is observed.

Conclusion: Mild localised slow wave abnormality involving posterior temporal region. (Grade III).

Case No. 26: Age 43, diagnosed - psychotic depression. He had marked obsessional traits prior to his psychotic illness. He was severely depressed for several years preceding his crime.

EEG: Poorly developed partially reactive symmetrical alpha which is mixed with a considerable amount of theta activity seldom exceeding in voltage that of the dominant rhythm. Theta also appears diffusely in runs in all channels. Some fast activity is present in anterior regions but it is interrupted by slower rhythms. No significant change was noted after hyperventilation.

Conclusion: Diffuse dysfunction. (Grade III).

Case No. 27: Age 34, certified mentally deficient at the age of 15. There is a family history of behaviour disorders. He is reputed always to be destructive and aggressive.

EEG: Record is dominated with low amplitude theta (5-6 c.p.s., 20 μv.). Some fast activity is seen in all areas. Anterior regions also show theta activity predominantly. On at least two occasions the record was interrupted with generalised bursts of slow waves (period 500-600 m.sec., amplitude 100 μv.) lasting about a half second.

Conclusion: There is generalised abnormality. (Grade III).
Case No. 28: Age 56, diagnosis: schizophrenia. First admitted for the treatment of a schizophrenic illness when 20 years of age. Leucotomised at the age of 51.

EEG: The record is dominated by 5-6 c.p.s. activity with an amplitude up to 40 μv. In both frontal regions delta waves (1-2 c.p.s.), with an amplitude up to 80 μv., are seen. The record is grossly altered after one minute of hyperventilation and only delta-theta polyrhythmia is seen. (Fig. 20.)

Conclusion: There is a generalised abnormality. Frontal abnormality is probably a sequel of his leucotomy. (Grade IV).

Case No. 29: Age 27, diagnosis: schizophrenia. He was a poor scholar and has always been considered aggressive. He was first admitted for a schizophrenic illness at the age of 20 and treated with ECT and insulin coma.

EEG: Alpha-theta polyrhythmia constitutes the background activity. Runs of theta activity mixed with fast activity are seen anteriorly. At times generalised bursts of 4 per second high amplitude waves lasting ½ - 1 second also appear. After hyperventilation runs of irregular slow waves appear on both temporal lobes.

Conclusion: Generalised abnormality. (Grade IV).

Case No. 30: Age 50, diagnosis: psychopathic personality. He was a poor scholar and had an unsatisfactory work record. He had several previous convictions and was considered aggressive and violent. He sustained a minor head injury at the age of 8.

EEG: A poorly developed bilaterally symmetrical alpha is dominant from 9-10 c.p.s. and amplitude seldom exceeding 30 μv. Runs of 6 c.p.s. activity of the same voltage as the dominant rhythm appear diffusely in all leads with 3-5 c.p.s. activity appearing in runs is seen bifrontally. Hyperventilation accentuated the slower rhythms.

Conclusion: Diffuse abnormality. (Grade III).

Case No. 31: Age 48, diagnosis: psychopathic personality. His alcoholic intake was excessive. All his life he has been poorly adjusted and aggressive. He twice attempted suicide. His family history is positive for psychiatric illness.
EEG: A fairly well developed alpha rhythm is dominant from 8-9 c.p.s. and amplitude up to 40 µv. Alpha activity extends forwards into the anterior region. Runs of 4-5 c.p.s. with amplitude up to 40 µv. appears diffusely in all leads. Hyperventilation resulted in continuous irregular slow activity on both temporal lobes.

Conclusion: Diffuse abnormality. (Grade III).

Case No. 32: Age 48, a diagnosis of psychopathic personality has been made. He was treated for a depressive illness at 26. He also attempted suicide with carbon monoxide; no family history of mental illness.

EEG: There is a polyrhythmic background activity with an amplitude seldom exceeding 30 µv. Runs of irregular slow activity appear diffusely, at times simultaneously, in all regions. Slow rhythms are seen on the temporal lobes which become even more pronounced after hyperventilation.

Conclusion: Diffuse abnormality. (Grade III).
APPENDIX II

Instructions to the Technicians

The success of this research project depends upon the quality of the records. Therefore, it is imperative that every effort should be made to obtain records of the highest possible technical standard. The following are some instructions to assist you achieve this object and also to standardise the technique:

1) All recording will be done lying down in semi-darkened surroundings in your EEG laboratory. Each patient should be recorded on two different occasions, once during morning; and have a second record after lunch.

2) In order to make this project as objective as possible it is essential that interpreters should not know the name of the patient. Every record whether experimental or control will be represented by a different code number. You should keep a record of the code numbers.

3) The following information must be written on every record:

   i) Code number
   ii) Date and time of recording
   iii) Time of patient's last substantial meal
   iv) Handedness of patient
   v) Note of time Constant, filters and gain
   vi) Any drugs given in past week (if possible all drugs should be stopped for a week before recording)
   vii) Date of any ECT administered in the past six months
   viii) Technician's personal observation of the patient's attitude and state of consciousness

4) Controls for Calibration must be - Gains 1 cm per 100 uv
   Time Constant 0.3 sec.
   H.F. position No. 2

5) Electrodes should be chlorided once a month. Electrodes must be placed according to the "10-20 system". You may place electrodes by eye but must confirm with a tape to within 2 mm before connecting them to the machine. Resistance must be checked as a routine and ensured that it is below 5 Kohms.
6) **1st Record**

Calibrate machine before and after each record; and before commencing the actual record run one montage through all Channels

<table>
<thead>
<tr>
<th>Pattern</th>
<th>Duration</th>
<th>E/O &amp; E/C</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>5 - 8 mins.</td>
<td>E/O &amp; E/C</td>
</tr>
<tr>
<td>II</td>
<td>5 - 8 mins.</td>
<td>E/O &amp; E/C</td>
</tr>
<tr>
<td>III</td>
<td>5 - 8 mins.</td>
<td>E/O &amp; E/C</td>
</tr>
<tr>
<td>V</td>
<td>5 - 8 mins.</td>
<td>E/O &amp; E/C</td>
</tr>
</tbody>
</table>

3 mins. hyperventilation
5 mins. subsequent record
Run BNI analyser throughout pattern V

(Total recording time 28-37 minutes)

**2nd Record**

Repeat as Record I until Pattern II

<table>
<thead>
<tr>
<th>Pattern</th>
<th>Duration</th>
<th>E/O &amp; E/C</th>
</tr>
</thead>
<tbody>
<tr>
<td>III</td>
<td>5 mins.</td>
<td>E/O &amp; E/C</td>
</tr>
<tr>
<td>V</td>
<td>5 mins.</td>
<td>E/O &amp; E/C</td>
</tr>
<tr>
<td>VI</td>
<td>2 mins.</td>
<td>Eyes closed throughout, followed by 2 mins. stroboscopic activation with eyes closed throughout. Build up slowly to 30 F.P.S. Return to 10 F.P.S. and again build up to 20 F.P.S. Concentrate at frequency of resting alpha activity but employ any frequency which proves evocative. Use B.N.I. analyser throughout</td>
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</table>

(Total recording time approximately 35 minutes)

7) It is crucial that you should make a note of all the artefacts resulting from movements etc. on the record.
APPENDIX III

The following scheme was used for evaluation of the EEGs.

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<thead>
<tr>
<th>CODE NO:</th>
<th>R:</th>
<th>L:</th>
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<tbody>
<tr>
<td>Dominant frequency:</td>
<td>R:</td>
<td>L:</td>
</tr>
<tr>
<td>Amplitude of dominant frequency:</td>
<td>R:</td>
<td>L:</td>
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<th>L.V.A.</th>
<th>S.A.F.</th>
<th>Alpha Factor</th>
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<td>VIII</td>
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<td>Σ</td>
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<td>Mean</td>
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<th>S.A.F.</th>
<th>Alpha Factor</th>
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<table>
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<th>Hyperventilation; Lateral</th>
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<th>S.A.F.</th>
<th>Alpha Factor</th>
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<tr>
<td>Mean</td>
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</tbody>
</table>
Quality of Alpha:

i. Blocking (Reactive/Poor/Non-reactive)

ii. Well sustained/Poorly sustained

iii. Whether seen in anterior region in addition to usual sites

iv. Asymmetry

**REACTION TO PHOTIC STIMULATION**

1. Is there any significant change?

2. Response of abnormalities already existing in the resting record.

3. Occurrence of any abnormal responses which were not present in the resting or hyperventilation record:

   i. Wave and spike discharges
   
   ii. Frontal spikes
   
   iii. Dysrhythmia
   
   iv. Runs of irregular slow waves
   
   v. Larval discharges
   
   vi. Focal abnormalities

Classification according to pattern of electrical activity

\[ P_1 \quad P_2 \quad P_3 \quad P_4 \quad P_5 \]

**Is this record Normal or Abnormal?**

**(TYPE OF ABNORMALITY)**

1. Paroxysmal:
   
   i. Wave & spike complexes or its variations (Resting/HV/IPS)
   
   ii. Runs of Slow Waves (Resting/HV/IPS)

2. Generalised:

3. Diffuse:

   Period and duration of slow waves
   
   Regular or irregular, unilateral or bilateral
   
   Resting/HV/IPS

4. Focal:

   Localisation
   
   Phase reversal
   
   Resting/HV/IPS

**Rating of the record on five point scale**

Rating I / II / III / IV / V

**Additional remarks**
APPENDIX IV

The following check list was used for collection of the clinical data from the case notes

<table>
<thead>
<tr>
<th>CODE</th>
<th>DATE OF ADMISSION</th>
</tr>
</thead>
<tbody>
<tr>
<td>AGE</td>
<td></td>
</tr>
<tr>
<td>DIAGNOSIS</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Birth injury</th>
<th>Yes</th>
<th>No</th>
<th>Not Known</th>
</tr>
</thead>
<tbody>
<tr>
<td>Childhood illnesses</td>
<td>Yes</td>
<td>No</td>
<td>Not Known</td>
</tr>
<tr>
<td>Illnesses associated with delirium or coma</td>
<td>Yes</td>
<td>No</td>
<td>Not Known</td>
</tr>
<tr>
<td>Febrile convulsions</td>
<td>Yes</td>
<td>No</td>
<td>Not Known</td>
</tr>
<tr>
<td>Neurotic traits</td>
<td>Yes</td>
<td>No</td>
<td>Not Known</td>
</tr>
</tbody>
</table>

SCHOOL: Age of entering Age of leaving
Educational achievement
Poor Scholar Average Above Average

OCCUPATIONS: Frequent change of jobs
Last job

SOCIAL CLASS:

MARITAL & SEX: Single Married Divorced
Marital Adjustments Sexual deviations
**ALCOHOL:**

**PREMORBID PERSONALITY**

**PREVIOUS ILLNESSES:** Psychiatric
Medical
Surgical Operations
Accidents
Yes  No  Not Known

**HEAD INJURY:** Age when sustained
How long unconscious

**EPILEPSY:** Yes  No
Age of Onset
Clinical description: Grand mal  Petit mal  Psychomotor  Focal
Aura
Post ictal Confusion

**FAMILY HISTORY**
(1st degree relatives only)
Specify relationship

<table>
<thead>
<tr>
<th></th>
<th>Yes</th>
<th>No</th>
<th>Not Known</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epilepsy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mental deficiency</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neurosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychopathy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Personality Disorder</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcoholism</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Well adjusted</td>
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<td></td>
<td></td>
</tr>
</tbody>
</table>
DRUG THERAPY & E.C.T.: Before admission
               After admission
               Present

EXAMINATION: Psychiatric
             Neurological
             Physical
             I.Q.
             Behaviour after admission
             Degree of personality deterioration if psychotic

CRIMINAL RECORD: Previous convictions
                 Nature of previous offences
                 Crimes against property
                 Violence
                 Sex
                 Age at first conviction

CAPITAL OFFENCE: Single or multiple murders
                 Motive of killing: Motivated & Premeditated
                 Motiveless
                 Sex murder
                 Provoked or Unprovoked
                 Victim: Relative
                 Acquaintance
                 Stranger
                 Victim below age of 16
                 Victim above 16
                 Female/Male

METHOD OF KILLING:

DEGREE OF VIOLENCE:

SUBSEQUENT AMNESIA:

ATTEMPT TO ESCAPE:
Was question of abnormal E.E.G. brought up during trial?

CONVICTION: Insane before trial or upon arraignment.
Guilty but Insane.
Certified insane after trial.
Convicted of manslaughter on the grounds of diminished responsibility.

ADDITIONAL COMMENTS: