22. Electroencephalographic Findings in the Apallic Syndrome

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Introduction

The electroencephalogram (EEG) records the activity of cortical neurons which are in close contact with thalamic structures and under the permanent synchronizing or desynchronizing influence of the formatio reticularis of the brain stem. Alterations in the cortical region, and also in the basal ganglia, the brain stem and the connecting pathways may modify normal EEG activity. The EEG, however, always reflects only the *functional state* of the cortex without providing information on the degree and localization of morphologic alterations in the individual cases. At the same time, the general viewpoint prevails that EEG activity is strongly correlated with wakefulness, as evidenced by the fact that (1) normal basic alpha activity of 8-13/s is observed only in the relaxed waking state, (2) increased attention leads to desynchronized EEG activity by activation in the ascendent formatio reticularis mesencephali, and (3) a decrease in wakefulness, in sleep or coma is accompanied by synchronized slow EEG waves. A reservation must be made with regard to this view concerning certain phases of sleep (REM phases) as well as comatose states due to lesions in the region of the pons of the brain stem (Loeb et al., 1959).

The apallic syndrome confronts us with a clinical picture pointing mainly to a functional disturbance in the region of the cerebral hemispheres. Initially it may be caused by modifications in the cortex itself or in subcortical structures and in the connecting pathways. Coma vigile as the cardinal symptom is characterized by maintained wakefulness and sleep-wakefulness rhythm and by the suppression of consciousness. This requires intact awakening systems in the upper brain stem (ascendent activating system of the formatio reticularis). The dissociation of wakefulness and consciousness and the other signs of the apallic syndrome, as well as the assumption of an underlying functional disturbance mainly in the area of the cerebral hemispheres, point toward the importance of undertaking a systematic study of the EEGs recorded during the completely developed apallic syndrome, during the individual remission phases, and during the defect stage.

Only those patients with a traumatic apallic syndrome which in all cases had developed after a midbrain syndrome caused by tentorial herniation were chosen for the present investigation. EEGs from apallic syndromes of other etiology (e.g., metabolic, anoxic) are only rarely discussed in other papers (Vigouroux et al., 1964).

Materials and Methods

followed up clinically and electroencephalographically. At the time, 18 patients were between the ages of 20 and 29 years, 3 patients between the ages of 32 and 38 years, 1 patient was 43 years old and another was 54 years old, making a total of 21 men and 2 women. At the time of follow-up examination, the craniocerebral trauma had existed for 1-2 years in 7 patients, for 3-4 years in 11 patients and for 5-6 years in 5 patients. After hospital treatment the patients had repeatedly been examined clinically and electroencephalographically, so that exact notes on the clinical course were available. On the basis of this documentation and according to the criteria formulated by Gerstenbrand (1967), the clinical development was divided into different stages: (1) acute midbrain syndrome, (2) transition stage evolving into the apallic syndrome, (3) completely developed apallic syndrome, (4) remission stage of the apallic syndrome, and (5) defect stage following the apallic syndrome. Of the 23 patients, 14 were largely resocialized, 4 patients were completely disabled, and 5 patients showed disablements of varying degrees.

Due to repeated follow-up examinations, 119 EEGs were available from the 23 patients. An EEG was recorded from 1 patient during the acute midbrain syndrome, during the transition stage from 3 patients, and during the completely developed apallic syndrome from 4 patients. Twenty-two patients were examined electroencephalographically during the remission stage, some of them repeatedly, and altogether 45 recordings were performed. Several recordings were made during the defect stage, so that in this stage 65 EEGs from the 23 patients were available. All of the 119 curves were evaluated by three of the authors independently from one another according to the following criteria: *basic activity* (generalized background activity); degree of *generalized changes* (s = severe generalized changes with predominant theta and delta waves; mo = moderate generalized changes with predominant theta waves and interposition of delta waves; m = mild generalized changes with increased theta activity; 0 = no generalized changes); *foci* and their localization in relation to the site of trauma; *irritative phenomena* in the form of paroxysmal delta waves and/or sharp waves; and *epileptic phenomena* in the form of spikes or spikes and waves.

Results

The analysis of the clinical course reveals developments of varying rates for the 23 patients, from the acute midbrain syndrome to the transition stage and to the completely developed traumatic apallic syndrome. Also, the duration of the completely developed traumatic apallic syndrome, and especially that of the remission stage, vary considerably. Table 1 demonstrates the shortest, average, and longest duration of the individual stages. The beginning of the defect stage, in which no more appreciable clinical remission took place, was observed on an average after 350 days, at the latest after nearly 800 days, and in one case as soon as 60 days after the accident. The onset of the defect stage and thus the end of remission could for some patients be stated only approximately because sometimes even after discharge from the hospital a further remission took place, the termination of which could not be exactly

Table 1. Duration of the clinical stages in 23 patients with traumatic apallic syndromes

	Shortest duration (days)	Average duration (days)	Longest duration (days)
Acute midbrain syndrome	0.5	2.7	6
Transition stage	2	11	19
Completely developed TAS	2	20	50
Remission stage	54	316	775

The correlation of the individual EEGs with the clinical stages for each of the 23 patients revealed considerable differences between individuals, but generally the same developmental trend was seen for the whole group. The different severity of EEG changes in relation to the individual clinical stages is shown in two examples. Figure 1 shows the

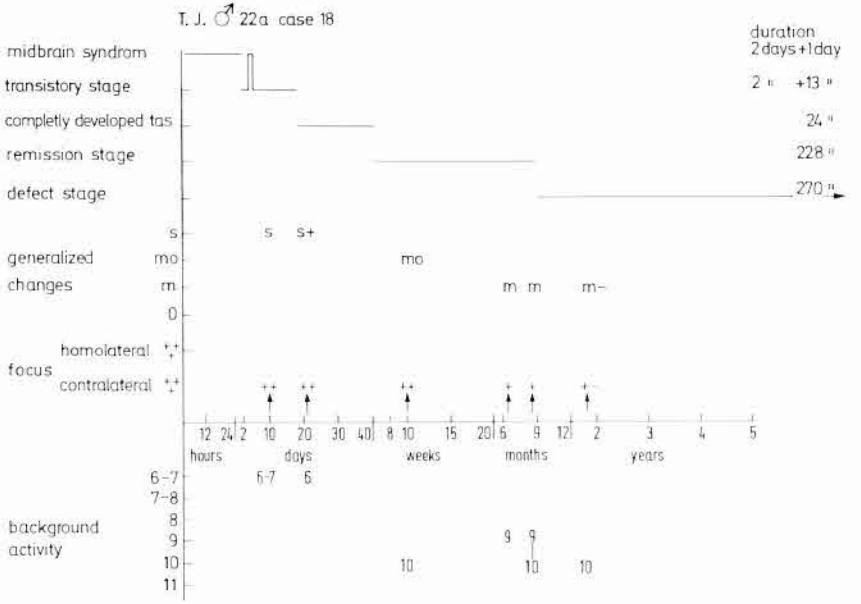


Fig. 1. Clinical and electroencephalographic course of 22-year-old patient (T.J.) with traumatic apallic syndrome (*tas*). Trauma left-frontally. *s* = severe (*s+* = more severe), *mo* = moderate, *m* = mild (*m–* = slighter). ++ = severe focus, + = mild focus, +– = barely perceivable focus

clinical and electroencephalographic course of a 22-year-old patient. The duration of the individual clinical stages corresponds approximately to the patient collective (see Table 1). During the transition stage there is a short-term relapse to the acute midbrain syndrome. The first EEG was recorded during the transition stage and indicates severe general disturbances, a distinct focus contralateral to the site of trauma, and a basic activity of 6-7/s. At the onset of the completely developed traumatic apallic syndrome the generalized changes have even increased. Ten weeks after the accident–already in the remission stage–generalized changes of a moderate degree with still-distinct signs of a contralateral focus and an acceleration of the basic activity to 10/s are observed. After 6 1/2-8 1/2 months following the accident the patient is still in the remission stage: the EEG patterns are largely the same, except for a somewhat slower basic activity. After 9 months the patient reaches the defect stage. More than 1 year later the EEG is nearly normal and presents only discrete generalized changes and focal symptoms.

Figure 2 shows the clinical and electroencephalographic development of another 22-

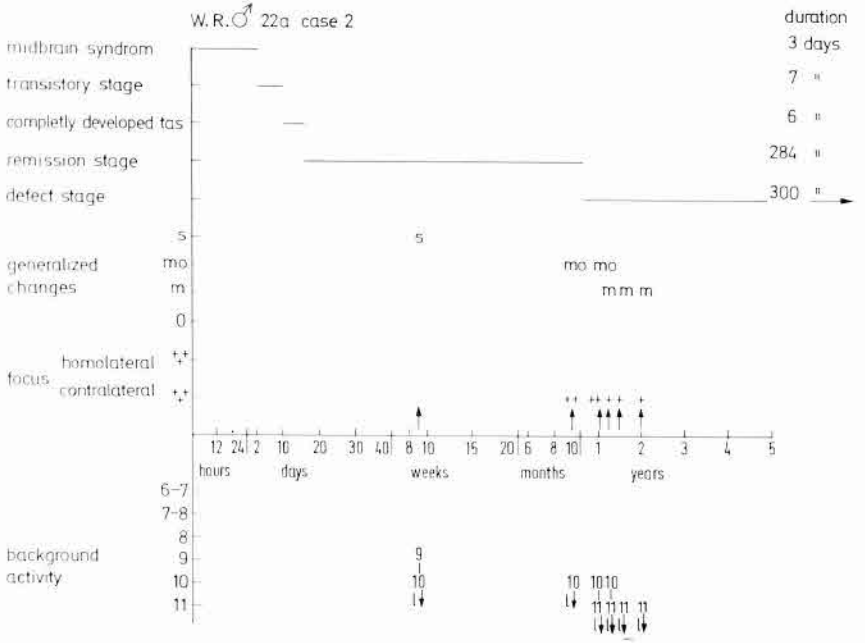


Fig. 2. Clinical and electroencephalographic course of 22-year-old patient (W.R.) with traumatic apallic syndrome (*tas*). Trauma right-fronto-temporal. For abbreviations see Figure 1. l = at left less marked

transition stage and the completely developed traumatic apallic syndrome very rapidly. During the remission stage (9 weeks after the accident), the EEG shows severe generalized changes with a less marked background activity of 9-10/s at the left and without other focal symptoms. Seven months later, toward the end of the remission stage, the generalized changes have slightly regressed, whereas a significant theta-delta focus is observed contralateral to the site of trauma. Three months later, already in the defect stage, this picture was found to be unaltered except for a slight acceleration in basic activity. In three further examinations, performed in the following years in which no further clinical improvement took place, the generalized changes had further regressed, the focus was only insignificantly marked, and the basic activity was stabilized at a frequency of 11/s with a further slight decrease over the left hemisphere.

Figure 3 shows representative EEG tracings for one patient with a traumatic apallic syndrome, taken from recordings performed during (1) the transition stage from the acute midbrain syndrome to the traumatic apallic syndrome, (2) during the completely developed traumatic apallic syndrome, and (3) during the defect stage, which was reached 9 months later. In the transition stage (*A*)–associated with the clinical picture of a marked brain stem lesion–sections of flat activity with a well-maintained basic activity of 7-8/s alternating with sections of diffuse delta interposition of a higher amplitude can be seen. The EEG in the completely developed traumatic apallic syndrome (*B*) is dominated by the theta waves and by delta waves which are more pronounced on the left. Almost 11 months after the accident,

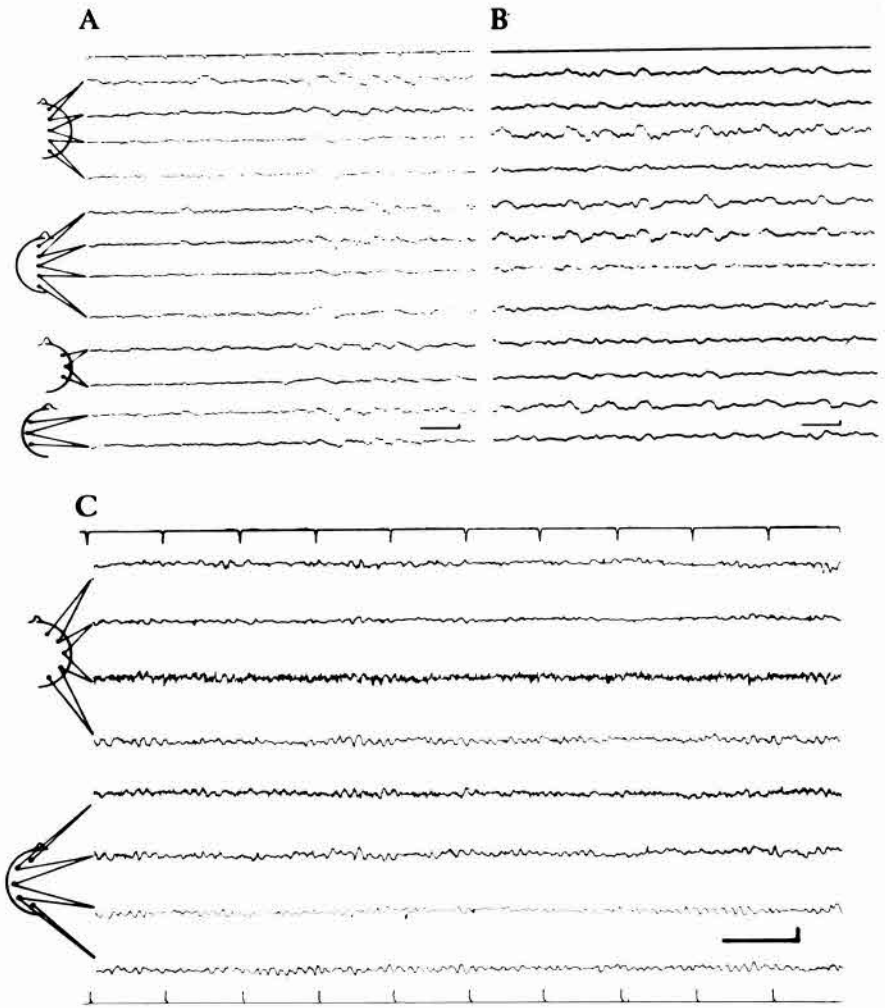
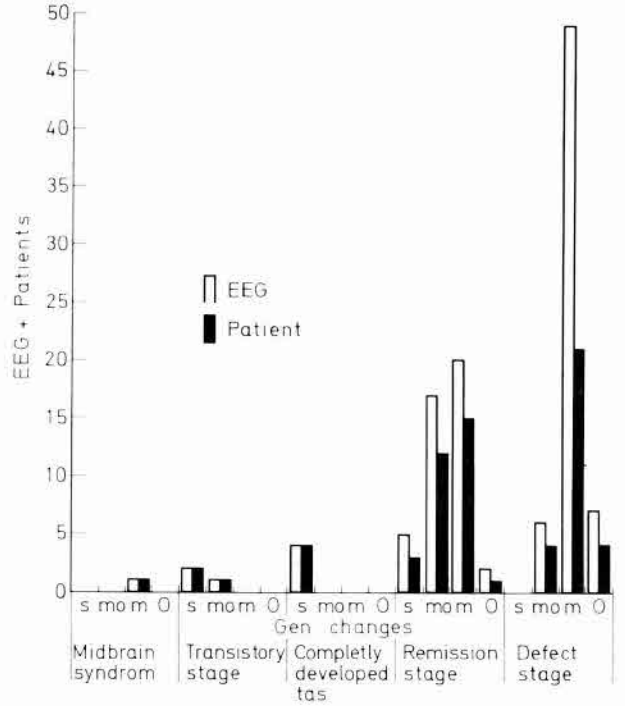
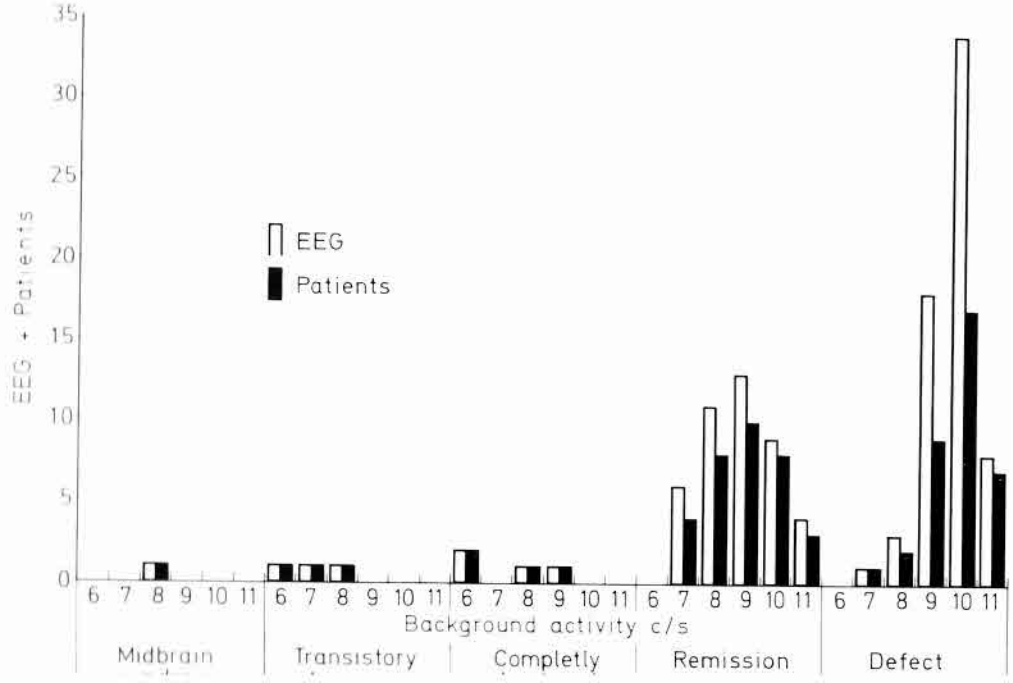


Fig. 3. EEG record of patient with traumatic apallic syndrome. *(A)* Transition stage. *(B)* Completely developed apallic syndrome. *(C)* Defect stage

When correlating the individual EEGs with the different clinical stages for the whole patient group, it must be taken into account that there are relatively few records available from the first three stages, and that for the remission stage and defect stage of some of the patients several EEGs were evaluated which showed disturbances of varying severity. An alteration in the EEG patterns is to be expected, especially in the course of remission, because in this often very long-lasting stage a considerable improvement in the neurologic picture often may take place. In Figure 4, the degree of the generalized changes (for definition, see *Materials and Methods* above) in the 119 EEGs from the 23 patients is correlated with the clinical stages. The only recording made in this group of patients during the acute midbrain syndrome shows only mild generalized changes. This finding has been re-

Fig. 4. Degree of severity of generalized changes in EEG in the individual clinical stages of 23 patients with traumatic apallic syndromes. For abbrevations see Figure 1





traumatic apallic syndrome moderate and severe generalized changes predominate. In the remission stage moderate and mild generalized changes predominate; only in rare cases one may see severe generalized changes. In the defect stage, on the other hand, we have not observed any severe generalized changes; a high percentage of patients show mild to just perceivable generalized changes, some patients show moderate abnormalities or even a normal EEG.

A quite similar development is found by correlating background activity with the clinical stages (Fig. 5). In the acute midbrain syndrome, in the transition stage, and in the completely developed traumatic apallic syndrome, the basic activity has a frequency of 6-9/s, in the remission stage mainly 8-10/s, and in the defect stage 10/s. Sometimes a normal curve with well-pronounced basic alpha activity with a normal distribution and without perceivable generalized changes is observed even before the onset of the remission stage (Fig. 6).

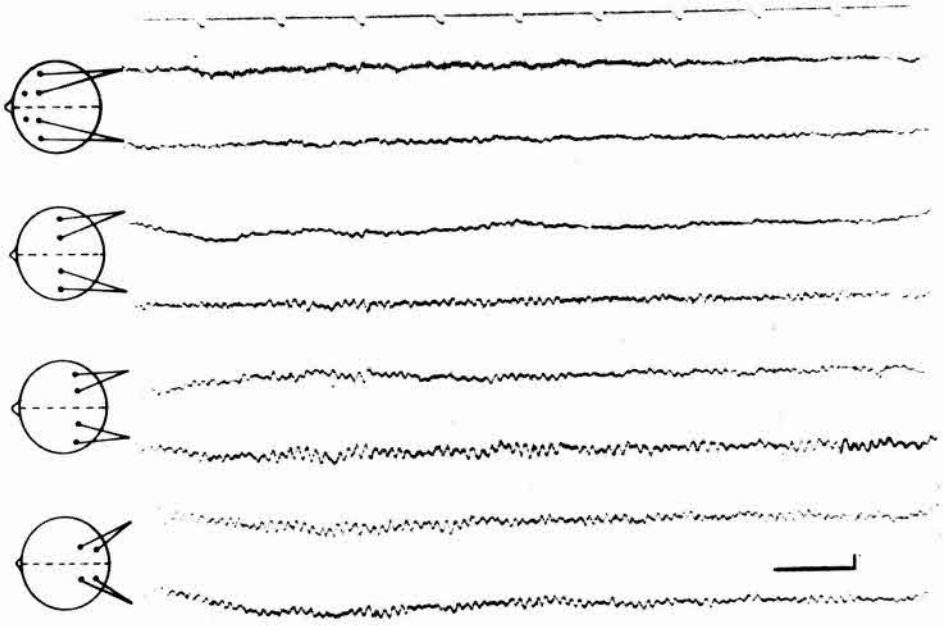


Fig. 6. EEG record of patient in remission stage of traumatic apallic syndrome 4 weeks after craniocerebral trauma; well-pronounced basic alpha activity

In addition to generalized changes and background activity the appearance of focal alterations and of irritative or epileptic phenomena have been analyzed. The focal alterations were, according to the amount of focal theta and delta activity, classified as mild and severe, and according to the site of trauma, in homo- or contralateral foci and correlated with the individual clinical stages. Bitemporal foci were taken into account separately. Table 2 demonstrates this classification in detail. It shows a significant predominance of

severe contralateral foci (eight compared with the three patients), but during this time severe and mild homolateral foci are observed more frequently. Figure 7 shows a slight focus over the left posterior quadrant in one patient at the beginning of the defect stage *(A)*, 7 months after the accident with a right frontal trauma. Two years and 3 months later *(B)*, still in an unaltered defect stage, the focus can be seen more clearly and has expanded more precentrotemporally. Bitemporal foci are found relatively seldom; they can manifest themselves only in the late course.

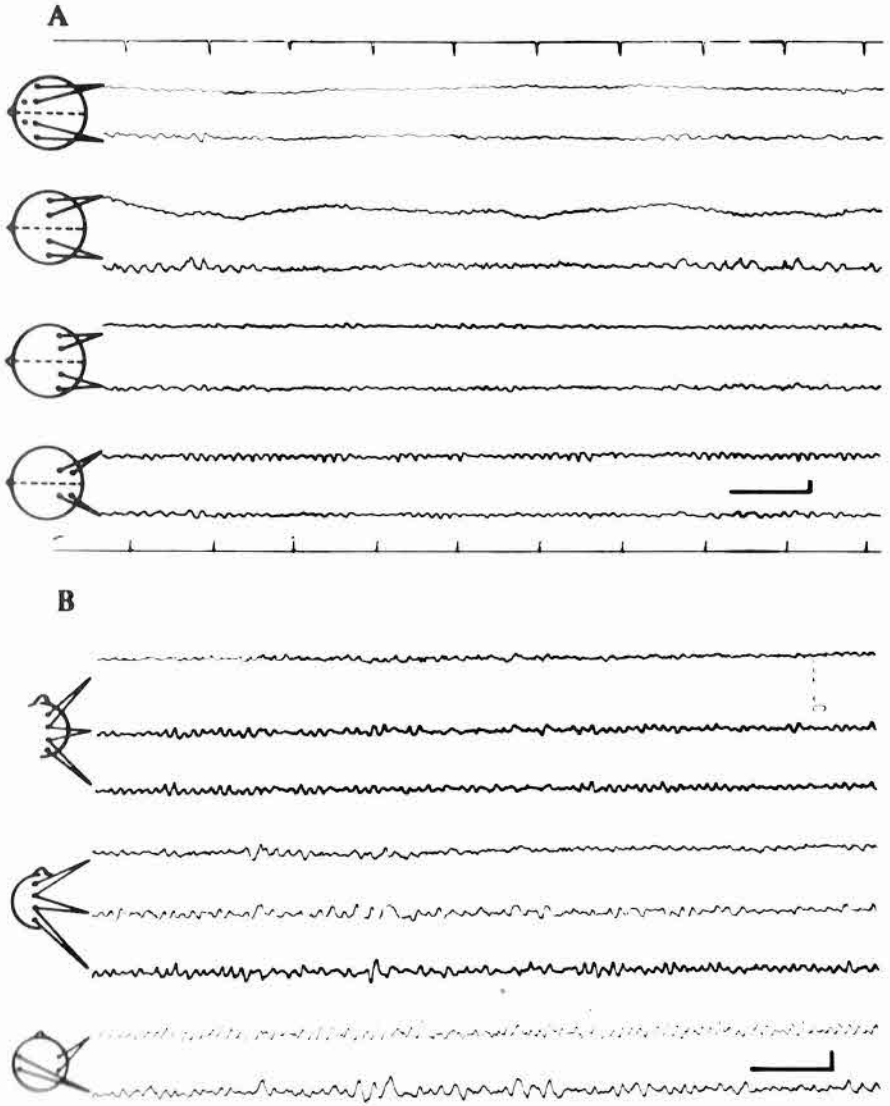


Fig. 7. EEG record of a patient at beginning of defect stage *(A)* and 2 years and 3 months

Table 2. Degree of severity and localization (in relation to the site of the trauma) of foci and appearance of irritative or epileptic phenomena in EEG during individual stages in 23 patients with traumatic apallic syndrome.

Clinical stage		Midbrain syndrome	Transition stage	Completely developed TAS	Remission stage	Defect stage
Number of EEG/ patients		1/1	3/3	4/4	45/22	65/23
Homolateral focus	severe	1/1	–	–	4/4	8/5
	mild	–	1/1	–	1/1	10/4
Contralateral focus	severe	–	1/1	2/2	8/8	3/3
	mild	–	–	–	17/12	31/12
Bitemporal focus		–	–	–	4/3	10/6
Irritative phenomena		–	–	1/1	4/3	8/6
Epileptic phenomena		–	–	–	3/2	10/5

Number of EEGs in relation to the number of patients is indicated EEG/patient)

A number of patients (Table 2) showed irritative or epileptic phenomena. In the completely developed traumatic apallic syndrome irritative phenomena were observed in one out of four patients. In three patients these patterns were observed in the remission stage, in three others only in the defect stage. Epileptic activity was observed in two patients in the remission stage, in five patients in the defect stage. During the remission stage one patient showed isolated spikes above the left centroparietal region immediately after photostimulation, which were found to exist unaltered in the defect stage more than 2 1/2 years later. Clinically this patient had never had an epileptic seizure. The second patient who showed epileptic activity in the remission stage had suffered from one jacksonian attack a few days after the accident. Another patient had a jacksonian attack during the defect stage 3 years after the accident and then showed isolated epileptic activity for the first time. Focal seizures were reported in three other patients without irritative or epileptic phenomena having been observed in former or later EEGs.

Discussion

This is a retrospective study. In order to be able to investigate an etiologically uniform patient group, only the traumatic apallic syndrome was taken into account, which developed in all cases as a result of tentorial herniation. With respect to possible prognostic criteria, patients were mainly chosen who presented a favorable clinical course and had been followed up with exact clinical and electroencephalographic examinations for several years (in most cases, 2-5 years). However, only a few EEGs were available from the early phases of the disease (midbrain syndrome, transition stages, and completely developed apallic syndrome). Chatrian et al. (1963) and Lücking (1970b, 1972) reported on the prognostic value of EEGs recorded in an early stage after a severe craniocerebral trau-

allows relevant prognostic statements even at that early stage. It must be taken into account, however, that in spite of an initially favorable electroencephalographic and cerebral development death can occur due to extracerebral complications.

The EEG findings from several months of observing apallic patients with mainly lethal courses are reported by Jellinger et al. (1963). During the first few weeks most severe generalized changes including a flattening of the curve without any reactivity to external stimuli were observed, and in the following months (during the completely developed apallic syndrome) a predominating theta activity with interpositon of delta waves without alpha activity was observed. In the late phase (5-6 months after trauma) further marked generalized changes with a low index of alpha waves and an incomplete blocking effect after stimulation were observed. Favorable clinical courses were associated with a faster regression of the theta waves and an early reinstitution of alpha activity. Focal signs and lateral differences were rarely hints of an increased susceptibility to attack. It is general experience that the degree of deficit in consciousness and the degree of EEG slowing, as well as the regression of the EEG changes and the recovery of consciousness, are parallel phenomena (Gastaut, 1954; Fischgold and Mathis, 1959). This observation generally refers to the suppression of consciousness during coma in which both wakefulness and conscious activity are extinguished. A separate disturbance of wakefulness and consciousness as manifested in the coma vigile of the apallic syndrome is thereby not taken into account.

The present investigation of apallic patients with an exact clinical and electroencephalographic examination of the remission stages confirms the observation which is known from other comatose states: In general, clinical improvement is associated with an acceleration in EEG activity. A close correlation between wakefulness and EEG changes, however, is not seen in any of the cases. In spite of increasing wakefulness, in the coma vigile the theta and delta waves frequently still constitute the predominating EEG activity. Only with the onset of conscious activity is it gradually accelerated and can, even after many years when no more appreciable clinical remission has taken place, further alternate with an increase in alpha activity and a regression of focal symptoms (Figs. 1 and 2). This is not surprising, since the EEG reflects the neuronal activity of the cerebral cortex to which are bound consciousness and the higher cerebral functions, but not wakefulness. Since an alpha activity may quite well predominate in the coma vigile stage (Fig. 6), there is no strong interdependence between conscious activity and rapid EEG activity. An alpha EEG may also be found in the case of suppression of both wakefulness *and* consciousness (Loeb et al., 1959; Lücking, 1972). Since the neuronal activity of the cortex and thus of the EEG is dependent on the intactness of the cortex as well as of the subcortical structures, different damage patterns of the brain and, at the same time, different clinical conditions may be associated with an identical EEG. While anoxia mainly results in cortical damage, a diffuse brain edema (posttraumatic or metabolic) rather produces damage to the cerebral white matter and, as a result of tentorial herniation, a lesion in the mesodiencephalic region. The prognostic value of the EEG in the apallic syndrome must also be seen under the following aspect: Predominating theta-delta activity which persists largely unchanged for several months during the transition to the clinical remission stage points to a significant lesion of the hemispheres. An increasing alpha activity already appearing at the beginning of remission suggests a slighter cortical involvement and a stronger subcortical lesion. Taking clinical symptomatology and development simultaneously into account, the EEG may contribute

of relatively minor importance. An acceleration of alpha frequency within the range of 9-11/s, for example, still occurs after many years (Fig. 2) without any further clinical improvement. Focal symptoms, contralateral as well as homolateral to the site of trauma, can often be recognized only after regression of the severe general changes, and in the course of normalization above the other regions it often appears as if these signs were increasing (Fig. 7). Temporal foci often show a jumping from one side to the other and predominate in the defect stage on the side of the trauma. Only in relatively few patients are constant bitemporal foci found, which can be expected as a result of tentorial herniation of the mediobasal components of the temporal lobe. Epileptic phenomena as a result of increased cerebral susceptibility to attacks may be important only in connection with the clinical findings: From among five patients with multiply evidenced spike potentials, only two patients had suffered from one jacksonian attack and only one patient was under anticonvulsive medication. On the other hand, in three patients who had suffered from focal attacks, epileptic activity of the EEG could not be verified at any time.

Summary

The investigation involves 23 patients who developed a traumatic apallic syndrome after severe craniocerebral trauma. All patients showed a favorable course with regression of the apallic syndrome to a defect stage of differing severity. The observation time was from 4 to 6 years with regularly performed clinical examinations and repeated EEGs. Altogether, 119 EEGs were evaluated. The correlation of EEG findings with the clinical development of the apallic syndrome showed considerable individual variability in this group of patients. The degree and regression of the EEG changes were correlated with the reinstitution of conscious activity and the higher brain functions rather than with the degree of wakefulness. Severe and moderate generalized changes which persist unaltered throughout the first months, point to a lasting functional damage of the cortex. On the other hand a reappearance of alpha activity already present in the coma vigile or early remission stage points to only slight involvement of the cortex without allowing definite conclusions regarding possible lesions of the subcortical structures and the brain stem. Prognostic statements are possible only within these limitations. Focal symptoms and lateral differences reflect local damage without a strong relation to the site of the trauma. Even with consistent irritative and epileptic phenomena, the development of cerebral attacks is rarely observed.

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