THE ELECTROENCEPHALOGRAM IN PSYCHIATRY

L. G. KILOH, B.Sc., M.D., M.R.C.P., D.P.M.

*Reader, Department of Psychological Medicine, University of Durham; Hon. Consultant, Royal Victoria Infirmary, Newcastle upon Tyne, and Newcastle upon Tyne General Hospital**

At present the interpretation of electroencephalograms is necessarily an empirical process. Whenever interpretation is attempted, a prediction is made which is entirely a matter of probability. It is rarely possible therefore to state with confidence that a particular pattern is diagnostic of any specific clinical entity. Seldom is it advisable to claim more than that the EEG findings support or fail to support the diagnosis suggested by the clinician. In many conditions—and this is particularly true of psychiatric disorders—the incidence of abnormal records is relatively low and the finding of a normal EEG does nothing to exclude the possibility of such an illness. Only in a few conditions, such as cerebral abscess or subacute progressive encephalitis, is the incidence of abnormality so high that the finding of a normal EEG virtually rules out its existence. The request made by so many clinicians to the electroencephalographer, to exclude a certain condition, is on the whole an unreasonable one.

Apart from the fact that normal EEG findings are frequent in the presence of indisputable disease, the repertoire of the EEG is very limited so that specific patterns of abnormality are consequently rare. The same appearances are commonly produced by a variety of conditions of widely differing ætiology, and it is a dangerous practice to attempt to correlate EEG abnormalities with specific pathologies. In psychiatry the position is made even more difficult by our limited knowledge of ætiological factors and by lack of agreement on such matters as classification.

Personality and its Disorders.

There is general agreement from a number of surveys that between 10 and 15% of the population at large have 'abnormal' EEGs—abnormal, that is, in a statistical sense. The abnormality consists of an excess of theta activity (4 c/s. up to but excluding 8 c/s.) sometimes with delta components (less than 4 c/s.) often best seen in the temporal areas. Such an EEG is usually described as 'immature', but it must be emphasized that

this does not necessarily mean that the patient is immature in any other way (Fig. 1). It does happen that the more carefully subjects are selected for stability, the smaller the incidence of such abnormalities. Thus Williams (1941) found that this pattern of EEG abnormality was present in only 5% of flying personnel as compared with 10% of other servicemen. The incidence is higher (25%) in mixed groups of psychoneurotics and highest of all (65 to 83%) in aggressive psychopaths.

Hill (1952) found in a series of 194 non-epileptic psychopaths that in addition to an excess of bilateral theta activity, 14% of cases showed focal activity in the posterior temporal areas (Fig. 2). Although usually bilateral and symmetrical, these were sometimes more prominent or limited to one side, commonly the right. Such slow activity, whether focal or not, is usually intermixed with normal cortical rhythms, and usually more responsive to eye opening and other physiological stimuli than is the not dissimilar activity sometimes produced by certain pathological cerebral states.

These so-called immature patterns occur normally in children (Fig. 1), but once again when one selects children with behaviour disorders, one commonly finds that the EEG is relatively immature in 50 to 60% of cases, containing slow activity of a frequency or amplitude characteristic of a younger age group.

In patients experiencing nocturnal enuresis, the EEG investigations of Ditman and Blinn (1954-55) have shown that the patients fall into two groups: those who micturate in a state of deep sleep and those whose EEGs show a waking pattern when micturition occurs. The former is usual in children, the latter in adolescents and adults. These findings suggest that the members of the latter group are awake but in a state of dissociation when they micturate, for they remain unresponsive to stimuli and unaware that they have done so.

Psychoneuroses

In patients suffering from *anxiety states* there tends to be less alpha activity and an increase in the amount of faster rhythms. Similar changes

* Present appointment: Professor of Psychiatry, University of New South Wales, Sydney, Australia.

FIG. 1.—(a) Normal EEG. Male aged 36 years. Alpha rhythm at $8\frac{1}{2}$ c/s. (b) Immature EEG. Female aged 20 years. 8 c/s. alpha rhythm intermixed with irregular theta components. (c) Normal EEG. Boy aged 10 years. 8 c/s. alpha rhythm intermixed with irregular theta components.

FIG. 2.—Aggressive psychopath. Male aged 18 years. Occasional focal delta waves in posterior temporal areas on a background of irregular theta and alpha frequencies.

occur when anxiety is induced in normal subjects and they appear to be related to preoccupation with anxious thoughts, for a well-marked alpha rhythm is a resting phenomenon and is easily lost—or blocked—by mental activity. A good alpha rhythm can often be elicited in anxious patients by sedation (Fig. 3). Apart from the higher incidence of immature patterns, no particular EEG features occur in patients with *hysteria* or *obsessional states*.

In 1952 Gibbs and Gibbs described the occurrence of runs of 14 and 6 c/s. positive spikes

during sleep in patients suffering from attacks which they claimed to have a thalamic or hypothalamic origin. Other observers have noted a high incidence of headaches, neurotic symptoms, behaviour disorders and syncopal attacks in patients showing this phenomenon (Refsum, Presthus, Skulstad and Östensjö, 1960), but others have failed to confirm these findings (Walter, Colbert, Koegler, Palmer and Bond, 1960). The existence of the 14 and 6 c/s. positive spike phenomenon must be accepted but its precise significance is still very much in doubt.

FIG. 3.—Anxiety state. Female aged 35 years. (a) Traces of alpha rhythm posteriorly with muscle artefact anteriorly. (b) Following sedation: mixed alpha frequencies posteriorly with low voltage beta runs anteriorly.

FIG. 4.—(a) Sedation threshold. Normal subject. Ordinate: mean amplitude of frontal fast activity in μ V. Abscissa: integrated amount of amylobarbitone sodium given intravenously in mg./kg. body weight. Arrow indicates inflection point corresponding to point of slurred speech. (From Shagass, 1954.) (By courtesy of the Editor, *Electroencephalography and Clinical Neurophysiology*.) (b) Percentage distribution of sedation thresholds in mg./kg. body weight for psychotic and neurotic depression groups. Mean sedation threshold for psychotics: 2.23 mg./kg. Mean sedation threshold for neurotics: 3.99 mg./kg. (From Nymgaard, 1959.) (By courtesy of the Editor, *Archives of General Psychiatry*.)

Manic-depressive Illness

In manic-depressive illness about 80% of patients have normal EEGs. Not surprisingly, records similar to those seen in patients with anxiety states occur frequently, containing little alpha rhythm but an excess of fast activity. There is a tendency for the alpha frequency to be

relatively high in cases of mania and low in cases of depression.

The sedation threshold test (Shagass, 1954) provides results which have great interest though the practical value of the procedure is limited. When 0.5 mg./kg. body weight amylobarbitone sodium is given intravenously every 40 seconds

FIG. 5.—Catatonic schizophrenia. Male aged 36 years. Burst of theta activity with low amplitude spikes posteriorly on a background of alpha rhythm at $9\frac{1}{2}$ c/s. and random theta activity.

the resulting clouding of consciousness and slurring of speech are accompanied by the appearance of 15 to 30 c/s. activity best seen in the frontal areas. The amount of this fast activity may be measured by means of a frequency analyser. When this is plotted against the quantity of amylobarbitone sodium given, a curve is obtained showing an inflection point which approximates in time to the onset of slurred speech (Fig. 4a). The total amount of amylobarbitone sodium in mg./kg. body weight given up to this inflection point is an index of the sedation threshold. It is unrelated to sex or age. Amongst neurotic patients the value is correlated with manifest anxiety, and Shagass and Jones (1958) found that the mean value in controls was 3.09 mg./kg., in hysterics 2.79 mg./kg., in anxiety hysteria 3.91 mg./kg., in obsessive compulsive states 4.42 mg./kg., in neurotic depression 4.78 mg./kg., and in anxiety states 5.27 mg./kg. A striking difference was noted between cases of neurotic and endogenous depression, the mean level being 2.81 mg./kg. in the latter group regardless of the amount of agitation present (Fig. 4b). Some authors, such as Nymgaard (1959) have confirmed these findings; others

(Ackner and Pampiglione, 1959) have failed to do so.

Schizophrenia

Many studies of the EEG in schizophrenia have been reported, but because of differing diagnostic criteria, many are not comparable. There seems no doubt that in the majority of cases the EEG is normal, and that when abnormalities do appear, they vary widely from case to case. Early reports of 'choppy' records (Davis, 1939), that is fast dominant records poor in alpha activity, have not been confirmed. 'Immature' records are no more common than in control groups (Hill, 1957) though in catatonic stupor generalised 2 to 6 c/s. activity may appear. The most striking abnormalities are those reported by Hill (1957), who noted discharges of low amplitude bilaterally synchronous slow activity, of fast spike and slow wave activity, or of multiple spikes, usually in the post central areas, in 20 to 25% of cases (Fig. 5). Goldman (1959) has claimed that following the rapid intravenous injection of 100 mg. thio-pentone sodium up to four times at intervals of two minutes, changes characteristic of schizo-

FIG. 6.—Delirium due to bronchopneumonia. Female aged 60 years. (a) Delirium severe: frontally predominant high voltage 2 c/s. runs. (b) One week later, moderately confused: bilateral delta activity evident, but of much lower amplitude.

phrenia appear. These include bilaterally synchronous theta activity with an amplitude of 100 μ V, persistent fast activity with an amplitude greater than 25 μ V and bursts of 25–100 μ V fast activity having a duration of 0.25 to 1 sec. This work has not yet been confirmed.

Clouding of Consciousness

Clouding of consciousness is a symptom shared by many conditions in which the function of the reticular activating system of the mid-brain and diencephalon becomes impaired. This may result from distortion of the blood vessels supplying these areas, suddenly and catastrophically in head injuries, or more insidiously due to raised intracranial pressure; to specific metabolic disorders resulting from certain vitamin deficiencies, uræmia, cholæmia, hypoglycæmia and anoxia; or to the less well defined metabolic disturbances which occur in the deliria associated with infections and toxic states.

When clouding of consciousness occurs, whatever its cause, the EEG shows characteristic changes. Sometimes these are associated with other abnormalities, if, for example, a focal cerebral disturbance coexists. The presence of mild degrees of clouding of consciousness may often be very difficult to establish on clinical grounds, and frequently the EEG provides confirmatory evidence of its existence. Even so, in the absence of a previous EEG to establish the usual alpha frequency, it may be impossible to declare a record abnormal, for the earliest change is a slowing of the alpha rhythm. If, for instance, this slows from 12

to 9 c/s., it will be abnormal for the individual, although it still falls within the somewhat arbitrary limits of normality—8 to 13 c/s. Retrospectively the abnormality may be detected or confirmed when the patient's clinical state and EEG have returned to their premorbid states. By the time confusion is evident clinically, the EEG as a rule is theta dominant, and in its distribution and response to eye opening and closing, this theta activity often behaves like the alpha rhythm. With greater impairment of consciousness, activity at lower frequencies becomes more prominent and less responsive. As coma ensues, delta activity becomes dominant, and the record is made up largely of 1 to 3 c/s. activity. This is generalized though often frontally predominant, bilaterally synchronous and frequently tends to wax and wane (Fig. 6).

Such EEG findings characterise the *acute meningitides* and *encephalitides*, the *encephalopathies* associated with *heavy metal poisoning*, *hypertension*, *acute porphyria*, *uræmia*, *anoxic states*, severe degrees of *adrenal and pituitary insufficiency* and *hypoglycæmia*. In *hepatic encephalopathy* (portal systemic encephalopathy) the same EEG pattern appears when the blood ammonia level reaches 200 to 400 μ g./100 ml. Such changes may be provoked or accentuated in patients with little reserve of liver function by giving methionine or a high protein diet. In some stuporous cases, in addition to the generalized slow activity, triphasic waves become prominent. Each complex consists of two electro-negative waves separated by an electro-positive wave of higher amplitude. Some-

FIG. 7.—Raised intracranial pressure. Female aged 52 years with midline cerebellar tumour. Papilloedema marked, some clouding of consciousness: bilateral 2 c/s. runs of frontally predominant delta activity on a background of mixed alpha and theta frequencies.

times these have quite a sharp outline and may then resemble the complexes which accompany myoclonic attacks (Bickford and Butt, 1955).

Psychiatric abnormalities are frequently present in cases of *pernicious anaemia* and particularly common is the picture of a mild and reversible subacute organic reaction. EEG abnormalities—of the type characteristic of clouding of consciousness—are frequently present, though commonly these too are only mild in degree (Walton, Kiloh, Osselton and Farrall, 1954).

In *hypothyroidism* reversible psychiatric and EEG abnormalities of a similar kind are often present. Usually the EEG tends to be of low amplitude, and if hypothermia develops, it becomes almost featureless (Nieman, 1959).

Raised Intracranial Pressure

Space occupying lesions causing distortion of the upper brain stem directly or indirectly by uncil herniation, frequently give rise to widespread EEG abnormalities. The correlation between the level of intracranial pressure and these EEG abnormalities is by no means close, but the evidence that the rise in pressure is responsible in the great majority of cases is very strong. Such a procedure as Torkildsen's operation, for instance, which relieves the raised pressure without influencing the underlying lesion, can be expected to give rise to an improvement in both the level of consciousness and in the EEG abnormalities. The abnormalities have the same characteristics as those described as accompanying clouding of consciousness and are seen in purest form with infratentorial tumours.

The slow activity is commonly rhythmic and indeed almost sinusoidal (Fig. 7) but in 15% of cases is more irregular. Its distribution varies somewhat from case to case and though commonly generalized, it may be limited—for a time at any rate—to the anterior, temporal or posterior regions of the hemispheres. Moderate degrees of asymmetry are not uncommon but have no localizing value (Daly, Whelan, Bickford and MacCarty, 1953).

It has long been recognized that cerebral tumours may present initially with psychiatric disorders. The latter do not always follow one of the classical organic patterns and if the picture is that of a functional psychosis the possibility of an underlying cerebral disorder may be overlooked. It is in such cases that the EEG may score a signal success, for the finding of a localized focus of slow activity may draw attention to the presence of the unsuspected space occupying lesion.

A *subdural haematoma* is a variety of space occupying lesion and the most characteristic clinical feature is fluctuation in the level of consciousness. The EEG may prove of great value in the investigation of such a case, particularly if it presents with a somewhat obscure psychiatric picture. The EEG is almost always abnormal and in addition to generalized slow activity, there may be a slow wave focus or an area of suppression of electrical activity over the lesion (Fig. 8).

Head Injuries

Although the EEG abnormalities following head injury may be very varied in character and in

FIG. 8.—Subdural hæmatoma—right. Male aged 54 years. Right fronto-temporal slow activity with ipsilateral suppression of alpha activity.

duration due to the great variety in the type, extent and distribution of the resulting lesions, it is the 'diencephalic ischæmic syndrome' (Dott, 1960) that is responsible for both the impairment of consciousness and the accompanying generalized EEG abnormalities. These too have the same features as those associated with impaired consciousness due to other causes.

In very minor head injuries when consciousness is lost for no more than a matter of seconds, the diffuse theta and delta activity which occur, disappear within a few minutes. Following more severe injuries there may be relative suppression of electrical activity for a period, particularly in those cases whose prognosis is poor. Later slow activity often at 4 to 6 c/s. appears and as recovery ensues, this shows a progressive increase in amplitude and frequency. Slow alpha activity appears and gradually its frequency increases until it reaches its premorbid value. There is a rough correlation between the degree of abnormality of the EEG and the level of consciousness. (See Dawson, Webster and Gurdjian, 1951.)

Where localised cerebral damage has occurred, the EEG is likely to show concomitant focal slow wave activity. Spikes or sharp waves are rarely seen in the acute phase but may appear later.

The Dementias

Old age is associated with a gradual decrease in the amount, amplitude and frequency of the alpha rhythm, and there is a tendency for the amount of fast activity to be increased. There is

no increase in the amount of slow activity (Weiner and Schuster, 1956).

The EEG abnormalities in *senile dementia* are seldom striking, but a completely normal record is the exception. The remaining alpha rhythm may be slowed below 8 c/s. and this occurs against a background of low amplitude, diffuse theta activity, sometimes with delta components (Fig. 9a). At a late stage the trace tends to be flat, lacking recognizable alpha or faster frequencies. In the *simple presenile dementias* and in *Huntington's chorea* similar patterns occur.

The EEG in *arteriosclerotic dementia* also shows the same characteristics although the abnormalities may be somewhat more marked. Evidence of a localized disturbance—usually a delta focus—is usual in cases with cerebral infarction.

In *Alzheimer's disease* and to a lesser extent in *Pick's disease*, the incidence and severity of the EEG abnormalities is very much greater. Little or no alpha rhythm survives and the record consists of low to medium amplitude theta activity which is generalized and interspersed with runs of higher amplitude irregular delta activity which may be frontally predominant (Fig. 9b) (Letemendia and Pampiglione, 1958). The changes bear little relation to the severity of the dementia.

There are a number of uncommon, pathologically distinct conditions which run a subacute course and are characterised by a progressive dementia, striking EEG abnormalities and a hopeless prognosis. In *Jacob-Creutzfeldt's disease* bilateral sharp wave or sharp and slow wave discharges occur rhythmically and are accompanied

FIG. 9.—(a) Senile dementia. Male aged 82 years. Irregular theta components posteriorly, superimposed on low voltage mixed alpha and beta frequencies. (b) Alzheimer's disease. Male aged 54 years. Irregular generalized high voltage theta and delta activity.

FIG. 10.—Jakob-Creutzfeldt's disease. Female aged 63 years. Occasional bilateral spike discharges superimposed on a background of generalized slow activity.

by myoclonic or choreiform movements. (Abbott, 1959.) Initially the background record may be normal but soon it comes to be made up of theta and delta activity (Fig. 10). As the disease advances the amplitude of the sharp waves and of the background activity diminishes, and the record becomes flat.

In *subacute spongiform encephalopathy* (Nevin, McMenemey, Behrman and Jones, 1960) formerly known as Heidenhain's presenile dementia, the normal rhythms diminish and disappear, being replaced by widespread high voltage slow activity

interspersed with bilaterally synchronous polyphasic sharp wave complexes which occur repetitively and sometimes rhythmically (Fig. 11). They are usually but not invariably symmetrical and are accompanied by myoclonic jerks. In most cases these abnormalities are interspersed with periods of low voltage slow activity which may also recur regularly. The abnormalities tend to increase as the disease progresses, but in the terminal phases the amplitude of the trace diminishes and the sharp wave complexes disappear.

In *subacute progressive encephalitis* (Van Bogaert's

FIG. 11.—Subacute spongiform encephalopathy. Female aged 70 years. Bilateral sharp and slow wave complexes occurring periodically on a background of irregular slow activity.

FIG. 12.—Subacute progressive encephalitis. Female aged 7 years. Periodic generalized high voltage slow wave complexes on a low voltage background.

subacute sclerosing leucoencephalitis; subacute inclusion body encephalitis of Dawson) progressive slowing of the background rhythms occurs and dramatic high amplitude paroxysms of 1 to 3 c/s. activity appear, often with sharp components (Fig. 12). Characteristically these are bilaterally synchronous, symmetrical and generalized, but exceptions occur. Once again the complexes are accompanied by myoclonic jerkings. Progression of the disease is associated with flattening of the background rhythms and ultimately with disappearance of the complexes.

In some cases of *cerebral lipidosis* in infants, changes of a similar kind are seen (Cobb, Martin

and Pampiglione, 1952). Generalized high voltage triphasic waves sometimes with a sharp appearance are superimposed on a background of irregular $1\frac{1}{2}$ to 6 c/s. activity.

The precise significance of the periodic discharges in these various conditions is not known. It is generally believed that they are directly related to the pathological changes in the cerebral cortex which are common to this group of disorders, but clearly they cannot be attributed merely to degenerative changes. It is possible that the thalamic and brain stem reticular formations play some part in their genesis and spread. Not dissimilar periodic discharges sometimes occur

FIG. 13.—Hypsarrhythmia. Male aged 2½ years with phenylketonuria. Generalized high voltage slow activity with random spike and slow wave discharges.

after attacks of grand mal, following head injuries, in occasional cases of acute encephalitis and as the result of anoxic states following cardiac surgery.

Subnormality

As might be expected the incidence and variety of EEG abnormalities in the subcultural group of subnormal individuals is much the same as in the general population. It is to the pathological varieties that one must look for gross abnormalities.

In *congenital hydrocephalus*, *porencephaly*, *tuberous sclerosis*, *microcephaly* and *phenylketonuria*, abnormal EEGs are usual. Many cases show bilateral spike or spike and slow wave activity which has its clinical counterpart in attacks of epilepsy. In the latter two conditions the degree of abnormality of the EEG may be sufficient to be called *hypsarrhythmia* (Gibbs and Gibbs, 1952). This is a purely descriptive term and is employed when the EEG is continuously abnormal showing widespread multifocal spikes or spike and slow wave discharges against a background of diffuse irregular slow activity (Fig. 13). This pattern is rarely seen after the age of 4 years. It is often associated with 'salaam' attacks and almost invariably with progressive organic deterioration. These cases form a group of varied aetiology though natal or prenatal brain injury is the commonest factor. The EEG abnormalities usually diminish markedly with age whilst the epileptic attacks but not the mental subnormality often respond well to ACTH.

Temporal Lobe Epilepsy

The estimate that 40% of all epileptics have focal epileptogenic lesions in one or both temporal lobes may well be a conservative one. The association between epilepsy and a wide range of psychiatric disorders has long been recognized and it is for the most part those patients with temporal lobe attack who are likely to develop epileptic personality changes and psychoses.

Many varieties of focal attack—or auras—have a temporal lobe origin. Those of psychiatric origin include brief experiences of depersonalisation, déjà vu, fear or panic, depression or elation, as well as hallucinations of smell, taste, hearing and vision. Psychomotor attacks, which are essentially brief periods of clouding of consciousness in which the patient is still capable of semi-purposeful behaviour, are largely confined to these patients, as are the more prolonged episodes or 'twilight states'.

Although a wide range of pathological conditions involving the temporal lobe may give rise to epilepsy, in the great majority of cases the lesions are atrophic and are situated on the medial aspect of one or both lobes. Many are due to birth injury with uncal herniation (Earle, Baldwin and Penfield, 1953). The medial aspect of the temporal lobe is peculiarly sensitive to anoxia, and damage may consequently occur as a result of a wide range of conditions including attacks of grand mal of centrencephalic origin (Scholtz, 1959). It also has the lowest threshold to electrical stimulation of any area of the cerebral cortex so that

FIG. 14.—Temporal lobe epilepsy. (a) Male aged 34 years. Right mid-temporal spike focus. (b) Female aged 37 years. 'Mirror image' anterior temporal spike and slow wave foci. (c) Male aged 42 years. Independent foci: right anterior temporal sharp and slow wave focus, left mid-temporal spike focus.

FIG. 15.—Temporal lobe epilepsy—recording with sphenoidal electrodes. (a) Right-sided spike focus confined to sphenoidal leads. (b) Suppression of barbiturate induced fast activity in right fronto-temporal area, well shown in sphenoidal leads.

scarring of this region is particularly liable to be followed by epileptic seizures.

Interseizure Patterns

The most characteristic feature of the interseizure EEG which indicates the presence of an epileptogenic lesion is a spike or sharp wave focus. The spikes are frequently associated with slow waves (Fig. 14a). Occasionally the discharge consists of paroxysmal activity in the alpha, theta or delta ranges. The paroxysmal epileptiform activity is often superimposed on a background of normal cortical rhythms, but often the lesion may give

other indications of its presence. Focal slow activity, either continuous or fluctuating, is likely in the case of a tumour, while particularly with extensive atrophic lesions, there may be an area of decreased amplitude of the cortical rhythms. Such a diminution of amplitude—sometimes referred to as 'suppression'—is best demonstrated by injecting intravenous thiopentone sodium which induces widespread fast activity. In such cases this fast activity will be less evident or even absent over the lesion (Fig. 15b) (Pamplione, 1952).

The epileptiform discharges seen in cases of

FIG. 16.—Psychomotor epilepsy—seizure pattern. (a) Before attack: generalized low amplitude slow activity. (b) During attack: patient unresponsive, staring and salivating excessively. Bilateral high voltage rhythmical delta discharge with some sharp components.

temporal lobe epilepsy are frequently bilateral. When this is so, they may occur independently indicating that both lobes are damaged; but frequently the discharges are synchronous on the two sides—so-called 'mirror foci' (Fig. 14b). In this latter group the lesion may be unilateral and if the discharges are asymmetrical it is likely to be on the side of the higher amplitude sharp waves. When the discharges are of equal amplitude, it may be impossible from a routine EEG to lateralize the lesion, but asymmetry of barbiturate induced fast activity may enable this to be done. The spike or sharp wave discharges are often very inconstant and in a suspected case it may be necessary to carry out several recordings in order to identify them.

A number of special techniques is employed to aid diagnosis. As the lesion is deeply situated on the medial aspect of the temporal lobe in so many cases, it is not surprising that little or nothing abnormal is detected by using ordinary scalp electrodes. By directing a needle electrode through the mandibular notch below the zygomatic arch so that its tip lies close to the foramen ovale, electrical activity from the inferomesial aspect of the temporal lobe can be recorded (Fig. 15).

A useful method of provoking epileptiform activity is sleep. If the patient does not become drowsy spontaneously, sleep may be induced by any barbiturate or non-barbiturate hypnotic (Gibbs, 1958). Intravenous thiopentone sodium may also be used and has the advantage that drug-induced fast activity may also be studied. The activation of the epileptogenic focus by intravenous leptazol, bemegride, chlorpromazine or imi-

pramine is also useful on occasion, and with the former two drugs the investigation can be extended in order to induce a convulsion. Unfortunately the value of such provocative techniques is severely limited because so often these analeptic drugs give rise to bilaterally synchronous and symmetrical discharges which provide no clue as to the lateralisation of the lesion.

Seizure Patterns

The usual EEG accompaniment of a focal seizure is a train of focal spikes, sharp waves or spike and slow waves, which may remain strictly localized or spread widely in the same hemisphere. Sometimes the opposite hemisphere may also be involved. Immediately prior to the epileptiform activity there may be some flattening of the record. If the discharge and the attack remain localized, the epileptiform discharge will be followed by normal rhythms or, for a period, by low voltage random slow activity. Frequently the focal seizure is followed by an attack of grand mal, in which case the focal discharge gives way to bilaterally synchronous spikes at 8 to 12 a second.

Should a psychomotor attack follow upon the focal seizure, the EEG shows bilaterally synchronous discharges at 2 to 10 c/s.—usually within the range 4 to 6 c/s.—which are most evident in the fronto-temporal areas (Fig. 16). Only occasionally are there sharp wave components. In a few cases the psychomotor attack is accompanied by generalized 14 to 20 c/s. activity, and in others the record merely shows a general diminution in amplitude. Rarely, no discernible change can be detected in the EEG. The bilateral

distribution of the abnormalities in an attack of psychomotor epilepsy, together with the clouding of consciousness which occurs, indicate involvement of the midline structures, although most always secondary to a temporal lobe attack.

In cases showing personality changes or epileptic psychoses the temporal lobe abnormalities are commonly bilateral. If dementia should ensue the epileptiform discharges become less frequent or disappear, the alpha rhythm slows, often to below 8 c/s. and low amplitude diffuse theta and delta activity becomes prominent.

The term 'petit mal' should be restricted to brief absences occurring almost exclusively in children and accompanied by the characteristic bilaterally synchronous and symmetrical wave and spike discharge at 3 c/s. Rarely, prolonged episodes of wave and spike discharge occur accompanied by so-called 'petit mal status' in which the child becomes very disturbed, showing behaviour similar to that seen in an attack of psychomotor epilepsy (Zappoli, 1955).

The occurrence of bilaterally synchronous epileptiform discharges in both the interseizure and seizure patterns associated with focal lesions has already been mentioned. This phenomenon is termed *secondary bilateral synchrony* and, though not common, is most often seen with lesions of the insular region or the medial aspect of one of the temporal lobes. Sometimes the waveform may be very similar to the wave and spike phenomenon seen in petit mal, and there is danger that the discharges will mistakenly be regarded as centrencephalic in origin. Rovit, Gloor and Rasmussen (1961) have described a technique which enables a distinction to be made between these cases. In cases of centrencephalic epilepsy, the intracarotid injection of 25 to 100 mg. amylobarbitone sodium has no effect upon the discharges, whereas in cases of secondary bilateral synchrony such an injection when made on the side of the lesion, abolishes the discharges over both hemispheres.

Effects of Physical Treatments on the EEG E.C.T.

Following E.C.T. the patient's EEG is dominated by slow activity but returns to normal after a variable period. After successive treatments the slow activity takes longer and longer to disappear. In the average patient when treatment is given three times a week, the abnormality becomes persistent after three or four treatments. The EEG consists of generalized theta and delta activity, often frontally predominant, while the alpha activity is diminished in amount and frequency and may disappear. Clinically such a patient is likely to be confused. Roth (1951) has shown that following E.C.T. slow activity is induced more

readily by intravenous thiopentone sodium and that its amount has predictive value in relation to the outcome with E.C.T. When followed up those cases with more slow activity have the better prognosis.

Prefrontal Leucotomy

Following prefrontal leucotomy the EEG is often dominated by high voltage, frontally predominant delta activity which after a few weeks is replaced by theta activity. In most cases the EEG returns to its pre-operative state in about three months. In cases where tension is relieved there may be an increase in the amount of alpha rhythm and a diminution in the amount of fast activity.

Hemispherectomy

Patients suffering from infantile hemiplegia including those with *névroid amentia* (Sturge-Kalischer-Weber syndrome) usually show disturbed behaviour in addition to fits and subnormality. As a rule the EEG is grossly and continuously abnormal. Although the lesion is unilateral, the EEG often shows bilateral spike or spike and slow wave discharges on a background of generalized delta activity. Following hemispherectomy, the disturbed behaviour and fits may cease and the EEG may show a remarkable improvement. In spite of the absence of one hemisphere the EEG may be quite symmetrical though more often there is some diminution in amplitude on the side of the operation.

Conclusions

It is clear from the above account that although certain psychiatric abnormalities are associated with interesting EEG changes, the clinical value of electroencephalography to the psychiatrist is limited. So often, as in delirium, it merely provides additional evidence to support what is already clinically evident. In other cases, to the disappointment of the clinician, the EEG is reported upon as normal or showing a non-specific abnormality. Even in indisputable cases of epilepsy, negative EEG findings are frequent, particularly when special techniques are not employed. Nevertheless, it is in epilepsy that the EEG has its greatest value for in cases of cortical epilepsy, especially those with temporal lobe lesions, it provides information which is unobtainable by any other method.

Although of no value in the diagnosis of hysteria it must be admitted that the EEG can on occasion be of great value in creating doubt as to the accuracy of such a diagnosis. Hysteria is such a frequent misdiagnosis when organic disorders present with bizarre behaviour; psychomotor

epilepsy, acute porphyria, attacks of spontaneous hypoglycaemia, Addison's disease, and cerebral tumour are examples of conditions that may be regarded as hysterical until the EEG findings prompt rescruity of the clinical picture.

It is true of psychiatric cases, as of other referrals for EEG examination, that the clinician should have some knowledge of what electroencephalography has to offer and particularly be

aware of the limitations of the method. Only when full clinical details of the case are available can adequate interpretation be attempted and in addition to supplying these, it is highly desirable that the clinician should pose a precise question to which he requires an answer. When an EEG and its accompanying referral form are scrutinized it must be admitted that often one learns more about the clinician than the patient.

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