

Systematic EEG Follow-up Study of Traumatic Psychosis

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Summary. A systematic 2 year follow-up study of EEG in 100 patients suffering from traumatic psychosis with amnesia lasting more than 1 week led to the following results. (1) EEG foci were demonstrated in 95% of cases, and bilaterally in 70%. Normalization occurred within 3 months in 48% of patients, with foci persisting for more than 2 years in 22% mostly in patients with traumatic epilepsy. Focal signs initially consisted of delta foci (85%) and finally of focal dysrhythmia (72%), with temporal localization increasing from 58% to 82%. EEG foci were associated with neurological focal symptoms in 49% of cases and skull fractures in 78%. (2) During psychosis a general slowing of EEG was constantly observed. Normalization occurred within 3 months in 28% of patients. Rarely slowing lasted longer than 6 months. (3) It took longer to normalize general slowing than EEG foci, but slowing disappeared more completely. The left predponderance of EEG foci in traumatic psychosis could not be confirmed, the hypothesis of a pathoplastic role of the speech dominant hemisphere was not proved.

Key words: Traumatic psychosis – EEG follow-up study – Foci – General slowing

Introduction

A former retrospective study of EEG in traumatic psychosis by Schneider and Hubach indicated a preponderance of left-sided EEG foci. This led to the assumption that injury of the dominant hemisphere occurred more frequently than lesions of the nondominant hemisphere in traumatic psychosis [25] and

could be considered a pathoplastic factor for the manifestation of the psychosis [13]. On the other hand we had noticed in a follow-up study of unselected recent head injuries that traumatic psychosis initially presented bilateral foci in most cases [15]; however, only a few cases were examined. The present study, a continuous assessment of all traumatic psychoses in our cerebral trauma ward, intended to investigate this observation in more cases. At the same time the significance of EEG foci for the manifestation of psychosis as well as regularity and development of concomitant and ensuing changes in EEG pattern in general were studied.

Materials and methods

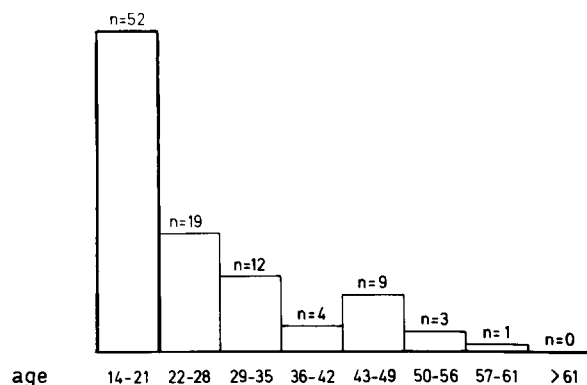
The study included, after excluding cerebral dysfunction independent of trauma, all patients up to 65 years of age who presented amnesia due to traumatically caused disturbance of consciousness lasting for longer than the 1st week, and in whose cases it was possible to record the EEG during psychosis within the 1st months and after the 1st year. As a rule, the follow-up recording of patients took place 2 years after the trauma. We followed a quarter of the cases for up to 7 years.

Between January 1975 and November 1980, 100 patients fulfilled these criteria. Table 1 shows the time of the first EEG recording. In total 1004 EEG tracings were made on the 100 patients, and all of them were finally evaluated within a uniform framework. The recordings were made using the Siemens Mingograph on 8 channels with 16 positions of electrodes in 8 programs uni- and bipolarly after Jung's schema. The characterization of the general and local EEG changes also followed Jung [12]. EEGs that showed only intermittent delta wave dominance were termed alternately severely generally changed EEGs.

The patients were between 14 and 61 years old (94 men, 6 women). Half of them were 21 years old or younger (see Fig. 1). The comparatively low proportion of women was due to our ward's conditions of admission. Among traffic accidents, 67 occurred with, and 4 without motor vehicle involve-

Table 1. Time of the first EEG recording

Day after trauma	1st	2nd	3rd–7th	8th–30th
Number of patients	21	15	34	30

**Fig. 1.** Age range of 100 traumatic psychoses

ment. In 21 cases trauma was caused by falls from varying heights, in 7 cases by violent contact with a solid object, and in 1 case it was a missile wound. The duration of amnesia, discovered by systematic enquiry, was used as a scale to assess the severity of the injury.

Clinical Data

Most of the traumatic psychoses presented the classical sequence of coma, delirium, and Korsakow. In the other cases simple lack of drive in combination with varying, sometimes purely euphoric, changes of affect were observed in place of delirious agitation. Duration of psychosis, measured by duration of amnesia, extended from the 6th week to a maximum of 8 months in 27% of the cases (Table 2).

Severity of injury was emphasized by the presence of skull fractures in 67% of cases, and the existence of epidural and subdural hematoma, each in 18% of the cases (23 right, 13 left). Interestingly, there was no evidence of fracture in a quarter of the cases of hematoma.

Initially, neurological focal symptoms could be observed unilaterally in 58 cases (32 left, 26 right), and bilaterally in 12 cases. Two patients exclusively suffered from complete anosmia. There was aphasia in 13 cases, each one accompanied by motor deficiencies. In 28 cases there was no neurological indication as to the hemisphere.

EEG Foci

Only 5 of the 100 patients had no EEG foci. Unilateral foci were found 25 times (12 left, 13 right). In 70

Table 2. Duration of amnesia in 100 traumatic psychoses

Days	7–14	15–18	29–42	>42
Number of patients	16	30	27	27

Table 3. Period during which normalization of EEG foci could be demonstrated

1st month	17	48%
2nd month	43	
3rd month	20	
4th–6th month	15	9%
>6th month	34	21%
Persisting	36	22%

cases there were bilateral foci; 19 of these initially showed unilateral foci, and 7 none at all.

At the end of the study, 34 out of 95 patients (36%) had remaining foci, with 2 patients having bilateral foci. The unilateral ones were equally divided between the hemispheres (18 left, 18 right). With reference to the total number of foci, 22% (36 out of 165) had not normalized. Their degree of severity was minimal, and only 7 of 36 foci were more clearly emphasized. These 7 patients had experienced particularly severe trauma with amnesia lasting longer than 6 weeks, or with complications such as epilepsy or hydrocephalus malresorptivus. Persisting EEG foci were the rule in traumatic epilepsy (14 out of 15 cases). Almost half of all persisting foci were concomitant with traumatic epilepsy. In almost half of the cases the foci had normalized within 3 months (Table 3).

Normalization of foci after the end of the 1st year could only be demonstrated in 6 cases (4 times in the 2nd, twice in the 3rd year after trauma). In another 21 cases we cannot rule out the possibility that normalization occurred after the end the 1st year following the trauma, because of the varying intervals between the follow-up tracings.

As far as the type of foci was concerned, there was a preponderance of delta waves in the first recordings in 141 cases (85%). Focal dysrhythmias with occasional occurrences of delta waves could mainly be found when the recordings were made after the 1st week following the trauma (16 out of 23 cases with focal dysrhythmia). Alpha reduction only occurred once as an initial finding. This was a complicated case with a subdural hematoma; the initial finding of alpha reduction was still observed in several follow-up recordings, even after evacuation of the hematoma.

The last recordings showed the following types of foci in the 115 cases of normalization that had been

Table 4. Types of foci before normalization (within brackets those foci that had not normalized)

Delata focus	14	(0)
Focal dysrhythmia	88	(26)
Alpha activation	1	(7)
Alpha reduction	9	(1)
Focal seizure patterns	3	(2)

Table 5. Localization of foci

	Initial recording (165 foci)	Residual recording (148 foci that had been registered more than once)
Frontoprecentral	35 (21%)	15 (10%)
Occipitoparietal	19 (12%)	10 (7%)
Exclusively temporal and basal	59 (36%)	88 (66%)
Temporal region with extension anterior and/or posterior	37 (22%)	34 (23%)
Complete hemisphere	15 (9%)	1

Table 6. Hemispheric coincidence between skull fractures ($n = 67$) and EEG foci

Positive correlation	52 (78%)
Negative correlation	13 (19%)
No EEG focus	2 (3%)

Table 7. EEG findings during the first week (within brackets number of cases in which the initial recording took place between the 8th and 30th day)

Mild general slowing	13 (+3)
Moderate general slowing	17 (+14)
Alternatingly severe general slowing	12 (+4)
Severe general slowing	20 (+9)
Abnormal sleep EEG	8 (—)

recorded more than once in the course of the study, and in those 36 cases that had not normalized (Table 4). Foci with preponderance of delta waves were observed as a residual finding at the latest on the 35th day. Focal dysrhythmia predominated in persisting foci (72%). Alpha activation (19%) was observed only in the course of further developments. In 3 out of the 8 cases with alpha activation traumatic epilepsy had occurred; in another case there was a traumatic cerebral insult. Focal seizure patterns were found residually in 5 cases, but only in 2 out of 15 cases with traumatic epilepsy.

Table 5 shows localization of foci in the initial and residual recordings. It appears that in the course of time concentration in the temporal region increased in frequency, and concentration in the other regions decreased. Concentration in the temporal region occurred with almost equal frequency from anterior (42 times) and posterior (44 times) regions of the cerebrum. While 58% of the foci included the temporal region in the initial recording, this was the case in 82% of the foci in the residual recording. The residual finding of unilateral changes in a complete hemisphere was found on the 26th day; a follow-up recording on the 77th day showed no remaining focus. There was no substantial difference with regard to localization between normalized and persisting foci.

In 60% of the unilateral EEG foci there were ipsilateral neurological focal symptoms: there was no case with contralateral neurological focal symptoms. In the case of bilateral foci with preponderance in one hemisphere, neurological findings still coincided with this hemisphere in 45% of cases, but indicated the other hemisphere in 16% of the cases. Neurological clinical symptoms that indicated that there was a cerebral contusion were anosmia in 1 patient, and discreet hemilateral neurological symptoms in another case of the 5 psychoses without EEG foci.

Table 6 shows the percentage of coincidence between fractures and EEG foci. When cases with epidural and subdural hematoma were excluded, the rates of positive correlation (77%) and negative correlation (23%) were equally high. Only 2 cases with fractures and subdural or epidural hematoma showed no EEG foci. In addition, 3 cases with bilateral fractures presented only unilateral EEG foci.

General Slowings

None of the 70 patients whose EEG was recorded during the 1st week of psychosis presented normal alpha waves; they all showed general slowing. In 8 cases these were preceded by an abnormal sleep EEG with sleep spindles that could not be interrupted by arousing stimuli. Also, temporary sleep spindles were found in 10 cases of severe general slowings. The distribution of the findings can be seen in Table 7.

The follow-up check showed an increase in general slowing beyond the initial finding for 20 out of the 70 patients, in 10 cases with initially only mild general slowing. If these increasing general slowings and the general slowings following abnormal sleep patterns (severe general slowings in 6 cases, moderate general slowing in 2 cases) were included, and the initial EEG was recorded during the 1st week, the

Table 8. Period during which normalization of traumatically caused slowing of alpha rhythm was demonstrated

1st month	4	28%
2nd month	6	
3rd month	9	
4th–6th month	25	37%
7th–12th month	18	27%
After >12th month	5	8%

following distribution appeared: 3 cases that did not develop beyond mild general slowing, 23 cases of moderate general slowing, 8 cases of alternately severe general slowing. The remaining 36 cases showed severe general slowings at different times during the psychosis; i.e., in 51% of the cases severe general slowings were observed during psychosis. Mild general slowings were thus extremely rare under the conditions mentioned and were only found in psychoses with a duration of amnesia of less than 2 weeks. On the other hand, these psychoses of short duration did not show abnormal sleep patterns. In 7 cases, abnormal sleep patterns were observed exclusively until 6th day. In the 8th case they were present on the 1st day, and again after deterioration of the state of consciousness on the 11th day. The tendency of severe general slowing to increase with duration of psychosis (in 42% of the cases where the amnesia lasted up to 2 weeks, in 55% of the cases where the amnesia lasted for more than 6 weeks) was not significant.

As a rule, general slowing could be demonstrated at least as a slowing of alpha waves beyond the end of the psychosis. There were only 3 exceptions out of our 100 cases to this rule. These 3 were cases of psychosis lasting longer than the average, with amnesia of more than 50 days. In 1 of them a mild general slowing persisted constantly up to 6 years after trauma; a persisting pretraumatic abnormality could not be excluded. On the whole, 12 out of our 100 cases showed general slowing at the final recording, 4 times as mild general slowing, and 8 times as a slowing of the alpha rhythm to 8–8.5/s. Since traumatic epilepsy had occurred in 4 of these cases, only 8 cases remain that showed persisting abnormality of the alpha rhythm in spite of an uncomplicated development. The final mean level of frequency of 9.98/s out of all 100 cases changed to 10.14/s if the 15 cases with traumatic epilepsy and their tendency towards slowing due to anticonvulsive medical treatment were omitted.

The period during which normalization of the alpha rhythm could be demonstrated is shown in Table 8. Because of lack of intermediate recordings we can say little about during what period normalization took place in the remaining cases. Excluding the

cases with anticonvulsive medication, only 2 remain in which it can be stated with certainty that normalization occurred only between the 6th and the 12th month. It is therefore possible to conclude that normalization of traumatically caused general slowing of the alpha rhythm, initially occurring at a slower rate than normalization of foci, generally terminated within the first 6 months, even in cases of severe cerebral trauma with traumatic psychoses. Most of the normalizations occurred between the 4th and the 6th month.

Discussion

The results do not support the hypothesis of a pathologic significance of left-sided EEG foci for the development of traumatic psychosis [13, 25]. A preponderance of left-sided foci in traumatic psychosis could also not be found in those cases with unilateral foci, as had already been shown in studies with a smaller number of cases and with later initial recordings by Reckel [28] and Flügel [7]. Objections to the interpretation of the preponderance of left EEG foci in traumatic psychoses as described by Schneider and Hubach [31] had already been confirmed by the fact that a preponderance of left EEG foci could also be ascertained in the continuous assessment of head injuries of varying degrees of severity [8, 15, 30]. As the same phenomenon had been known to occur in temporal lobe epilepsy [13] and cerebral vascular lesions [2, 10, 32], we pursued the question of whether the preponderance of left EEG foci could be considered a general phenomenon [17], using extensive material on EEG foci registered continuously and independently of the diagnosis. It appeared that the left side predominates in all states of disease with concomitant changes of foci, although in varying frequency. The dependence on age described by Helmchen et al. [10] could only be confirmed for the first decade. According to our observations, traumatic psychoses do not show the general pattern of left-sided preponderance because in the majority of cases they are concomitant with bilateral EEG foci. The discrepancy with the findings of Schneider and Hubach [31] could be explained by the larger basis for conclusions, as developments were assessed systematically beyond the stage of predominant delta wave activity. It was thus possible to assess even transitory or sporadic side differences, consisting partly of leftsided alpha reduction, by using follow-up recordings to confirm such a focus in addition to strict adherence to the criteria stated by Jung [12].

Alpha reduction considered typical for persisting remaining foci after cerebral contusions is [9, 11, 13,

20, 27, 31], according to our observations [16] a rather rare phenomenon compared to focal dysrhythmias. At any rate, alpha reductions can only be related to cerebral injury with the required certainty if they are present as focal slowing in the early phase, as has also been indicated elsewhere [4, 27]. The different evaluation of alpha reduction could, in addition, influence statements about greater frequency of persisting foci after cerebral injury in general [9, 11, 21, 22, 29, 31], without detriment to Jung's estimate [13] that foci remaining after traumatic psychosis are more frequent than in cases of cerebral contusion without psychosis, which was confirmed by our study. This, however, does not seem to be true in the majority of cases [26].

EEG foci during traumatic psychosis indicate the necessary severity of injury for the manifestation of psychosis, because of their frequent bilateral occurrence. At a later stage they are interesting because they almost always accompany the development of traumatic epilepsy. Estimation, however, is uncertain without knowledge of the EEG of the acute phase and of the further development [14]. The concentration of EEG foci in the temporal region in the later stages as described by Schneider and Hubach [31] was confirmed as a tendency in our study. However, the qualification has to be made that this is not a specific property of cerebral contusions, as more foci are located in the temporal region than in the other areas, both generally and with increasing age [17].

As the CT scans show, examinations within the first 10 days in the case of traumatic psychoses almost regularly demonstrate cerebral edema. Faust's opinion [6], subsequent to pneumoencephalographic examinations that cerebral edema is the initial precondition for the development of traumatic psychosis, which has been questioned [24], is thereby supported. As shown in our study, traumatic cerebral edema regularly occurs with clear general slowing that can increase with increasing edema, and outlasts psychosis as a slowing of alpha rhythm by months and not infrequently up to half a year. In some cases of longer lasting psychoses these general slowings are preceded by an abnormal sleep EEG in the stage of coma or clouding of consciousness that Courjon [5] had described as a diffuse 12–14/s activity against a background of low voltage and corresponds to a pathological sleep that cannot be interrupted by arousal stimuli. An accompanying functional disorder of the formative reticularis that could be expected [3, 19] has been demonstrated in several cases [1, 19, 33]. Systematic investigation of posttraumatic comata shows that a spindle coma EEG is by no means rare [18].

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