HANDBOOK OF ELECTROENCEPHALOGRAPHY AND CLINICAL NEUROPHYSIOLOGY

EDITOR-IN-CHIEF A. REMOND

VOLUME 14

Clinical EEG, IV

EDITOR: O. MAGNUS

St. Ursula Kliniek, Wassenaar (The Netherlands)

PART B

Traumatic Disorders

EDITOR: J. COURJON

Hôpital Neurologique, Lyon (France)

ELSEVIER

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Editor-in-Chief: **Antoine Rémond**
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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

TRAUMATIC DISORDERS

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Preface

Study of EEG in traumatic disorders implies different problems. Most of the authors divide the period after head injury into two stages: acute and chronic. Great discrepancies exist in the literature regarding the duration of the acute and the onset of the chronic post-traumatic stage. According to Bickford and Klass (1966) the chronic stage would correspond to an unchanging and unchangeable final state. Big differences will be seen between patients.

The following problems will be treated in this Part:

1. Analytical study of generalized and localized abnormalities (amplitude, frequency, unusual grapho-elements).

2. Gross modifications of the EEG in relation to disturbances of consciousness in prolonged recordings and the evolution in serial EEG studies, influence of age and anterior state, combination of various EEG signs and correlation with results of the other procedures.

3. EEG aspects in different manifestations, various deficits, meningeal haemorrhage, early haematomas and early seizures.

4. Modifications of electrical activity in (associated) non-cranial injuries in post-concussion states, EEG aspects in late subdural collections, infectious complications, post-traumatic epilepsy and medico-legal implications.

Section I. Introduction

Head injuries comprise lesions both of the brain and of its protective coverings, and each of these can exert its own influence on cerebral electrical activity. Courville (1937) and Gurdjian *et al.* (1955) have studied the mechanism of fractures. Linear fractures occur as a result of moderate force, and are situated at the boundary between an area of depression and an area of elevation. The region of damage corresponds to the displacement of the internal table of the skull, whereas with a penetrating wound caused by a sharp object the dura may be torn and the brain may be lacerated. Tearing of the branches of the middle meningeal artery, or of extradural or subdural veins, may give rise to haemorrhage in the damaged area, producing an extradural haematoma or a subdural haematoma. The dural tear may give rise to infections, particularly to septic meningitis. A depressed fracture of the skull can cause such a tear, but this is uncommon. Subarachnoid haemorrhage may occur as a consequence of fractures.

Central nervous system lesions are of three types:

1. Contusion consists of superficial or deep bruising, and haemorrhagic plaques spread throughout the brain tissue and accompanied by oedema.

2. A laceration is composed of a mixture of blood and torn brain tissue, and is surrounded by an area of oedematous brain tissue.

3. An intracerebral haematoma consists of a mass of blood, coagulated to some degree, which is situated in the white matter, commonly of the frontal or temporal lobes and which is usually associated with a deep craniocerebral injury.

The damaged cells are destroyed and cleared away by macrophages, mobilized in association with neuroglia, and this stage is followed by astrocytic proliferation, which produces a scar. Such scars are found either at the point of impact or at the site of a contrecoup injury, but similar damage may result from trauma due to impact of the brain against the lesser wing of the sphenoid, producing contusion of the orbital surface of the frontal lobe or the anterior pole of the temporal lobe. Similar movements result in tearing of blood vessels, particularly of the veins over the surface of the cortex.

The brain stem may be damaged in one of two main ways: either by direct collision with the bones of the base of the skull, which is usually fatal; or by compression and distortion, *e.g.*, by a basal haematoma, or herniation of the temporal lobe through the tentorial hiatus, as may occur with diffuse cerebral oedema. The white matter of the hemispheres is damaged in more serious injuries. Areas of disintegration are found in the centrum semi ovale and in the corpus callosum.

Cerebral concussion is the immediate reaction to damage of the nerve cell membrane and is responsible for loss of consciousness. The majority of authors consider that loss of consciousness is due primarily to a disorder of the cortex (Unterharn-

scheidt and Higgins 1969). Lesions due to contusion and laceration may produce a progressive disorder, because they can be followed by anoxia. If a contusion is purely cortical, it produces local damage with focal neurological deficits. If the brain stem is involved, disorders of consciousness are likely to recur. Secondary damage from ischaemia and hypoxia may produce cortical lesions; the most vulnerable site is the Ammon's horn, followed by other regions of the brain the vascular supply of which is limited.

Several factors appear to be involved in the development of cerebral oedema, such as venous stasis as a result of compression of the superior vena cava or of a haematoma, direct intracerebral compression, hypoproteinaemia, vasodilatation and altered cellular permeability. Anoxia will accentuate this last process, which then results in a state of shock, with disturbances of respiration and of metabolism. Cerebral oedema may be maintained by water and electrolyte imbalance, in association with malfunction of the supra-optico-hypophyseal system and the adrenal cortex and also by inadequate medical management.

Section II. Historical Data

Williams (1941) gave the first full paper on the EEG modifications in head injuries. Walker *et al.* (1944) studied the physiological basis of concussion. Jasper *et al.* (1945) published personal data. Dow *et al.* (1944) studied EEG results of experimental trauma in dogs. Fischgold and Bounes (1946) insisted on the EEG modification in relation to vigilance. Ward (1948) in another experimental work observed slow waves or permanent flattening. Melin (1949) reported on a special study of EEG disturbances in head injuries of children. Cobb (1950) pointed out the effects of concussion. Dawson *et al.* (1951) observed 45 patients with repeated records and studied the evolution of abnormalities.

Cazzulo (1954) analyzed the value of the EEG for the diagnosis of traumatic cerebral lesions. Foltz *et al.* (1953) attributed flattening of EEG records to alteration of the reticular formation. Meyer and Denny-Brown (1955) reported modifications of cerebral circulation in brain injury. Steinmann (1959) gave a complete study of head injuries.

Section III. Early EEG Changes

Abnormalities of the EEG are varied, as they may be due to both diffuse and focal changes which may vary from one moment to another. They depend on the time from the onset of injury, its evolution, the appearance of complications, and above all on the patient's level of consciousness. Therefore it is necessary to know the state of the patient's responsiveness in order to be able to interpret an EEG at any given moment. The time of day, electrolyte and endocrine abnormalities and therapy may influence the record.

A. GENERALIZED ABNORMALITIES

The most severe damage is represented by *electrical silence* (Fig. 1) defined as absence of electrical activity despite high amplification (minimum 25 microvolts per cm), using montages with adequate inter-electrode distances. Sometimes an isoelectric trace may be interrupted by periodic bursts of cerebral activity and it is important to continue recording for an adequate length of time.

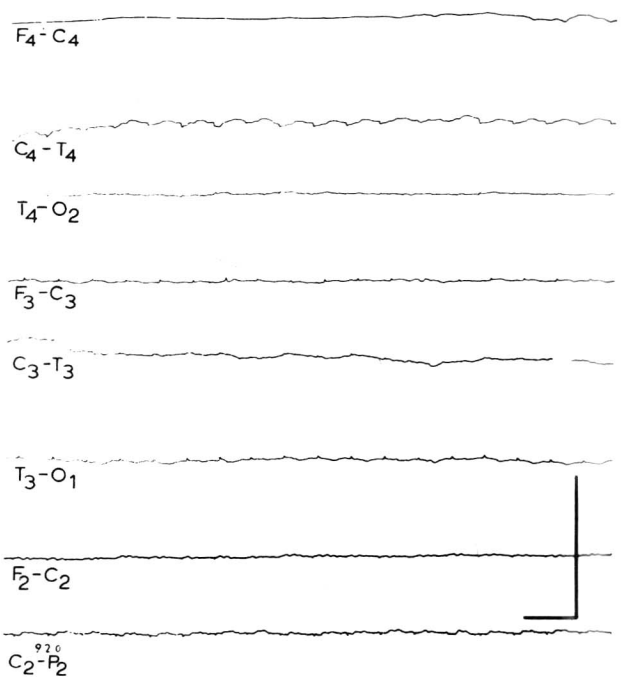

Fig. 1. Isoelectric activity; cerebral death. Time constant: 0.3 sec; calibration: 100 μV.

Silverman *et al.* (1970) made the following recommendations: the recognition of an isoelectric tracing, that is to say, a total absence of cerebral electrical activity, can only be made if certain conditions are fulfilled. At least ten electrodes must be spaced equally over the scalp, with resistances below 10,000 ohms and at least eight channels of recording must be obtained; the amplification must be at least 25 microvolts per cm and recording must be carried out for at least thirty minutes at a time. The noise level of the machine must be checked at an appropriate gain. A time constant of 0.3 must be employed, with intermittent use of the longest time constant available. No high frequency filters must be employed (Arfel 1970).

Bipolar derivations should be used, at both short (5–6 cm) and longer distances; simultaneous records must be made of the electrocardiogram and of respiration, with particular attention to the artefacts which may be produced by automatic ventilation. The patient's response to sensory and painful stimulation should be recorded.

When there are signs of severe coma, the first EEG recording should be taken as soon as possible and should continue for one hour. Subsequent recordings should be obtained every two hours. When the subject is likely to be used as a donor for organ transplantation, EEG monitoring should continue for at least 24 hours from the time when the recording first fulfilled the criteria of cerebral death. Recently the isoelectric EEG has been employed only as an indication for cerebral panarteriography in which a complete stop of cerebral circulation is considered as evidence for cerebral death.

Diffuse depression of cerebral activity occurs rarely. General diminution in voltage appears predominantly as a prelude to an isoelectric tracing, at a stage when the activity appears only in isolated bursts (Fig. 2).

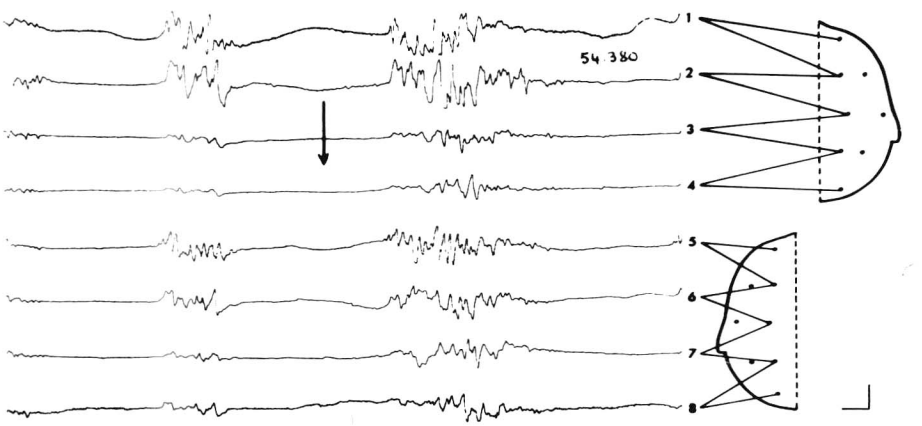

Fig. 2. Anoxic lesions; delta wave bursts alternating with flat activity. Time constant: 0.3 sec; calibration: 100 μV.

Generalized slowing can affect all frequency ranges. The slowing is most marked in children (Fig. 3), when it is accompanied by an equally marked increase in amplitude. Rates of $\frac{1}{2}$–1 c/sec and slower correspond to the most severe cerebral reactions. Other delta and theta activity may be either intermittent or continuous; sometimes the acti-

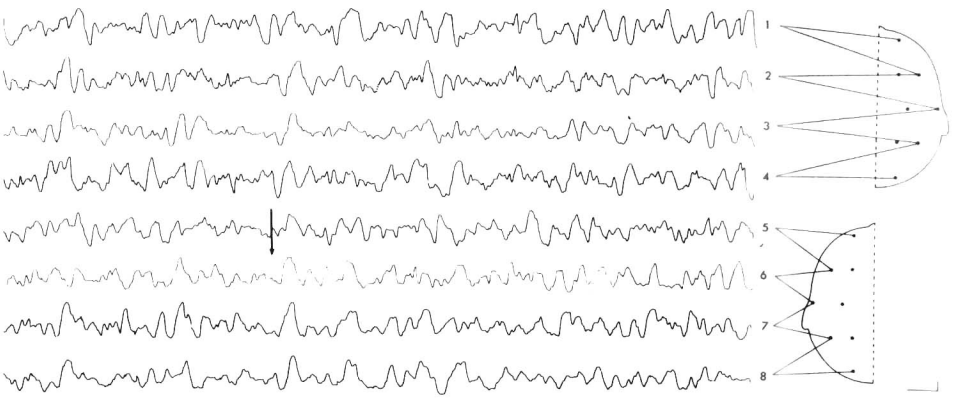

Fig. 3. Boy 9 years old; permanent diffuse slow waves; no response with stimulus (arrow). Time constant: 0.3 sec; calibration: 100 μV.

vity is markedly changed by external stimuli, but sometimes it is totally unreactive. Sometimes generalized slowing appears only in response to sensory stimulation and may be seen for several minutes at the beginning of the recording, after placement of electrodes, but the EEG may show no signs of reactivity to auditory or painful stimulation. A noisy environment may be responsible for continuous stimulation, giving rise to such activity in the record. Its significance can only be assessed by recording for a sufficient length of time and by taking into account the external conditions.

Generalized slowing is usually accompanied by a loss of clear spatial distributions of rhythms. All regions show virtually the same pattern, often consisting of various frequencies.

The mildest form of this abnormality is a *slowing of the alpha rhythm*, as has been emphasized by Jung (1953) and Meyer-Mickeleit (1953). Schneider and Hubach (1962), and later Scherzer (1965a), have studied the general slowing of alpha rhythm, and have considered it to be the slightest degree of generalized disturbances after cerebral injury. Follow-up EEGs during several weeks show a gradual increase of frequency, until it returns to normal.

In the alpha frequency a particular activity sometimes occurs which consists of a *uniform 8 c/sec rhythm* and which is not modified by any form of stimulus, even a painful one. It is associated with states of deep coma. It has been described by Loeb *et al.* (1959) and by Fischgold and Mathis (1959). On the whole the spatial distribution of rhythms and their reactivity are most important, for more important than their rate. Similar data have been obtained by Loeb and Poggio (1953) in acute vascular accidents of the brain, which gives some idea of the likely site of damage in these cases.

Generalized fast activity at 16–20 c/sec is uncommon. It is usually unreactive to stimulation, and is a feature of profound coma. One particular variety of generalized fast activity, which will be discussed later, is represented by 12 and 14 c/sec activity appearing in short bursts, alternating with diffuse slow waves. This pattern was described by Courjon and Bonnet (1955), and was studied in detail by Chatrian *et al.* (1963).

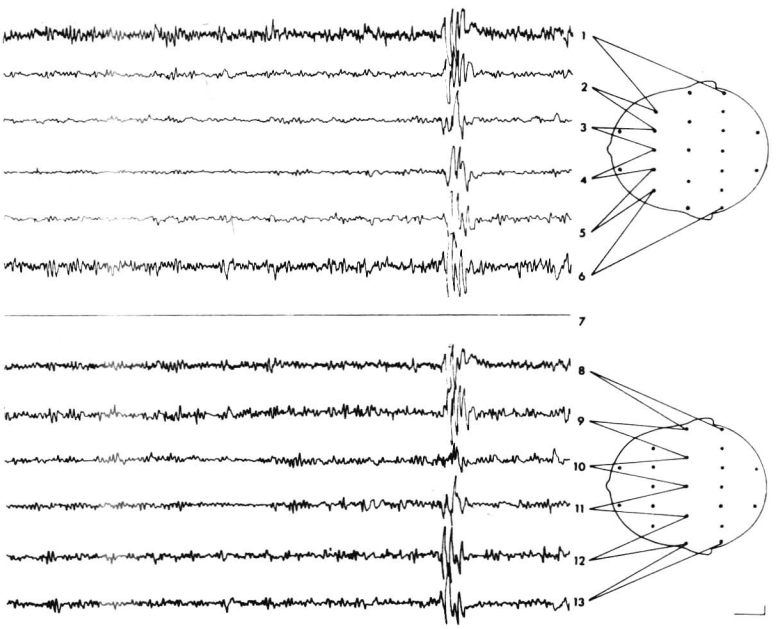

Fig. 4. 12 years old; six weeks after trauma; spontaneous diffuse spikes and waves; no later epilepsy. Time constant: 0.3 sec; calibration: 100 μV.

Diffuse spontaneous spike-and-wave activity (Fig. 4) is rarely observed at an early stage. It was described by Gurdjian *et al.* in 1955, and was the subject of a follow-up study by Courjon (1970b). This abnormality occurs only in children and appears in short irregular bursts, which never take on the rhythmic aspect of 3 per second or $3\frac{1}{2}$ per second spikes-and-waves seen in petit mal or generalized epilepsy. It is not accompanied by any clinical change. Follow-up studies show that it disappears after about six months. Occasionally it persists longer, but even so it is not accompanied by clinical epilepsy. This prognostic fact is obviously of great importance.

However, if such abnormalities appear after the age of 15 years, a past or family history of epilepsy should be investigated. Occasionally diffuse polyspikes and waves accompanied by generalized myoclonus occur in the first few hours or days after an accident; in such cases it is usually acute anoxia rather than the head injury itself which is responsible for their appearance.

B. LOCALIZED ABNORMALITIES

The distinction between general and local abnormalities is somewhat arbitrary, for many cases of diffuse abnormality are associated with focal signs; this is particularly true of local slow waves associated with a generalized slowing. The significance of local changes depends on their constancy for some of them are extremely labile.

Local abnormalities consist of a local change of frequency or amplitude, or the appearance of abnormal elements.

1. Changes of amplitude

Perhaps the smallest change which can be recognized is *focal reduction of alpha rhythm* in the parietal, posterior temporal and occipital regions of one hemisphere; this is usually preceded by the appearance of localized slow waves (Jung 1953; Meyer-Mickeleit 1953). Radermecker (1964) has stressed the difficulty of distinguishing this abnormality from physiological alpha asymmetry. In cases of focal reduction, the alpha rhythm is not only of lower voltage but also less regular than on the healthy side, and often shows brief periods of interruption. The alpha index is thus reduced on the affected side (Meyer-Mickeleit 1953).

Localized flattening (Fig. 5) is an important abnormality but it is often difficult to recognize. It is important because it may imply the existence of intracranial haematoma or severe laceration, provided that subcutaneous oedema or haematoma have been excluded as possible causes. It is also important to ascertain that there has been no short circuit between two electrodes by measuring inter-electrode resistance. Such flattening rarely occurs over a whole hemisphere; it is usually localized to a limited region, such as the centro-parietal or the temporal area. It can be present constantly or occur intermittently, without any evident accompanying change in the level of consciousness.

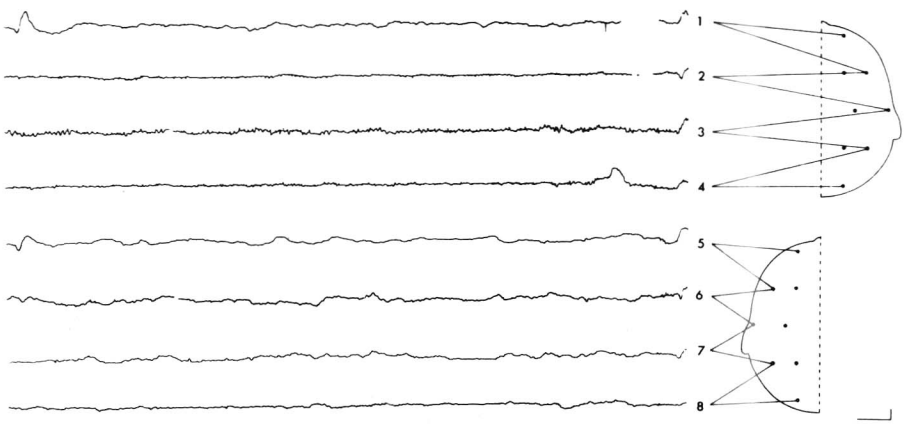

Fig. 5. 40 years old; localized flattening; haemorrhagic contusion. Time constant: 0.3 sec; calibration: 100 μV.

High amplitude usually occurs with slow waves, and is proportional to their slowness; the slowest waves usually have the highest amplitude. It may be difficult to interpret differences in amplitude, as they may represent depression of activity on one side or enhanced amplitude on the other.

2. Changes in frequency

Localized change of alpha frequency has been particularly studied by German workers. It has been largely overlooked by French authors, probably because they habitually use a 15 mm per second paper speed.

Focal slowing of the alpha frequency is transitory and is always preceded by a focus of slow wave activity. A slowing of at least 1 c/sec or 1.5 c/sec should be observed before it is regarded as significant. The use of automatic frequency analysis in at least 2 channels simultaneously may be necessary to detect constant frequency asymmetries. Such focal slowing usually disappears rapidly and may sometimes be followed by a focal reduction in amplitude (focal suppression of alpha rhythm). Focal activation of alpha rhythm (Duensing 1948b) is rare. It occurs in association with a localized slowing of alpha rhythm, and with a loss of its reactivity on eye opening. It is usually transient (Meyer-Mickeleit 1953).

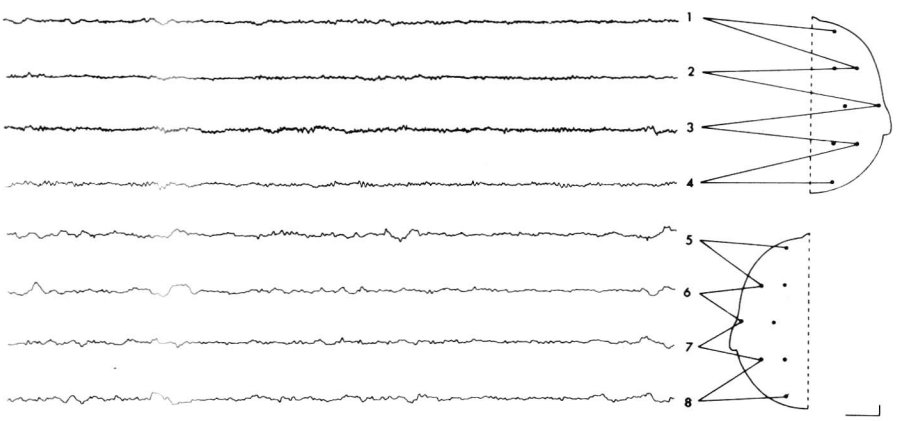

Fig. 6. 29 years old; 36 days after trauma; papilloedema; delta activity over the left hemisphere; surgical exploration shows cerebral oedema. Time constant: 0.3 sec; calibration: 100 μV.

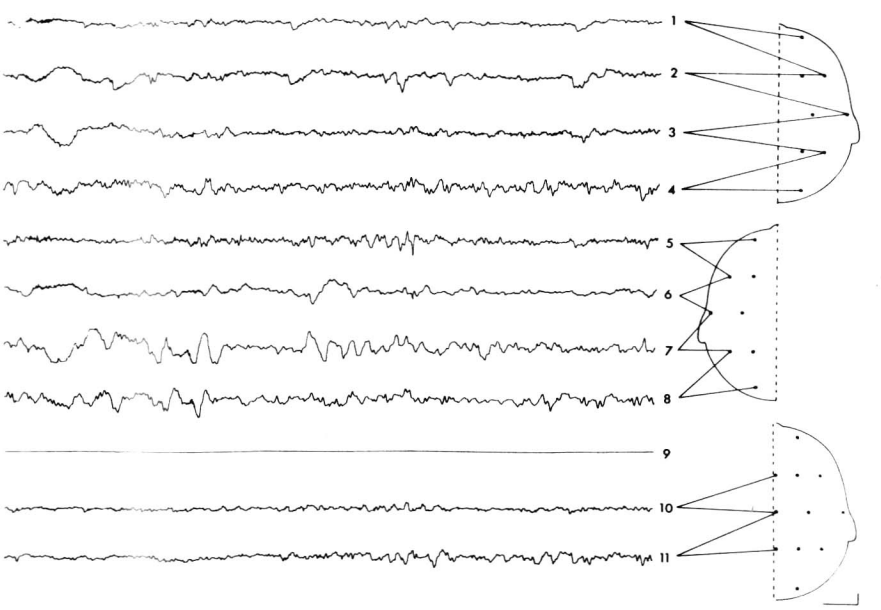

Fig. 7. 17 years old; fifth day after trauma; left posterior temporal delta activity. Time constant: 0.3 sec; calibration: 100 μV.