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REVIEW

Behavioral Aspects of Frontal Lobe Epilepsy

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There is growing interest in disorders of behavior, personality, and mood associated with focal epilepsies, though the neuropsychological and behavioral or psychiatric aspects of epilepsy have usually been treated separately. The causes of behavioral disorders in patients with focal epilepsies are multifactorial, though the positive effects of seizure control on behavior suggest that state dependency is a major contributing factor. Patients with temporal lobe epilepsy manifest depression, anxiety, neuroticism, and social limitations, as well as impaired memory. By contrast, studies of cognitive function in patients with frontal lobe epilepsy show executive dysfunctions in response selection/initiation and inhibition, as well as cognitive impairment, hyperactivity, conscientiousness, obsession, and addictive behaviors. © 2001 Academic Press

Key Words: **frontal lobe epilepsy; cognition; seizure semiology; behavior; personality; quality of life.**

To date, neurobiologists interested in behavior and epilepsy have focused primarily on temporal lobe epilepsy (TLE) and mesial temporal lobe epilepsy (mTLE), in particular, mostly because TLE accounts for the majority of patients with focal epilepsies seen at epilepsy referral centers. For example, in the Bonn series of patients with pharmacoresistant epilepsies, approximately 80% had TLE. Another reason is that patients with mTLE often constitute a well-defined cohort with respect to underlying pathology (hippocampal sclerosis), a frequent history of febrile convulsions, an early onset of epilepsy, and memory problems as the prominent neuropsychological impairment. Further, the affected cerebral structures and epileptogenic regions associated with TLE are usually circumscribed, and the structural pathology can be readily characterized by quantitative MRI (T2 relaxometry and volumetry) or histopathological examinations of resected specimens.

Thus, frequency of occurrence, homogeneity of phenotypic expression, and well circumscribed and quantifiable pathology provide ideal prerequisites for the study of the functional and behavioral correlates of TLE. Accordingly, great progress has been made in recent years with respect to the neuropsychological and cognitive aspects of TLE. Additionally, studies have led to a rediscovery of the role of the temporal lobes in emotion and psychiatric symptoms.

The conditions for studying frontal lobe epilepsy (FLE) are quite different. FLE, despite the size of the frontal lobes, is less frequent than TLE. In our own series, patients with FLE represent about 15% of the patients with pharmacoresistant epilepsies. Further, the site and type of the underlying pathology are very heterogeneous. Finally, ictal and interictal clinicoelectric manifestations of FLE are infrequently localizable because multiple connections to most other brain areas enable fast and widely distributed propagation of epileptic activity. The functional correlates of frontal pathology in epilepsy are thus less well understood.

FRONTAL LOBE: ANATOMY, FUNCTION, AND ASSOCIATED BEHAVIOR DISORDERS

Based on its cytoarchitectonic structure, the frontal lobe is traditionally divided into two parts, each with

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important functional characteristics. The posterior part controls motor movement and is subdivided into a premotor area and a motor area, which control preparation for movement and actual execution of movement, respectively. The anterior part, the prefrontal cortex, is especially important for higher mental functions, such as anticipation and planning, initiative, judgment, and in the control of mood, will power, and the determination of personality (1, 2). The prefrontal cortex can be further subdivided into the dorsolateral cortex and the orbitofrontal cortex, though this is simplistic because the orbitofrontal cortex is a heterogeneous area connected with a wide range of other prefrontal, limbic, premotor, sensory areas in addition to subcortical nuclei (3).

Damage of the dorsolateral prefrontal cortex is typically associated with impairment of executive functions and working memory, whereas damage of the orbitofrontal cortex leads to impairment of the choice of behavior, the establishment of emotional valences, and the evaluation and balancing of the past and future consequences of a given behavior (4–6). Studies in common marmosets suggest a dissociation between the lateral and the orbital medial divisions of the prefrontal cortex according to which the former selects and controls actions on the basis of higher-order rules and the latter controls different behavior on the basis of lower-order rules (7). The significance of the orbitofrontal cortex for social and interpersonal behavior in humans was again demonstrated by the recent report of two patients, one with a traumatic brain injury at 15 months of age and the other with a frontal tumor resected at 16 months of age, who both showed severe impairment of social and moral behavior (8).

Traditionally, behavioral dyscontrol in epilepsy has been attributed to dysfunction of temporolimbic structures. Evidence for the involvement of the amygdala in aggression comes from human and animal stimulation studies, from the effects of antiepileptic drugs on activating and inhibiting aggression, and, recently, from direct correlations of amygdala volumes with aggression in patients with mesial epilepsy (9–12). Aggression associated with involvement of the amygdala appears to be defensive rather than offensive in nature (13).

Disinhibition phenomena or a loss of impulse control as observed in patients with frontal lesions suggests the importance of frontal regions in the genesis of impulsive aggressive behavior. The orbitofrontal cortex, as the border zone between the frontal lobe and the limbic system, links the frontal and limbic components involved in disorders of behavioral control. The anterior cingulate gyrus, which is also strongly connected to the amygdala, has also been associated with deviant social behavior and pathological affective states when damaged (14).

Attributing antisocial and aggressive behavior to frontal lobe damage is not new. A prominent and often-cited example is the historic case of Phineas Gage, who after an accident with a severe frontal brain injury changed from a well-behaved man into an irresponsible and convention-neglecting person (15, 16). One new concept, brought out by the case reports of Anderson *et al.* (8), is that patients with frontal lobe damage not only may display severe behavioral disorders but also may neglect the moral aspects of their behavior, depending on the age at which the damage occurred (17). Consequently, the orbitofrontal cortex seems important both for behavior control and for the acquisition of social and interpersonal rules of conduct. It is important to note that irresponsible, aggressive, and sociopathic behaviors can occur irrespective of intellectual abilities, which are often well preserved in frontal lesions.

The orbitofrontal cortex and medial prefrontal cortex are believed to play a central role in addictive behavior, attention deficit–hyperactivity disorder, and negative emotion and major depression, respectively (18–21). Davidson and co-workers further propose a key role of the prefrontal cortex in the regulation of emotion in violent subjects and those predisposed to violence (22).

Damasio proposed the "somatic marker" hypothesis as a theoretical basis for the role of the prefrontal cortex in the interplay of cognition and emotion (23). This hypothesis posits that responses to external stimuli do not rely on either conditioning processes or cognition alone, but on somatic "marker signals" or autonomic response sets, which determine the conscious/unconscious connection between stimulus conditions, feelings, and behavior.

EPILEPSY AND ASSOCIATED BEHAVIOR DISORDERS

Case reports of behavioral and personality disorders in patients with severe brain lesions raise the question of whether there might be parallels in the behavior of patients with seizures arising from the same brain regions. With the exception of rare cases of ictal aggression, postictal confusional states, or psychosis (24), behavior and personality disorders observed in patients with frontal lobe epilepsy appear to

TABLE 1
Factors Affecting Cognitive and Mood States in Epilepsy

States of epilepsy	Preictal
	Ictal
	Postictal
	Interictal (seizure free after successful surgery)
Seizures	Frequency
	Generalization
	Nonconvulsive status epilepticus
Epileptic dysfunction	Local versus distant effects
Lesion	For example, alien tissues versus migration and developmental disorders (confounded with different ages at lesion/epilepsy onset)
	Extent, location, lateralization
AED	Positive versus negative psychotrophic effects
	Individual incompatibility
	Drug-induced encephalopathy
	Intoxication

be less dramatic than found in the published case reports. Furthermore, as with TLE, one can hardly expect to find the prototypical "frontal epileptic personality" or "Wesensänderung," respectively. Personality is by definition more trait than state dependent. In patients with epilepsy, it is particularly difficult to determine whether a given behavior has trait characteristics or not. Concluding that abnormal behaviors have persisted requires follow-up observations over long time intervals, which generally are not reported in the literature. Several epilepsy-related variables can account for reversible changes in cognitive abilities and mood states (see Table 1). Finally, despite the long history of patients becoming seizure free from epilepsy surgery, it is still not clear whether seizures are necessary for the development of epilepsy-related behavior and mood disorders. Behavior in patients with epilepsy should be defined by state as it relates temporally to seizure events, e.g., ictal, postictal, or interictal. Recent findings with regard to seizure prediction by nonlinear measures of complexity loss as recorded by intracranial EEG suggest that the preictal period should also be considered (25). Accordingly, patients may report a prodrome consisting of increased dysphoric mood and cognitive problems well before their seizures begin. Finally, the observation of behavior problems following successful treatment with epilepsy surgery or antiepileptic drugs implies that an additional state that should be evaluated is that of seizure freedom associated with treatment.

Epileptic activity can affect brain areas distant from the epileptogenic zone, causing cognitive and behavior problems (26). Notwithstanding seizures and interictal epileptic activity one must also differentiate the underlying pathology(ies) and when they occurred (which could influence effects on brain maturation and the development of cognition and personality). We must also consider the effects of chronic antiepileptic medications, which may have positive or negative psychotropic side effects (27). Antiepileptic drugs can have different effects in patients with lesional epilepsy compared with nonlesional patients, and they may act differently dependent on seizure control (28). Thus, the effects of underlying pathology, seizures, and pharmacological treatment must be considered individually and as they may interact in a given patient.

FRONTAL LOBE EPILEPSY: NEUROPSYCHOLOGY AND EVIDENCE OF SPECIFIC BEHAVIORAL DISORDERS

The development of neuropsychology in frontal lobe epilepsy is probably best reflected by Brenda Milner's description of her evaluation of Penfield's patient K.M., the frontal counterpart of the temporal patient H.M. This patient had a penetrating head injury in 1928, developed seizures, and underwent surgery of the anterior parts of both frontal lobes. Surgery successfully controlled the seizures and led to improved behavior as well as improved IQ. However, when reevaluated with the newly developed Wisconsin Card Sorting test in 1962 he showed severe impairment in flexible categorical thinking and concept formation while the IQ still was average (see Milner (29)). This case exemplifies how much outcome interpretation depends on test sensitivity and test selection.

Since that time surprisingly few attempts have been made to comprehend the cognitive characteristics of patients with frontal lobe epilepsy in group studies (26). Unfortunately, most data from Milner's era stem from operated patients and thus tell us more about frontal lobe lesions than about frontal lobe epilepsy. Furthermore, most earlier studies focused on single functions more or less following a rather monistic view of a frontal "central executive" (30). Major impairments indicated by these studies are problems in concept formation, response inhibition (31), estimations (32), conditional associative learning (33, 34), and profit from information provided in advance in choice reaction tasks (35). Focusing on memory Delaney *et al.*

found no differences in measures of memory when nonoperated patients with unilateral frontal lobe foci were compared with healthy controls (36). The first of our own studies found that deficits in attention are the most significant problem in patients with frontal lobe epilepsy (37).

Later systematic group studies in nonresected patients with FLE followed the theoretical suggestion of different frontal subfunctions (38) and met the requirements of the manifold frontal lobe pathology by the use of a broader range of tests. These addressed different aspects of attention, motor coordination, psychomotor speed, fluency, response inhibition, conceptual formation and shift, as well as planning, guessing, and estimating.

Between 1996 and 1999 Upton and co-workers published a series of five articles reporting different findings on neuropsychology in the their sample of 74 subjects with frontal lobe epilepsy (39–43). Using a test battery with different measures of executive functions and motor skills they came to the conclusion patients with frontal lobe epilepsy show a deficit pattern similar to that found in frontal lobe dysfunction in general (39). As compared with patients with temporal lobe epilepsy, frontal patients show poorer motor cordination, guessing, estimation, and response inhibition. Similarly, we found in 23 patients with frontal lobe epilepsy that cognitive problems could be diagnosed with a broad range of 10 "frontal" tasks with about double as many test parameters. The great number of test parameters, however, turned out to be highly redundant and could be statistically reduced to four relatively independent functional areas: "psychomotor speed / attention," "motor coordination," "working memory," and "response inhibition." These four factors explained 70% of the total variance. When compared with patients with temporal lobe epilepsy, those with frontal lobe epilepsy were characterized by prominent impairment in motor skills and response inhibition (44). Problems in speed / attention and working memory were frequent but they appeared rather nonspecific since they were also observed in the temporal lobe group. This, however, does not necessarily contradict the assumption that these are frontal functions. An imaging study by Jokeit *et al.* showed in this respect that in patients with temporal lobe epilepsy, prefrontal metabolic asymmetries are evident that are associated with "frontal lobe measures" and intelligence (45).

In another of our own studies we addressed the cognitive consequences of frontal lobe surgery. We evaluated 33 patients pre- and postoperatively. First, we were able to confirm the impairment pattern of impaired motor skills and response inhibition. Second, we showed that frontal surgery does not cause considerable additional damage as far as eloquent cortex (SMA, motor and language area) is spared. However, when surgery included resection of the SMA the most prominent neuropsychological symptom besides neurological deficits directly after surgery was a SMA deficiency syndrome (impairment of initiation) with aphasia (speech arrest and transcortical aphasia) (46). Additional psychomotor slowing was observed in lobectomies as compared with lesionectomies.

Looking closer at clinical variables that might explain the impairment pattern in nonresected patients, no consistent picture emerges. According to Upton and Thompson seizure frequency and the duration of epilepsy have an effect on performance but this appears to be a nonspecific effect rather than a consistent finding over different tests (41). With the exception of motor skills, which were spared in early right-sided FLE, no systematic effect of the assumed influence of the age at the onset of epilepsy on cognitive development could be concluded from their data (42). The impact of having epileptic seizures on cognition can well be demonstrated by our postoperative findings indicating that in seizure-free patients adjacent functions recovered after surgery. Comparable release effects have been also reported after temporal lobe surgery (47). However, one should not go so far as to conclude that all deficits are due to epileptic dysfunction and thus reversible as has been suggested by Boone *et al.* in a single case report in 1988 (48).

In summary, from the neuropsychological findings in FLE, it appears that indeed different frontal subfunctions can be differentiated. Nevertheless, the measures that characterize FLE have in common the demand of adequate response selection and initiation, and response inhibition. This holds for tests that explicitly assess interferences and response inhibition but also for tests of motor skills or working memory. Ending up again with a unique central executive function, one may hypothesize that the particular problem in FLE is the impairment of response selection / initiation / inhibition with varying emphasis depending on different functional areas. Which area is affected then depends on the type and localization of the underlying lesion, including the possibility that symptoms are overshadowed by spreading epileptic dysfunction.

It is important to mention that the development of appropriate test instruments for the assessment of frontal lobe dysfunction is not yet complete and still represents a challenge for neuropsychologists. Most

psychometric tests that allow quantification of test behavior provide patients with a clear structure for behavior, i.e., with test instructions, rules, time constraints. This enables the patient to behave in an ordered way and real problems with behavior organization arising from frontal pathology are easily overlooked. If provided with more degrees of freedom and demands on spontaneous interactions with complex situations, the same patient would otherwise reveal deficits. A possible solution to this dilemma could be to design tasks that evoke spontaneous behavior and decisions that are up to the subject, as has been done by Bechara *et al.* with the gambling task (49), by Goldberg *et al.* with their cognitive bias task (50), or by Upton and Thompson with their twenty questions task (43).

ICTAL BEHAVIOR IN FRONTAL LOBE SEIZURES: "POSITIVE" AND "NEGATIVE" PHENOMENA

Like others before, we recently analyzed seizure phenomena in patients with frontal lobe epilepsy by video-EEG monitoring. The main purpose was to get hints from seizures for differential diagnosis. On the other hand seizures can be studied in terms of transient dysfunctions, which are more or less circumscribed and point to certain cerebral structures. Seizure semiology, preserved functions, as well as impaired functions can tell us something about the cerebral functional organization of cognition and consciousness. We studied "positive phenomena" in terms of seizure semiology and "negative phenomena" in terms of impairment when patients were neuropsychologically tested during their seizures (51–53).

Ictal phenomena in frontal seizures are mostly positive phenomena (see Table 2). On the one hand, this means a nearly 1:1 relationship between discharges and motor excitation when direct access to motor neurons is possible in primary motor area seizures, for example. On the other hand, this means release and disinhibition of complex behaviors and behavior chains when precentral areas are involved. Examples are posturing and contraversive movements in SMA and premotor seizures, and explosive, bizarre, and emotional unstable behaviors in prefrontal seizures including its mesial parts. Negative phenomena like loss of consciousness are commonly observed in seizures with mesial propagation and secondary generalization. For frontal seizures one can thus conclude that the prominent feature is impairment of executive control in terms of a pathological "hyperexcitation or disinhibition."

TABLE 2
Ictal Frontal Seizure Semiology ($N = 15$)

Localization	Positive symptoms
Motor area	Nearly 1:1 manifestation of seizure activity in myoclonic and tonic or clonic motor activity
SMA	Tonic posturing
Premotor	Contraversive head and eye movements
Prefrontal (including cingulate gyrus)	Explosive and complex motor automatisms (including vocalizations)
	Bizarre and hysterical behaviour
	Mood change
	Negative symptoms
Mesial propagation and secondary generalization	Loss of consciousness
	Impaired executive control: "pathological exitation and disinhibition"

Neuropsychological examination of the cognitive impairment during seizures can provide additional insight into the ictal event. We performed ictal testing in 116 patients, most of whom were candidates for epilepsy surgery. These patients underwent ictal examinations that included examination of orientation reflexes (verbal, nonverbal, tactile), expressive/receptive language (commands, naming repetition), nonverbal reception/expression (commands and imitation), and, finally, awareness and memory (interrogation after the seizure). Testing was performed by the video-EEG monitoring staff and started as soon as possible after seizure onset. Functions were tested hierarchically according to their complexity and testing was continued until the seizure ended. About half of the patients had implanted strip or depth electrodes for invasive EEG recordings. Table 3 shows the impairment pattern that results when distribution of ictal EEG activity at the time of testing is considered. In comparison to lateralized and bilateral temporal lobe seizures, frontal lobe seizures are characterized by prominent impairment of orientation reflexes and expressive speech, which are typical frontal functions. Receptive speech is often preserved. Patients can try, for example, to follow body commands even when they appear involved in excessive motor activity. In contrast to left and bitemporal seizures in particular, consciousness (awareness of any kind) and memory

TABLE 3
Negative Ictal Symptoms in Focal Epilepsy ($N = 116$)

	Location of seizure activity			
	Frontal $N = 29$	Right temporal $n = 21$	Left temporal $n = 38$	Bitemporal $N = 28$
	% Impaired when tested ictally			
Orientation reflex	62	10	18	57
Receptive speech (commands)	48	15	59	93
Expressive speech	77	11	47	76
Memory	31	0	46	100
Consciousness	33	12	39	100

for the test situation during the seizures are mostly preserved.

A very interesting behavior and neuropsychological pattern of impairment can be observed in patients with frontal nonconvulsive status epilepticus. It is important to note that in contrast to grand mal status, which is the repetition of the same seizure, the nature of frontal nonconvulsive status and frontal seizures is completely different. In contrast to frontal lobe seizures, seizure semiology of nonconvulsive status is dominated by negative seizure phenomena. Without EEG recording, the epileptic nature of this state is easily overlooked and patients appear somehow strange since they are slowed, dysphoric, morose, and adverse. When neuropsychologically examined during the seizure we found in five cases consistently generally reduced activity, fluctuating orientation, reflexive and no self-initiated behavior, perseverations, intrusions, apractic signs, problems to shift between tasks, impaired working memory on a higher cognitive level, and emotional instability (see Table 4). In 1997 we already described a single patient with a nonconvulsive status epilepticus who showed a generalized EEG pattern but focal cognitive deficits when neuropsychologically tested during this state. Today, with better diagnostic tools we would probably be able to reinterpret this case also as frontal nonconvulsive status (54).

In contrasting frontal seizures with frontal nonconvulsive status one could interpret the latter rather in terms of an impaired executive control by pathological "hyperinhibition." Impressive recovery to normal behavior can be observed in these patients when the status is successfully ended by injection of diazepam. This is thus one form of state-dependent cognitive impairment. Another form can be seen in postictal impairment.

Because they often do not lose consciousness during frontal lobe seizures, patients are accordingly quickly reoriented postictally. Figure 1 shows the course of verbal memory and decision times in pre- and postictal memory testing after frontal lobe seizures as compared with left/right temporal seizures and repeated testing in healthy controls. After lateralized temporal lobe seizures, material-specific memory impairment can be observed for at least 1 hour after complete reorientation. What is shown for left temporal patients in verbal memory in Fig. 1 has its counterpart for right temporal patients in figural memory. As for frontal lobe seizures it is remarkable that there is no postictal deterioration in memory nor significant slowing of reaction times. However, when seizures secondarily generalize, lasting memory impairment can be observed also following frontal seizures (55).

We can conclude so far that from frontal lobe seizures, a dysexecutive syndrome results with mostly preserved awareness and consciousness, reflexive but not self-initiated behavior, and a seizure semiology dominated by a state of hyperexcitation and disinhibition or hyperinhibition. This would confirm the impression from neuropsychological findings that the major problem in FLE is appropriate response selection/initiation and inhibition of behavior. A further

TABLE 4
Ictal Symptoms in Frontal Nonconvulsive Status Epilepticus ($N = 5$)

Performance	Impairment
Motor functions (including speech)	Generally reduced activity
	Rarely automatisms (fumbling etc. . . .)
Orientation	Fluctuating
Executive functions (including language)	No self-initiated directed actions
	Increased perseverations
	Intrusions
	Apraxia signs in object use and imitation
Reasoning	Problems with concept formation and shift (color/form, etc.)
Working memory	Impaired only when complex mental information processing is required
Emotion	Emotional instability (dysphoric)
Impaired executive control: "pathological inhibition"	

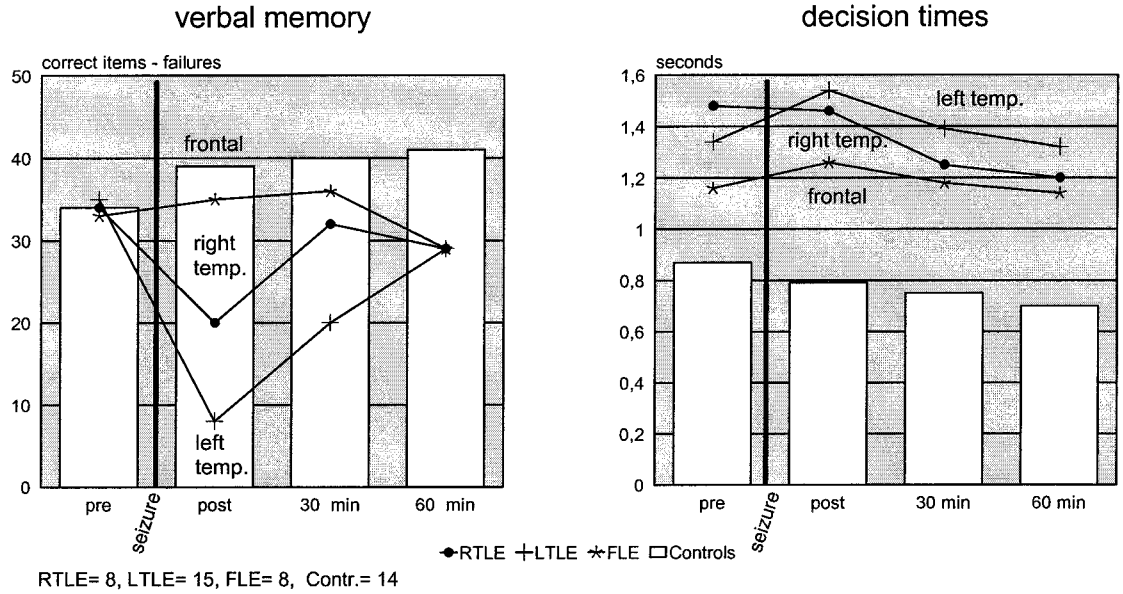

FIG. 1. Preictal baseline measures and postictal course of verbal memory and decision times in patients with frontal and left or right temporal lobe epilepsy. The bars indicate performance of healthy subjects when tested repeatedly in the same intervals.

differentiation according to lesions or foci within particular sites of the frontal lobes can be suggested but has not yet been proven. From a neuropsychological point of view it is still difficult to decide whether one central executive function or different executive functions should be assumed. As already mentioned a compromise is favored at the moment, which suggests that the frontal subfunctions are constituted by similar processes of response selection/initiation and inhibition in different domains and modalities of behavior, respectively.

BEHAVIORAL CORRELATES OF FRONTAL LOBE EPILEPSY

If we propose problems with behavior selection/initiation and inhibition as a functional complex that is affected mainly in frontal lobe epilepsy, the obvious question is whether or not this dysfunction has a correlate in personality and behavior.

With respect to this question we applied several self-rating scales to a group of 95 patients with either frontal (n = 18) or mesial (n = 77) temporal lobe epilepsy. Epilepsy groups were matched regarding sex, age at onset of epilepsy (mean, 11 years), and duration of epilepsy (mean, 24 years). The BPSE "activity subscale" was used to assess frequencies of activities (56), depression and anxiety were assessed by the Beck Depression Inventory (BDI) (57) and the Zung Self Rating Anxiety Scale (SAS) (58), and personality was assessed by the Neo Five Factor Inventory, a German version of the NEO personality inventory (59). Quality of life in epilepsy was assessed by a German modified QOLIE-10 (English version: Cramer *et al.* (60)), and finally we evaluated education and employment to add some objective data.

Group comparisons considering localization and lateralization of epilepsy revealed only slight differences (Table 5). Patients with M-TLE as a trend showed poorer mood and significantly increased anxiety scores; they described themselves more active at home, less active with respect to outdoor cultural activities, and less open for experiences than patients with FLE. It is important to note that, when compared with normative data for healthy control subjects, the result regarding outdoor cultural activities must be interpreted in the context that patients with FLE are more active than the controls and patients with M-TLE. Furthermore, when compared with normal data for a healthy control group, the neuroticism score of patients with M-TLE and the conscientiousness score of patients with FLE appeared elevated.

As regards quality of life, patients were categorized as having poor QOL when they showed scores below the 25% percentile. As shown in Fig. 2 patients with TLE generally tended to report poorer QOL than patients with FLE. Impaired mood, memory problems,

TABLE 5

Scale	Group	Mean	SD	Significance
	Mood (BDI SAS)			
Depression	FLE	7.6	7.3	n.s.
	M-TLE	11.1	9.0	
Anxiety	FLE	29.7	7.9	*
	M-TLE	35.9	7.4	
	Activities (BPSE: activity subscale)			
Home activities	FLE	25.3	4.9	*
	M-TLE	27.8	5.7	*
Social activities	FLE	20.2	5.3	n.s.
	M-TLE	18.7	6.1	
Cultural activities	FLE	16.3	6.5	*
	M-TLE	12.8	5.4	
	Personality (NEO FFI)			
Neuroticism	FLE	21.4	5.4	n.s.
	M-TLE	24.7	7.4	n.s.
Extraversion	FLE	26.2	4.3	n.s.
	M-TLE	26.0	6.2	
Open for experiences	FLE	28.6	6.6	*
	M-TLE	25.1	5.3	
Agreeableness	FLE	31.6	4.6	n.s.
	M-TLE	30.2	4.2	
Conscientiousness	FLE	34.9	5.6	n.s.
	M-TLE	33.2	5.3	

Note. $*P < 0.05$.

and social limitations correspond well to the features of TLE found with the other instruments in this evaluation.

Our current approach to behavioral problems and personality in patients with focal epilepsies is less led by classification systems, which may be useful in idiopathic psychiatric disorders. As already mentioned in the Introduction there is a long history of personality research in epilepsy and up to now no consistent features are discerned. So far this has been explained by the multifactorial determination of psychiatric problems in patients with symptomatic epilepsies. As far as psychometric approaches are concerned, previous studies of temporal lobe epilepsy mostly used the MMPI (61) or, more specifically, the Bear–Fedio Inventory (62, 63). It is our daily experience that commonly used psychiatric scales or psychological personality inventories largely fail to reflect objectively what seems to the examiner clearly to be an epilepsy-related change in personality or a behavior disorder.

At the moment we are evaluating our own clinical personality inventory, which was empirically designed according to a collection of behavioral problems perceived by the clinical psychological staff at the University Hospital of Epileptology, Bonn, Germany (64). For preliminary analysis the questionnaire was consecutively applied to 59 patients with TLE, 17 patients with FLE, 9 patients with parieto-occipital epilepsy, and 44 healthy subjects. It consists of 82 questions concerning 15 different behavioral domains. The answer style is a six-step frequency of occurrence rating with 1 = "occurs not at all" and 6 = "occurs very frequently." Second-order factor analysis resulted in six factors, which were interpreted as follows: (1) "organic personality change" with patients reporting communication problems, emotional lability, being indetermined, and suspectibilty to interference, perseverations, and hypoactivity; (2) "depressed mood," including depressive mood, reduced vitality, anxiety, and insensitivity; (3) "addiction and obsession" including addiction to legal and illegal substances, compulsion, and obsession; (4) "extraversion" comprising sociability, curiosity, and self-determined behavior; (5) "aggression" comprising aggression, sensation seeking, nonadaptive behavior, and violence; (6) "hyperactivity and adaptivity." When clinical data as well as sex are taken as independent variables some interesting results emerge (Fig. 3).

The data indicate that problems in the respective areas are evident in 20 to 30% of the patients. Organic personality changes are preferentially seen in left epilepsies of either origin, as well as in women rather than men. Addiction and obsession are more frequent in right epilepsies and frontal epilepsies in particular. Depressed mood is preferentially seen in patients with hippocampal sclerosis, a finding that is in line with one of our recent publications (65). All patients and patients with parietal epilepsies in particular show

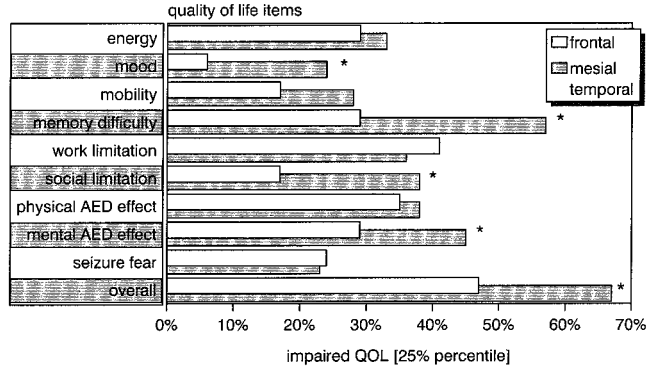

FIG. 2. Quality of life in FLE as compared with M-TLE. Values < 25% percentile were considered as reflecting perception of impaired QOL. Asterisks indicate significant group differences in χ^2 testing.

Scale	impairment < 1 SD	lateralization 37 right / 48 left	localization 59 T/17F/9P	sex 59/70	pathology 28 with AHS
1 organic pers. change	32 %**	LEFT*	TLE/FLE*	W > M	/
2 addiction/obsession	20 %*	RIGHT*	FLE*	/	/
3 depressed mood	28 %	/	/	/	AHS (right)*
4 extraversion	32 %**	LEFT/RIGHT*	PLE*	/	/
5 aggression	31 %**	LEFT*	/	/	/
6 adaptivity/hyperactivity	29 %*	/	FLE*	/	/

*p<0.05,** p<0.01 (significantly different from control subjects)

FIG. 3. Results obtained with the clinical personality inventory. *$P < 0.05$. **$P < 0.01$.

reduced extraversion. Aggressive behavior seems more frequent in left epilepsies, and patients with FLE show increased hyperactivity and adaptivity, which may parallel the finding of increased outdoor / cultural activities and openness for experiences. It is important to note that these results are preliminary and that larger control groups and validation studies are still required. However, the data indicate that the often-cited depressive mood is not the only behavioral problem in patients with focal epilepsy and that apart from this there are specific behavioral aspects that appear related to localized and lateralized lesions / or epileptic dysfunctions.

Although no differences between patients with FLE and TLE could be observed it is worth reporting the results with respect to organic personality change scale in more detail. As shown in Fig. 4 for selected items, about 20% of the patients report that they offend others,

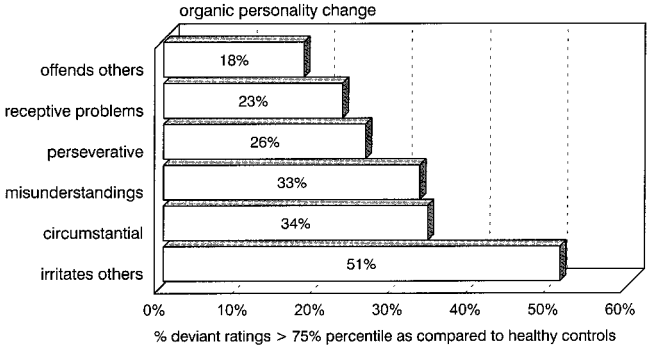

FIG. 4. Items extracted out of the "organic personality change" scale of the clinical personality inventory. Bars represent the percentages of patients with focal epilepsies reporting increased problems in communication and interpersonal contact.

between 20 and 35% of the patients report problems with reception, misunderstandings, or that they were perceived as perseverative or circumstantial, and 50% report that their behavior irritates others. This is similar to the "epileptic personality" and, taken together with the depressed mood, one might as well think of the dysphoric and paroxysmal mood disorder as has been proposed from a more psychiatric point of view (66).

ACADEMIC ACHIEVEMENT AND EMPLOYMENT IN FRONTAL LOBE EPILEPSY

It is well known that patients with frontal lesions may show unimpaired cognitive functions but nevertheless fail on everyday demands of job and career because of behavioral problems, unsteadiness, concentration problems, increased susceptibility to interference, and problems with timing and planning. Subjective data may not reveal behavioral problems because patients with frontal lobe lesions have been reported to underestimate their impairments. With school achievement and employment, however, we have indirect markers, which allow us to infer to what extent patients are adapted to everyday life. As indicated in Table 6 it is not the group with FLE but that with M-TLE that is less educated, and the job situation is comparable in both groups.

TRAIT OR STATE

The above data suggests that patients with frontal lobe epilepsy have behavioral disorders that appear

TABLE 6

	Academic achievement level				Employment
	No regular school	Low[a] (Hauptschule)	Medium (Realschule)	High[a] (Gymnasium)	Employed
FLE (n = 18)	17%	22%	22%	39%	68%
M-TLE (n = 83)	10%	54%	21%	15%	59%

[a] χ^2, significant difference.

very mild as compared with those reported in patients with frontal mass lesions. With respect to mood disorders they appear less affected than patients with temporal lobe epilepsy and they also show better academic achievement. The finding that hyperactivity, addiction, and obsession might be behavioral features of FLE is of great interest and can be discussed as reflecting frontal dysfunction in general and as being in line with the behavior observed in neuropsychological examination and during seizures. The question that remains is how consistent the behavior in focal epilepsies is over time.

We cannot yet conclusively answer this question on the basis of long-term follow-up observations. The impact of epilepsy and seizures on behavior, however, can be estimated by comparisons of patients who after surgery still have seizures and those who are completely seizure free. We therefore analyzed data from operated and nonoperated patients who participated in a long-term follow-up study, which was originally designed to show the cognitive development of these patients over time (67). At the long-term follow-up visit we also assessed depression by use of the Beck Depression Inventory and quality of life by use of a German modified QOLIE-10. For the present purpose we extracted from the total database only the data for patients with temporomesial epilepsy and hippocampal sclerosis as compared with those with frontal lobe epilepsy. Fifty-seven patients had mesial temporal lobe epilepsy with hippocampal sclerosis (27 had surgery, 20 were treated conservatively) and 30 patients had frontal lobe epilepsy (16 had surgery, 14 were treated conservatively). Taking depression and quality-of-life measures as the dependent variables in a multivariate analysis with consideration of surgery, localization, and lateralization of epilepsy as independent variables and age and follow-up interval (mean, 56 months, 2–10 years) as covariates, seizure outcome turned out to be the only significant predictor. Only 14% of the seizure-free patients in contrast to 51% of those who still had seizures showed elevated depression scores greater than the cutoff score of 12 points. It should be noted that 14% is much less than the usually reported 30% of patients with focal epilepsy and depressive mood, and that 51% clearly exceeds this number. Comparably, 45% of the seizure-free patients reported good quality of life with QOLIE, as compared with only 11% of the patients who continued to have seizures. Although these are not follow-up data and depression and quality of life represent only two facets of the whole range of behavior, these data show quite impressively what a difference the presence or absence of seizures can make. The finding parallels recent findings in children who after successful epilepsy surgery showed marked improvement in behavior disorders (68). Long-term follow-up studies on personality and behavior disorders are thus needed to complete our understanding of the interaction between brain damage, epilepsy, and behavior.

CONCLUSION

We can conclude that in frontal lobe epilepsy "frontal dysfunctions" characteristically become evident in cognition, seizures, and behavior. The main common feature of the behavioral problems in FLE is behavior control in terms of response selection / initiation and inhibition. The domains in which these problems become apparent may vary with clinical conditions. Following our own findings hyperactivity, conscientiousness, obsession, and addiction can be seen as behavioral correlates of frontal lobe dysfunction in frontal lobe epilepsy. Depression, anxiety, neuroticism, cognitive (memory) impairment, and social limitations, in contrast, seem to be features of mesial temporal lobe epilepsy. However, methodological difficulties regarding the adequacy of the clinical measures in use as well as confounding effects of lesions, epileptic dysfunction, AED, and psychosocial status do not yet allow further distinctions as they are made for example in neurobiological models about the frontal lobes

and behavior. Full-blown personality disorders are very rare in FLE and symptoms appear rather mild as compared with patients with mass lesions. As regards the state/trait discussion in epilepsy, the effects of seizure control indicate that a major component of the observed behavioral problems is indeed state dependent. However, follow-up evaluations are needed to understand the contribution of lesions and epileptic dysfunctions to behavior disorders and to demonstrate to what extent these are reversible.

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