HANDBOOK OF ELECTROENCEPHALOGRAPHY AND CLINICAL NEUROPHYSIOLOGY

EDITOR-IN-CHIEF A. REMOND

VOLUME 11

Clinical EEG, I

EDITOR: W. A. COBB

The National Hospital, London (Great Britain)

PART B

EEG Interpretation in Clinical Medicine

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ELSEVIER

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Editor-in-Chief: **Antoine Rémond**
Centre National de la Recherche Scientifique, Paris (France)

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International Federation of Societies for EEG and Clinical Neurophysiology

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International Federation of Societies for EEG and Clinical Neurophysiology

A great need has long been felt for a Handbook giving a complete picture of the present-day knowledge on the electrical activity of the nervous system.

The International Federation of Societies for EEG and Clinical Neurophysiology is happy to be able to present such a Handbook, of which this is a small part.

The decision to prepare this work was made formally by the Federation at its VIIth International Congress. Since then nearly two hundred specialists from all over the world have collaborated in writing the Handbook, each part being prepared jointly by a team of writers.

The Handbook begins with an appraisal of 40 years of achievements by pioneers in these fields and an evaluation of the current use and future perspectives of EEG and EMG. The work subsequently progresses through a wide variety of topics—for example, an analysis of the basic principles of the electrogenesis of the nervous system; a critical review of techniques and methods, including data processing; a description of the normal EEG from birth to death, with special consideration of the effect of physiological and metabolic variables and of the changes relative to brain function and the individual's behaviour in his environment. Finally, a large clinical section covering the electrical abnormalities in various diseases is introduced by a study of electrographic semeiology and of the rules of diagnostic interpretation.

The Handbook will be published in 16 volumes comprising 40 parts (about 2500 pages altogether). For speed of publication most of the 40 parts will be published separately and in random order.

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PART B

EEG INTERPRETATION IN CLINICAL MEDICINE

Editor: **W. A. Cobb**
The National Hospital, London (Great Britain)

Collaborators:

W. A. Cobb, *The National Hospital, London (Great Britain)*

P. Gloor, *Montreal Neurological Institute, Department of Neurology and Neurosurgery, McGill University, Montreal, Quebec (Canada)*

R. Hess, *EEG Station, Kantonsspital, Zurich (Switzerland)*

J. Kugler, *Nervenklinik, Munich (W. Germany)*

Preface

This part of Volume 11 is intended to be a general introduction to the interpretation of the clinical EEG and, as such, should be of interest to all those making their first essay in this field. It attempts to set out general principles and, in illustrating them, inevitably overlaps with much that appears in later volumes (12–15), in which the generalizations are applied to specific cases. Each Section is by a single author and no very strong attempt has been made to prevent him from encroaching on the others' topics, in the belief that a subject is often clarified by presentation from two or more points of view. Even had there not been multiple authors it would have been necessary to divide the work into sections with artificial boundaries. We have largely avoided discussion of a topic only on one side of such a boundary when it could properly be mentioned also on the other.

The primary approach is descriptive and empirical; it could hardly be otherwise since the gap between knowledge of the basic electrophysiology of neurones, as set out in Volume 2B and C, and the systems and mechanisms which subserve the clinical EEG remains extremely wide. Nevertheless, attempts have been made in the Sections which follow to account for some of the phenomena described; they should be regarded only as hypotheses and it should not be forgotten that too much familiarity with a hypothesis tends to promote it into the realm of fact. Witness to this is the widespread clinical use of the term "centrencephalic epilepsy", giving to a once plausible but now insufficient hypothesis the status of dogma.

The first step which the interpreter of the EEG has to take is the attempt to recognize and classify as many as possible of its various components, which he does in terms of their frequency or duration, distribution, form and response to stimuli, thereby analysing the total picture into a number of entities defined by their internal consistency or their resemblance to previously familiar features. In thus forcing all the activities into a limited number of categories he no doubt does violence to the individuality of some of them and his "entities" are surely less simple than it is convenient to believe. Even thus reduced the majority of EEGs retain a complexity which is beyond the verbal skill of most of us to depict adequately.

Among the activities to be defined, paradoxically the most important, to be recognized at the moment of recording if possible, are those of non-cerebral origin collectively called artefacts. These have been fully described elsewhere (Volume 3C and 11A) and here will be totally ignored.

Section I. General Factors Influencing the EEG

For interpreting an EEG all diagnostic conclusions are based almost exclusively on empirical correlations. Such correlations are, however, not necessarily direct, nor are they absolute. Any type of EEG abnormality can be caused in various ways, and different pathological processes may disturb cerebral functions through the same pathophysiological mechanisms. The abnormal function, as reflected in the EEG, is, furthermore, modified by a number of variables—some probably unknown, others uncontrolled—which must be accounted for in our diagnostic considerations.

Generally speaking, the principal factors which influence the bioelectrical activity and its deviations from the "normal" patterns, and which will be discussed in succession, are the following: (1) the condition of the brain prior to the onset of the disease; (2) the nature of the pathological influences; (3) the time at which the patient is examined during the evolution of the disease. The pre-existing condition is the result of a number of contributory factors, namely susceptibility, vascular supply, maturational stage and level of vigilance.

The susceptibility is in part genetically determined. The role of heredity is well established in the case of "symptomatic" or "acquired" epilepsy. It is highly probable that the brain's reaction to other pathological conditions is also influenced by inborn factors. Cerebral damage incurred in pre- or post-natal life is the other component of susceptibility and certainly of importance in the ability to compensate for new deficits. Vascular supply may be included in this same paragraph as far as variations in the circle of Willis or preformed anastomoses are concerned.

This is a decisive factor in the case of obstruction of a major cerebral artery, and also if such a vessel is distended or kinked by displacement of brain by an expanding lesion. Acquired vascular disease likewise influences the severity and type of abnormality induced by an additional pathological process, for example in the case of brain tumours in old people (Van der Drift and Magnus 1961a).

In the first decades of life, particularly in young children, on the other hand, the stage of cerebral maturation determines the form of EEG abnormality caused by a given disease (apart from the fact that many diseases are largely linked to certain age groups). The functional organization of the brain is subject to development, rapid at the onset and gradually slowing down, and the reactions to noxious influences change accordingly. They differ in a quantitative way in that the immature brain responds more readily and strongly, and also in a qualitative way, since different parts of the brain seem to be functionally more labile and more easily disturbed in various stages of maturation (Gibbs and Gibbs 1964; Fig. 1). Moreover, quite specific patterns are linked to well defined age groups in certain types of epilepsy.

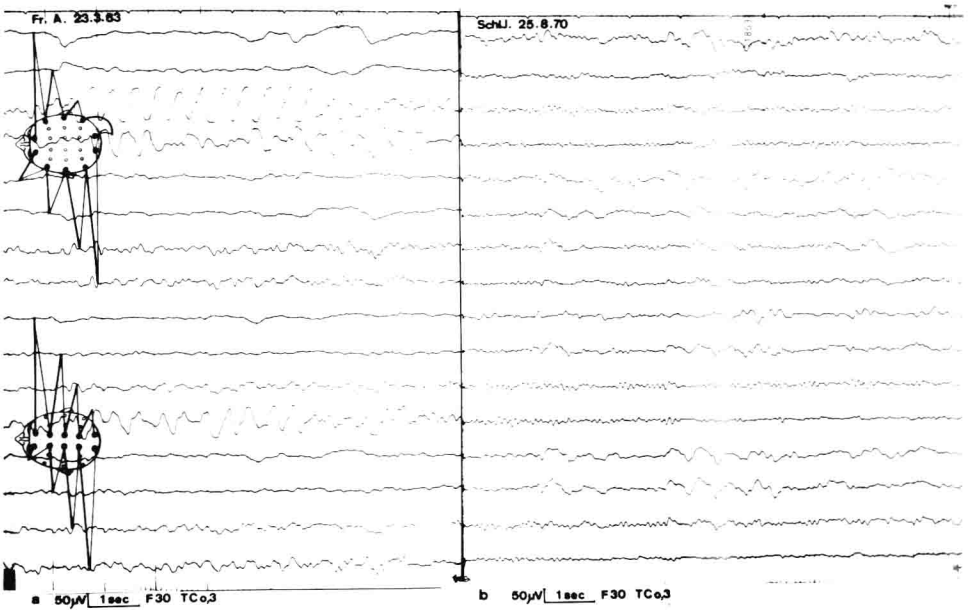

Fig. 1. Comparison of EEGs in chronic subdural haematomas: on the right side in a boy aged 6 (*a*), on the left side in a man aged 74 (*b*). In the child almost continuous high voltage slow rhythms are localized in the lateral occipital region, spared by the haematoma, with steep potential gradients. In the aged person with a haematoma of similar size and location, the delta rhythms are mainly anterior, smaller and show wider spread.

Under all these conditions the electrical activity of the brain depends to a high degree on the level of alertness (Fig. 2). This is in most cases a function of the 24 hour cycle, but also influenced by exterior circumstances such as noise, temperature, time elapsed since the last meal and many more. It must, however, be realized in this context that impaired consciousness may also be caused by the disease which is being examined, and the EEG signs of lowered vigilance are in themselves an important finding. Finally, the emotional state of the patient is of undeniable influence on the EEG: fear and excitement act as arousal stimuli and thus counteract synchronization of neuronal activity (Gastaut *et al.* 1956a). There is no doubt, on the other hand, that in some patients certain emotions (including fear) may also, by unknown mechanisms, lower the threshold for excessive discharge, as most often seen in cases of "temporal epilepsy".

A major element which determines form, extent and severity of EEG abnormalities is obviously the nature of the underlying pathological process. Certain changes of bioelectrical activity may be induced by abnormal discharges from distant brain structures, presumably in the form of distorted patterns of transmitted nervous impulses. This is how "projected rhythms" and mirror foci are explained (*cf.* following Sections). Primary abnormalities are caused by direct noxious influences on the cortical area from which we record. Apart from gross destruction it is nearly always a matter of altered, most commonly decreased, local metabolism, and hypoxia

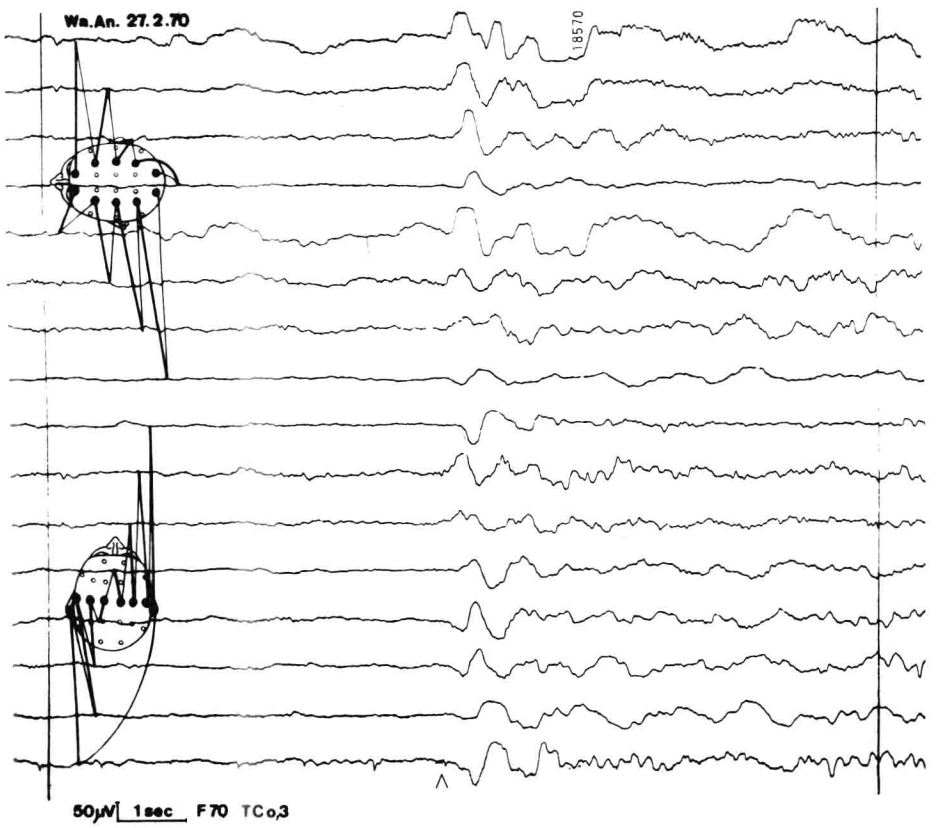

Fig. 2. Effect of acoustic stimulus (∧) in a stuporous patient aged 38, with a left frontal glioblastoma. Low voltage slow waves during decreased level of consciousness are replaced by much increased activity after an arousal stimulus, which is immediately followed by a K complex. The local maximum of abnormality can be seen rather indistinctly as very slow swings centring on the left frontal region, together with depression of fast activity, which appears after the stimulus, near the right frontal pole.

is often the decisive factor. Such is certainly the case in increased intracranial pressure: it tends to decrease the overall circulation, the slowing of which can be demonstrated by serial angiograms (Tönnies and Schiefer 1959). This may be reflected in the EEG by an excess of slow waves (Peiffer 1953), especially in the case of acute pressure (Daly *et al.* 1953; Hess 1961a, b; Langfitt *et al.* 1965b, 1966). Brain areas with previously precarious irrigation are the first to suffer from hypoxia (Obrist *et al.* 1970). Relief of high pressure by shunt operations is apt to lead to rapid improvement of both generalized and focal abnormalities (Fig. 3). This is where systemic blood pressure intervenes, as its rise counteracts the intracranial pressure to some extent (Langfitt *et al.* 1966), whereas hypertension per se does not seem to alter the EEG appreciably (Notermans and Boonstra 1969). In the case of cerebral disease with severely impaired consciousness and respiratory failure, hypoxia is often an aggravating factor, as demonstrated by dramatic improvement of the EEG following tracheotomy. Another possible cause of lowered metabolism leading to

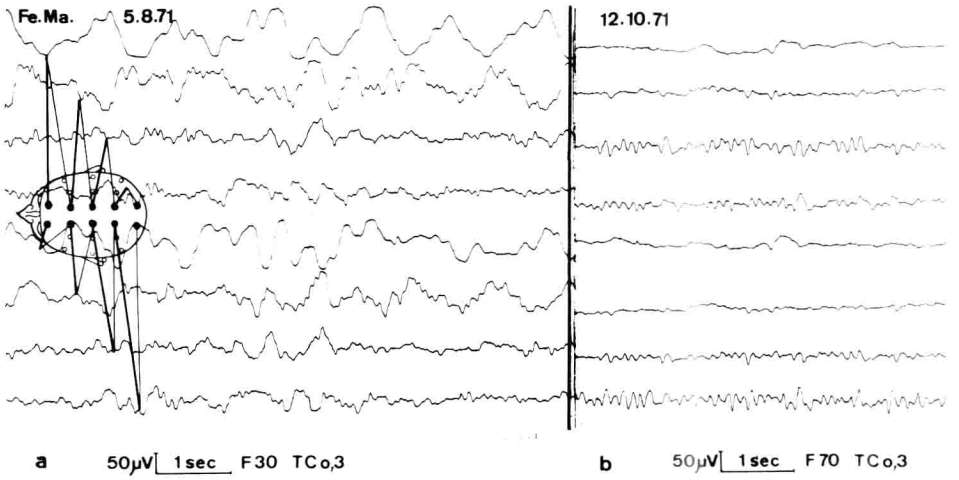

Fig. 3. 24-year-old man. One week after a head injury he suffered a haemorrhage from an aneurysm of the right pericallosal artery, bleeding into the left cingular gyrus. The aneurysm was clipped, the haematoma evacuated, the bone flap removed. The patient remained apathic and developed a frontal syndrome. The EEG (*a*) shows severe slowing most marked in the frontal areas (the right-sided maximum is to be attributed to the missing bone flap). Cisternal scintigraphy gave evidence of malabsorption hydrocephalus. A ventriculo-atrial shunt was effected and later the bone was replaced. An EEG four weeks after the shunt operation (*b*) is moderately abnormal, but much improved and no longer shows marked frontal slow waves.

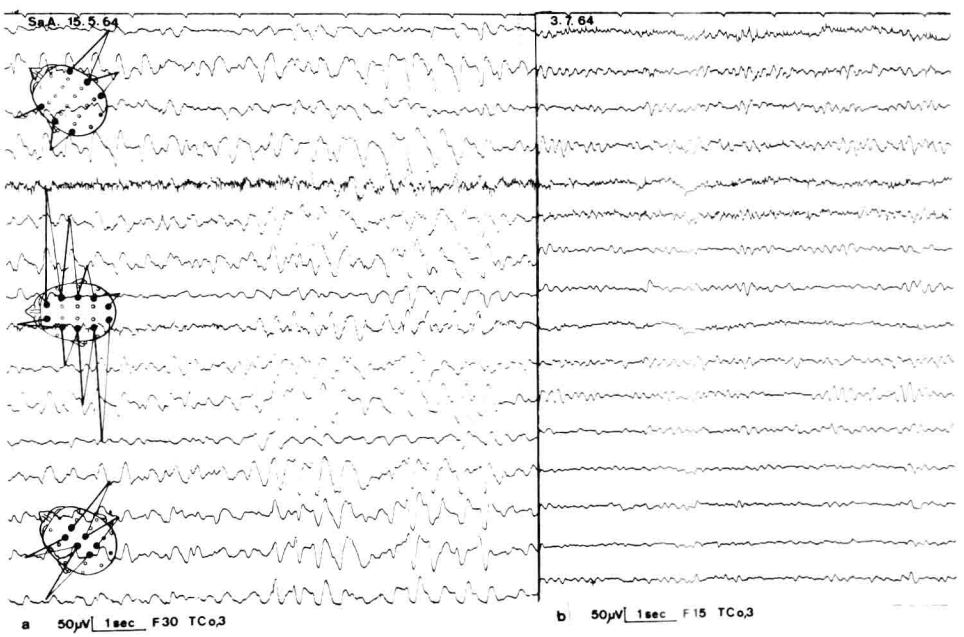

Fig. 4. 56-year-old patient with chronic hepatitis and cirrhosis for three years. *a*: Acute exacerbation, characterized in the EEG by pronounced bilateral delta rhythms, some slow waves locked with sharp transients, forming "pseudo-paroxysmal" patterns. *b*: During chronic period; although the patient shows severe mental disturbance and somnolence, the EEG is only moderately abnormal.

slowing of the EEG is hypoglycaemia. Disturbances of the electrolyte balance are responsible for EEG abnormalities in kidney disorders as well as in infectious diseases and also after severe destruction of brain matter; these latter causes of electrolyte imbalance seem to be most effective in early childhood. Endotoxins are known to alter the bioelectrical activity profoundly. Uraemia and hepatic encephalopathy are the most prominent examples, the latter, however, almost exclusively in the acute stage (Fig. 4). Excess or deficiency of hormones may lead to—usually minor—abnormalities.

The influence of pharmacological agents must be mentioned here for the sake of completeness. In this context it is of particular importance for differential diagnosis: in the case of a coma of unknown origin a record dominated by fast activity strongly suggests an intoxication with barbiturates, hydantoins or tranquillizers. It must, however, be remembered that the same drugs may also cause generalized slowing, especially when they have been used over longer periods. It is noteworthy that chronic overdoses of hydantoins, for instance, may also increase the severity of a

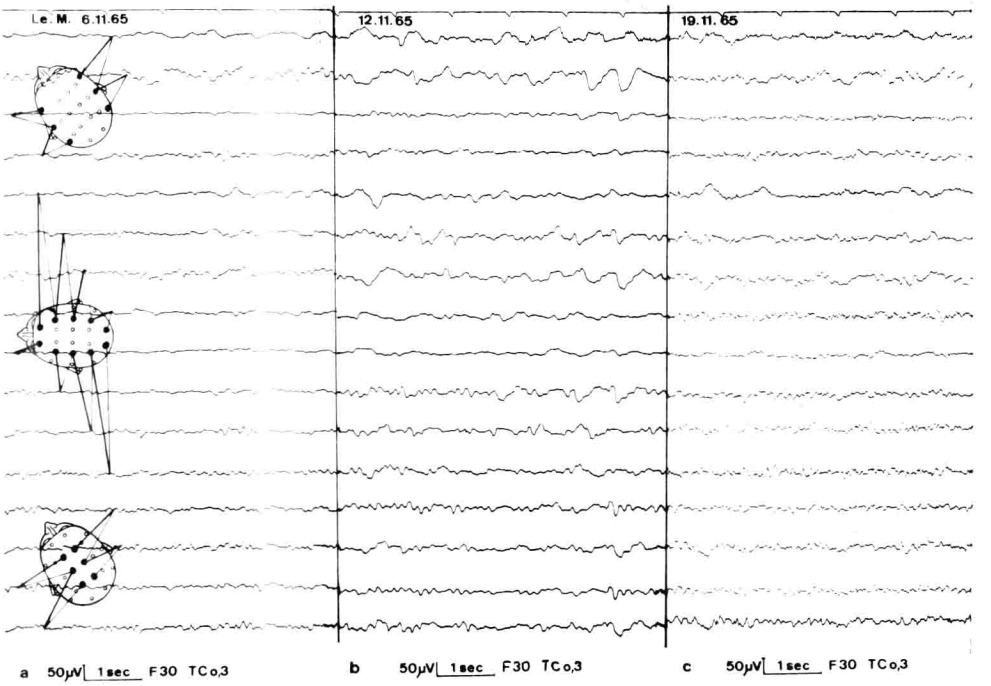

Fig. 5. 30-year-old woman with refractory epileptic condition due to an aneurysm and right temporal intracerebral haematoma, operated on fourteen years ago. Under combined therapy (hydantoins and Tegretol) the EEG (*a*) is moderately abnormal with extended delta area in the old operation field, centring on the temporal region. Eight days after increase of Tegretol (*b*) delta activity is more abundant and somewhat larger in amplitude over the whole of the convexity, as well as in the right anterior quadrant, where the focal disturbance is accentuated. Note the depression on the right, becoming more obvious as the slightly slowed alpha activity is increased on the left. The patient shows at the same time ataxia and nystagmus. Six days after reduction of hydantoins the clinical signs of intoxication have subsided and the EEG (*c*) shows decrease of delta waves, both generalized and focal, although the previous state (*a*) is not yet attained.

focal disturbance and thus lead to an erroneous diagnosis of a progressive lesion (Fig. 5). Changes in the opposite sense, namely a normalization of EEG patterns as a corollary of the therapeutic action of the drugs, are not usually to be expected as an immediate effect; they are often delayed by months or years.

As important as the noxious agent is the acuity with which it impinges on the brain. The faster the damage sets in, the more severe is the ensuing disturbance of function and the longer it takes to recover (Fig. 6). Where shortage of blood supply is the determining factor (which also applies to expanding processes), adaptation by opening of anastomoses takes place and may, in extremely slow processes, be sufficient to maintain normal function for a long time, especially in young subjects. A sudden interruption of blood supply, on the other hand, does not allow for compensa-

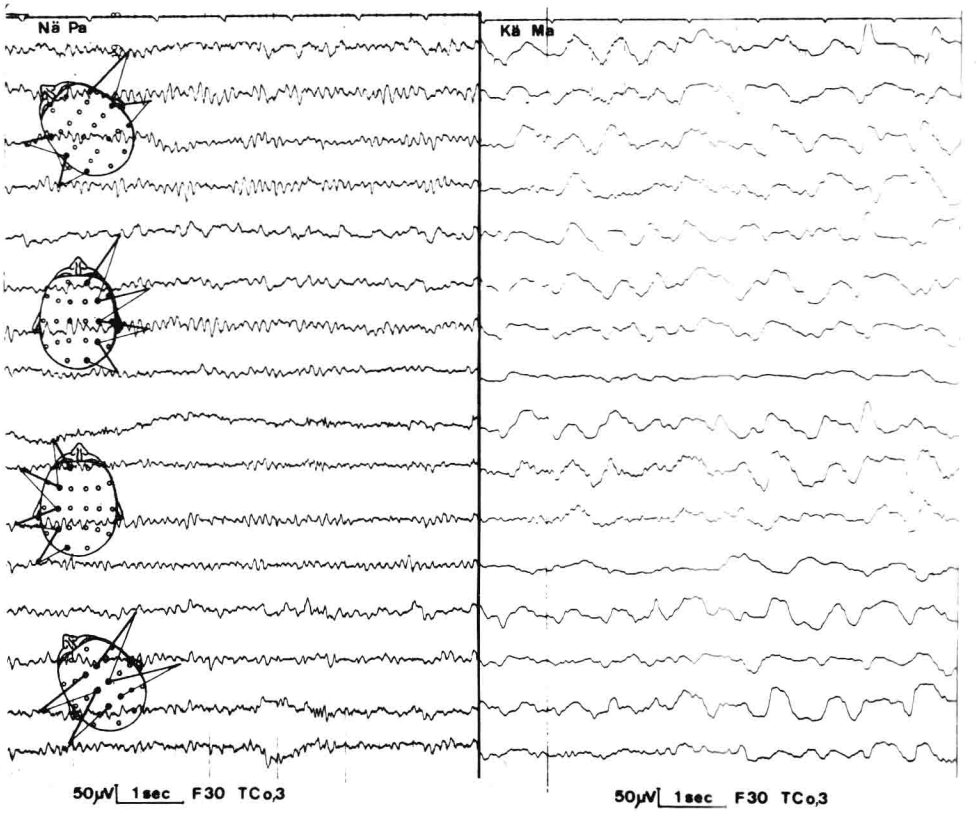

Fig. 6. Comparison of EEGs with chronic and acute lesions of right frontal lobe. *Left*: 37-year-old man with recurring epileptic seizures, following slight head injury nine years ago. All previous investigations had given essentially normal results. The EEG now shows a mild but continuous frontal delta focus on the right. The subsequent operation revealed a large oligodendroglioma in the right precentral area. *Right*: 73-year-old woman suffering from attacks of headache since years, during the last weeks repeatedly followed by short-lived left hemiparesis. Four days prior to EEG she suddenly lost consciousness, showed a left hemiplegia and remained comatose until death. Post mortem a ruptured aneurysm of the anterior communicating artery, with haemorrhage into the right frontal lobe, was found. The EEG is severely abnormal with frontal emphasis, the slowest waves being localized over the right frontal lobe. Superimposed faster activity is markedly reduced on the right.

tion within a useful time. A similar explanation is possible in the case of toxic agents, for instance, in as much as it takes a certain time to metabolize them.

Lastly, it is a matter of course that the extent of the damage is of much importance for the EEG changes it causes. Localized lesions, furthermore, influence the scalp EEG in quite different ways according to their site with respect to the cerebral convexity, and also within the hemisphere, since its various portions do not react in the same way to a given noxious influence.

The last of the initially mentioned principal factors which determine the result of an EEG examination is the time during the evolution of a pathological process at which the test is performed. It seems natural that in the earliest stages of a slowly progressing lesion the EEG disturbances are minimal or absent, and that they gradually increase in severity (Fig. 60) whereas in an acute lesion restitution goes parallel with slowly subsiding abnormalities. It must, however, be realized that the most severe abnormalities are not the most characteristic ones or the easiest to interpret. In the case of a hemisphere tumour, for instance, the first clearly discernible focal signs are most reliable for localization, obscured in later states by secondary disturbances. After an acute lesion the area of maximal damage may be best delimited in the EEG after the stage of initial oedema has subsided (Fig. 58). The first few hours after an acute event, when pathophysiological processes are still in the stage of formation, may be another convenient time for recording unequivocal EEG signs.

In chronic conditions the EEG abnormalities are liable to fluctuate to some extent in connection with the factors outlined in a previous paragraph, such as oxygen availability, systemic blood pressure, degree of hydration, level of consciousness, etc. It may, in addition, be assumed that other biological cycles are of some influence, although reliable data are lacking at the present time.

A major cause of change in the severity of EEG abnormalities is certainly the occurrence of epileptic seizures, in the wake of which generalized as well as focal disturbances are accentuated, sometimes for hours or even days, due to metabolic exhaustion. Occasionally a postictal slow wave focus is for a long time the only EEG evidence of an epileptogenic lesion. In other cases, on the other hand, paroxysmal patterns, diagnostic for epilepsy, are covered up by local slowing or inhibited following a seizure. The opposite event, the activation of focal spikes not seen in the previous record, has been claimed to be not uncommon within 6 hours after a seizure. An increased incidence immediately preceding an attack can also sometimes be observed by chance. There are then periods of evolution in which the EEG is more likely to give useful results than at other times. It is not, however, the rule that the time of examination can be freely chosen.

Section II. Changes in Background Activity

The term "background" is used in the title of this Section to avoid the need for precise definition of what is to be discussed; in usual practice it encompasses those aspects of the ongoing EEG which are not the immediate subject of description. (Any EEG activity representing the setting in which a given normal or abnormal pattern appears and from which such pattern is distinguished: IFSECN (1974).) Thus, all the following activities, whether "normal" or "abnormal", could together form the "background" for the one of them which at the moment is being described. The present Section is concerned with those elements which comprise the normal EEG but which are changed from the normal by disease processes, in the widest sense; it is therefore necessary to consider first the base or bases to which these changes can be referred.

The authors of Vol. 6A, "The EEG of the waking adult", have described these elementary activities in great detail, with copious references, and the emphasis here is therefore mainly on the diagnostic use to which changes in background activity can be put. It is obvious in that volume that the ranges (of frequency, amplitude and distribution) of each component are wide in normal adult subjects, without taking into account such factors as the state of alertness; they are of course much wider when the maturational processes of the first two decades are added. It is the large number of these variables which has made it impossible to define in any satisfactory way the normal EEG or, as must follow, the abnormal one. In fact, no attempt will be made here to discuss or solve this problem which, though of real importance when considering selected populations of "normal" people, such as air-line pilots, is generally of little significance in clinical medicine, that is to say diagnosis, in which the bald classification of the EEG as normal or abnormal is seldom of help to the clinician.

Nevertheless, some aspects of it are worthy of brief discussion. The difficulties are threefold: the large number of components of even a relatively simple EEG, their variability in time, both short term ("vigilance") and long term (maturation–maturity–senescence) and the unreliability of measurements. In the past these measurements have been imprecise and largely intuitive and the information they carried could not contribute to any meaningful statistical picture. Various kinds of analysis have offered some greater precision of measurement but in such forms as to hold out little hope of deriving statistical means which would have practical usefulness. However, modern methods of data processing do offer the possibility of precise measurement and the ability of the computer to deal with very large numbers of data suggests that in the foreseeable future it may be possible to define standards which are constant from day to day and transmissible from place to place.

Meanwhile, it is the conventional multiple voltage–time graph which we must perforce discuss.

If the displacement of a given parameter (*e.g.*, the alpha frequency) is so far as to be outside the normal range then no other reference base than the "normal" is needed and a conclusion on the particular point is possible from a single EEG. However, very frequently the displacement of the EEG from its own individual norms is much more subtle and then a new reference is needed and the patient must be his own control. As in so much of clinical neurology, this may be easy when the process is an asymmetrical one and the affected side can be compared with the unaffected; without clinical or other extraneous clues, however, it is sometimes difficult to decide which is which.

All too seldom the EEG of the patient can be compared with an earlier record of a time when he was well or, not so rarely, less ill and it may be apparent that, although the later record is normal by any accepted criteria, it has nevertheless moved toward a limit of the normal range. Naturally the same sort of comparison can be made much more often retrospectively; for example, the EEG after head injury or vascular accident may be rated as normal though a subsequent record will suggest that the normal for that individual had not been reached at the time of the previous record.

Finally, the subject provides his own control or reference during the course of a single recording, during which it is easier to recognize minimal changes with certainty. In all the foregoing comparisons it has to be assumed that the subject's state of vigilance is constant from one time to another or that the change of vigilance has been observed and documented. Thus, a fall in the amount and amplitude of alpha activity might be due to the disease process or to a change in the level of vigilance in either direction, toward anxious alertness or drowsiness, each of which in turn might be due to the disease, or unrelated to it.

A. REGULARITY

There is a general, perhaps partly intuitive, feeling that regularity of the EEG is a good thing and irregularity a bad one, possibly first expressed in the much abused term "dysrhythmia". It is fairly certain that the impression of irregularity, in the normal frequency ranges at least, is due to the mixture of several rhythms with none predominating and, if a regular EEG becomes irregular, it is because the previously dominant rhythm has become smaller, and hence more similar to the others, or a small rhythm has become larger. The apparently greater irregularity of background activity on the side of a lesion can usually be attributed to reduced alpha amplitude or to mixing of theta waves with the alpha rhythm. Similarly, the apparent irregularity of many children's EEGs may be due to the equal importance in them of theta activity and alpha rhythm and often also the prevalence of posterior slow waves. Thus irregularity, so far as it expresses diminished alpha activity and increased theta activity, may indeed be some measure of abnormality, but if it represents no more than complexity due to several normal rhythms it is doubtful