ORIGINAL ARTICLE

Epileptic EEG discharges and short non-convulsive crisis: Influence on cognitive and psychobehavioural functions in youths

Sergio Domizio¹
Emanuela Conte¹
Cristina Puglielli¹
Roberto Domizio^{1*}
Alberto Verrotti²
Rocco Pollice^{*}
Massimo Casacchia^{*}
Chiara M Conti³
Rita Roncone¹
Giovanni Sabatino¹
Robert Doyle⁴
Renato C. Barbacane⁵
Pio Conti⁵
Giuseppe Sabatino¹

¹Neonatal Intensive Care Unit, University "G. D'Annunzio" - Chieti, Italy

²Department of Pediatrics, University "G. D'Annunzio" - Chieti, Italy

³School of Psychology, Clinical Psychology Div., University "G. D'Annunzio" - Chieti, Italy

⁴Massachusetts General Hospital, Division of Pediatric Psychopharmacology, Boston, MA, USA

⁵Immunology Division, University "G. D'Annunzio" - Chieti, Italy

*Department of Experimental Medicine, University of L'Aquila, Italy

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Abstract

Purpose: Learning and behavioural difficulties often occur in benign childhood epilepsy. In recent years, several electroencephalogram (EEG) characteristics have been related to the occurrence of learning and behavioral problems. We determined if the cognitive characteristics of epileptic children depend exclusively on illness factors, or if epileptic electroencephalogram discharges during the crisis contribute to these changes.

Methods: We studied a randomly selected group of 150 youths with short non-convulsive crises, who completed cognitive testing and electroencephalographic studies. The inclusion criteria were: undefined crisis, variations in cognitive function and/or frequent epileptiform discharges on the electroencephalogram.

Results: Previous research indicates that the type of epilepsy and the patient's educational level can influence cognitive functioning. The electroencephalographic epileptic discharges during the crisis has been found to influence

cognitive transitory functions such as vigilance or swift-ness of mental functions. The type of epilepsy is correlated statistically with impairment of learning ability tests: reading ($F, 5.487, P = 0.005$) and mathematics ($F, 3.007, P \leq 0.05$). In addition, 40% of the epileptic patients had behavioural disordered versus 16% for the control group ($P < 0.02$).

Conclusions: Our results show dissociation between the characteristic directly dependent on epilepsy, particularly the type of epilepsy, on stable cognitive functions, such as the progress in school, and the effect of parosystic anomalies or the immediate effect of crisis and EEG discharges on cognitive processes.

Alteration of cognitive function and psychiatric disorders are common in epileptic children but the underlying causes of these problems remain unknown.¹⁻⁴ One

possibility is that alteration of cognitive function is a consequence of the epileptic syndrome. However, these changes could also depend on the type of epileptic crisis.⁵⁻⁷ In the clinical setting, the choice of therapy, in part, is influenced by the potential for the crisis to cause damage to neural tissue.⁸⁻⁹ The greater the potential for neurological damage the more aggressive is the therapeutic intervention. The short non-convulsive crisis manifests itself as behaviour or attention variation.¹⁰⁻¹² These kinds of crises are difficult to identify in clinical practice.

In children with short non-convulsive crises, the decrease in school performance is often the first indication of a problem. Poor academic performance may be caused by a cumulative effect of undiagnosed crises over time.¹³ The epileptiform intercritical discharges can be a complication in most of these children.¹¹ In 1939, Schwab¹⁴ showed the negative influence of epileptiform intercritical discharges on cognitive function in the absence of clinical manifestations. Patients with epileptic subclinical activities exhibited slowing of their reaction time. One study¹⁵, made with videotelemetry electroencephalogram (EEG), showed that epileptiform EEG discharges have a similar effect on cognitive function as do short crises. Cognitive transitory alteration occurs in cases where epileptiform EEG discharges appear associated with cognitive function changes.

This study seeks to identify the degree to which cognitive function alterations, in epileptic children, depend on illness factors (such as the type of epilepsy or crisis), versus the epileptic EEG discharges during the crisis. We excluded patients with epileptic syndromes that influence cognitive function, such as West's syndrome. In our study, stable characteristics of epilepsy that depend on illness, such as the type of epilepsy or crisis, are considered to be influencing factors on cognitive functions, independent from the epileptic EEG discharge characteristics.¹⁶ In some epileptic patients we can observe cognitive alteration even if crises are pharmacologically controlled or EEG discharges are rare. In our study, we considered

the type of epilepsy and the type of crisis to be closely correlated. Therefore, the EEG discharges may be an epilepsy modulation factor (that can increase or decrease the epilepsy or crisis effect on cognitive function). This was tested with correlation and statistical analysis. Cognitive alteration, for individual cases, also depends on the test used.^{13,15,17} Most of the cognitive theories consider both the stable cognitive aspects (school level and vocabulary) and the mechanistic and transient cognitive ones (attention and vigilance).¹⁸⁻²⁰ Based on the current literature we theorize that the stable cognitive function is changed by illness factors, while mechanistic processes are altered by acute epileptiform discharges.²¹⁻²²

Patients and methods

The study was approved by the Ethical Committee of the University of Chieti, Chieti, Italy and formal consent was given by the parents of the young patients recruited for the study.

We conducted a prospective study of 150 children and adolescents aged 10 to 15 yr who were recruited from our clinic between January 2004 and December 2005. The inclusion criteria were: 1) undefined crisis and variations of cognitive functions and/or 2) frequent epileptic EEG discharges during recording and 3) absence of epileptic syndrome.

All cases received part of their cognitive evaluation while simultaneous EEG recording was performed for two hours. After the EEG recordings we administered an intelligence test to assess the behaviour of the subjects. Their parents completed the child behaviour checklist (CBCL). The same neurologist evaluated all of the EEGs. In 31 subjects, we did not confirm the diagnosis of epilepsy so they were used as the control group. All of these subjects had a normal IQ.

We analysed a final total of 119 patients with short non-convulsive crisis.

We used the following variables:

1. Measurement of epileptic illness effects:
 - a) Epilepsy type
 - b) Crisis type
2. Measurement of acute discharge effects:
 - a) Frequency of EEG anomalies during cognitive tests
 - b) Non-convulsive crisis that was continuous for at least two hours during the recordings. We analysed the number of crises, their duration and location during the cognitive tests.
3. Measurement of stable cognitive functions, based on history:
 - a) Verbal and visual-spatial tests.¹⁸
 - b) School level tests: reading and mathematics.^{19, 23}
4. Measurement of mechanistic and transient cognitive functions:
 - a) Attention tests and tests that estimate speed of information elaboration.
 - b) Tests that estimate late memory.

Cognitive tests

Stable cognitive functions based on acquaintance

We used the Wechsler intelligence test for children (Wisc-RN).²⁰ During the EEG recording we administered two parts of Wisc-RN: a verbal (vocabulary) and a visual-spatial test (drawing). The other parts of the test were administered after the EEG registration. We also administered tests to determine the school level: reading of phrases for a time of 1 minute and simple mathematical calculations for the same amount of time.²⁴⁻²⁵

Attention and information elaboration functions

To analyze activity status, reaction times to a casually presented visual (white square on the screen) or an auditive (800 Hz sound) stimulus were recorded. The time was measured in ms separately for the dominant and the non-dominant hand.²⁶⁻²⁷ The binary reaction test (BCRT) was used to evaluate the aspect of decision-making. The children had to react in different

ways according to the various visual stimuli (red square in the left part of the screen and green squares on the right part of the screen). The reaction time, calculated in milliseconds, reflects the information elaboration efficiency.²⁶⁻²⁷ Reaction times on the Goldstein's visual search test were calculated in seconds.²⁶⁻²⁷

Memory function

To analyze picture and word recognition, we showed 6 words and 4 pictures for 1 second for each article. After two seconds, we showed one of these articles with different pictures. The child had to recognise the article. The final score depended on the number of articles recognized for a total of 24 stimuli showed.²⁶⁻³⁰

We also performed Corsi's test, which calculates continuous attention time. The child had to repeat the same sequence of pictures presented on the screen. The final score is representative of the total recall.³¹

Assessment of behaviour

The patients' parents completed the Child Behaviour Checklist³²⁻³⁴, which is an internationally established instrument for the assessment of behavioural disorders, that has proven validity in children and youths with epilepsy.³⁵ The questionnaire consists of 113 questions on problematic behaviour; it provides a general behavioural problem index (total score) and scores on different subscales. According to previous research on behavioural problems in children with epilepsy³⁶ we used the scales of internalizing behaviour problems, externalizing behavior problems, social problems, thought problems and attention problems. Behavioural problems are measured by *T* scores. Scores equal to or < 59 represent no behavioural problems, scores between 60 and 63 are considered borderline, whereas scores higher than 64 indicate extensive behavioural disorders.

TABLE 1. Demographic and clinical characteristics.

	Control group	Epileptic patients
Number of patients	31	119
Sex (M/F)	1 9/11	1 1/59
Age (yr)	13.8	12.9
QI (Wechsler test)	97.6	82.6
Drugs (median dose, mg/die)		No drugs, n= 47 ^a Valproate, n=41 (747.8) Carbamazepine, n=39 (439.49) Topiramate, n=13 (125) Lamotrigine, n=12 (202)
Epilepsy type		Cryptogenetic focal epilepsy, n=67 Symptomatic focal epilepsy, n=28 Generalized idiopathic epilepsy, n=18 Other epilepsy type, n=6
Crisis type ^b		Partial crisis, n=83 Simple crisis, n=10 Complex crisis, n=70 Partial crisis with generalization, n=3 Generalized crisis, n=36 Atypic absence crisis, n=26 Absences, n=8 Myoclonic crisis, n=2 Tonic-clonic crisis, n=15 unclassified crisis, n=1

^a16 epileptic patients in the absence of therapy during the study. All of these patients had crises early.

^b Crisis classification at the beginning of the study. 13 epileptic patients with number of crises >1.

Statistical analysis

We used the CVST, which is a test that evaluates mechanistic and transient cognitive functions. Many studies have demonstrated the relationship between cognitive alteration, shown by CVST and epilepsy.^{11,13} The effect for CVST was calculated by subtracting the median score of epileptic patients from the median score of non epileptic patients, and then dividing by the SD (standard deviation) of the two samples.

The type 1(α) and 2(β) errors are included as a sample effect index of 0.7 SD in the statistical analysis. The statistical power is 1- β = 80%, and the sensitivity is α =5%. Cohen's statistical power of the patients were >26.37-40 We performed variation analysis (Manova) using the cognitive variables as

dependent factors and a general linear model. The correlational analysis was calculated with Spearman's rho as the parametric correlation coefficient. The significance level was set at 5% for the test. For multiple tests we used Bonferroni's correlation.

Results

Children with short non-convulsive crises demonstrate lower school performance, which is often the first indication of a neuro-psychiatric problem. We divided the 119 adolescence into two groups:

Epileptic-illness characteristics

1. Epilepsy type: a) idiopathic generalized epilepsy, n=18; b) cryptogenetic focal epilepsy, n=67; c) symptomatic focal epilepsy, n=28. Six patients were excluded.

2. Crisis type: a) partial crisis, n=83; b) generalized, n=36. (Table 1)

Epileptiform acute discharges

1 Epileptiform acute EEG discharge frequency during cognitive tests:

- a) sporadic discharges (<1% of total test time), n=63;
- b) frequent discharges (>1% of total test time), n=55.

The group with frequent discharges (>1% of total test time) was subdivided as follows: 1-10%, n=33; 10-50%, n=15; 50-75%, n=4; >75%, n=3 (medium, 1.35%; SD, 1.13%). Three patients were epileptic but they did not have any EEG alterations during cognitive tests. They had focal symptomatic frontal epilepsy.

2. Crises that occurred during cognitive tests:

- a) no crisis during tests, n=87;
- b) presence of crisis during tests, n=34.

The correlation between the four epileptic factors (epilepsy type, crisis type, presence of crisis, or absence of crisis) was statistically significant ($P < 0.001$). This confirms that the four factors are correlated with each other. The highest correlation values noted are between epilepsy type and the type of crisis ($r = 0.88$). The correlation between these factors and the presence of EEG discharges during the cognitive tests was lower, but remained statistically significant (EEG discharges present in relation to the epilepsy type, $r = 0.58$; EEG discharges present in relation to crisis type, $r = 0.68$). The calculation of the Euclid distances confirmed the straight correlation between the epilepsy type and the type of crisis; however, while there are major distances between the EEG discharges and crisis during the tests.

Table 1 shows demographic data and clinical characteristics of the study population. There is no relationship between the control and the epileptic groups. Moreover, there were important differences for age and sex. Total IQ was inferior in the epileptic group (82.6 vs 97.6, $P \leq 0.05$). Pharmacological treatment consisted of traditional anticonvulsive drugs at low dosage. Thirty-four children had epileptic crises during the cognitive tests: 10 absences, 1 myoclonic crisis, 3 simple partial crises and 20 complex partial crises. The median number of crises during the two hours of recordings was 28 (SD 69.5, mean 9.5) with a average duration of 4.2 sec (SD, 3.5 sec). Crises during the test usually had partial onset, had a short duration, and were highly frequent. Comparing the number and duration of the crisis with the type of epilepsy, we found a high frequency and a short duration ($P < 0.05$ in each case) in patients with absences (47.1 crisis of 3.0 sec duration time) as compared to patients with partial crisis (17.8 crisis of 4.9 sec duration time).

Table 2 shows medium values for cognitive variables, calculated independently for each factor depending on epilepsy. MANOVA analysis was performed prior to the tests that represented the stable

aspects of cognitive functions (two Wechsler tests: the vocabulary and drawing test and the reading and mathematics test). The test was significant for epilepsy type ($F, 2.995; P = 0.02$), while there were no effects for type of crisis ($F, 0.366, P = 0.83$), for the relation between type of epilepsy and type of crisis ($F, 0.522, P = 0.84$) or when there were the covariates of presence of crisis ($F, 0.726; P = 0.58$) and EEG discharges during the test ($F, 1.648; P = 0.17$). The “post hoc” analysis for the type of epilepsy shows that this data is significant in the school tests: reading ($F, 5.487, P = 0.005$) and mathematics ($F, 3.007, P \leq 0.05$). Differences between groups were noted with the lowest scores for the groups with partial epilepsy (symptomatic and cryptogenic) compared with the control group and the group with generalized idiopathic epilepsy (t test $P < 0.001$). The analysis of median scores for reading tests (Tab. II) shows no learning delay in the control group (score: -1.5) and that a delay of 2.3 months was seen for patients with idiopathic generalized epilepsy; Substantial delays of 19.2 months for the group with partial cryptogenetic epilepsy and 12.2 months for the group with symptomatic epilepsy were observed. Similarly, mathematics tests showed delays of 2.9 months for the control group to 17 months for the group with partial cryptogenetic epilepsy. Delay in patients with partial epilepsy was equivalent to 2 school years. With Cohen’s analysis²⁹, a difference was evident between small, middle and large cognitive effects (0.20, 0.50 and 0.80 SD). The difference between median scores of the groups was divided by the Standard Deviation (SD) score of all the groups. The final score for the reading test was 1.95 SD and for the mathematics tests was 0.96 SD. We also analyzed transient cognitive function (attention and information elaboration functions) using four tests of reaction (visual and auditive RT, BCRT, CVST) as dependent variances. Tests were not significant for type of epilepsy for the type of crisis nor for the relationship between the two factors.

TABLE 2. Median score of the cognitive functions in relation to factors related to epilepsy.

	Vocabulary tests (SD)	Drawing tests (SD)	Reading tests (retardation, months)	Mathematics tests (retardation, months)
Knowledge stable functions (in relation to cognitive level)				
<i>About the epilepsy type</i>				
Absence of epilepsy	8.9 (3.3)	9.7 (3.5)	-1.5 ^a (5.6)	3.9 (4.5)
Idiopathic generalized epilepsy	7.3 (2.4)	8.6 (3.4)	2.3 (9.1)	4.5 (9.8)
Cryptogenic partial epilepsy	7.5 (3.2)	8.0 (3.5)	19.2 (15.0)	17.0 (15.4)
Symptomatic partial epilepsy	7.4 (3.1)	8.2 (3.6)	12.2 (16.1)	13.2 (14.8)
<i>About the crisis type</i>				
Generalized crisis	6.8 (2.6)	8.2 (3.3)	10.0 (16.7)	11.0 (14.8)
Partial Crisis	7.2 (3.1)	7.5 (3.5)	21.3 (13.8)	19.8 (14.4)
<i>About EEG discharges frequency</i>				
EEG discharges <1%	7.8 (3.1)	8.6 (3.5)	13.6 (14.5)	13.6 (15.0)
EEG discharges >1%	6.3 (2.7)	6.7 (3.0)	18.9 (15.4)	18.0 (14.6)
<i>About the presence of crisis during tests</i>				
Absence of crisis				
Presence of crisis	7.5 (3.0)	8.1 (3.4)	18.4 (17.0)	16.9 (16.7)
	6.3 (2.7)	6.7 (3.1)	15.8 (12.3)	16.4 (11.0)
Reaction time score	Auditory reaction time (MS) ^b	Visual reaction time (MS) ^b	BCRT (MS)	CVST (s)
<i>About the epilepsy type</i>				
Absence of epilepsy	320 (97.8)	435.6 (225.2)	479.6 (181.5)	21.2 (10.1)
Idiopathic generalized epilepsy	370 (174.1)	470.4 (264.3)	537.6 (200.1)	30.8 (14.1)
Cryptogenic partial epilepsy	353.1 (148.4)	441.6 (181.1)	482.6(172.5)	30.8 (15.5)
Symptomatic partial epilepsy	363.3 (112.1)	455.1 (119.1)	559.4(180.4)	34.6 (16.0)
<i>About the crisis type</i>				
Generalized crisis	357.4 (154.5)	465.9 (498.5)	572.9 (235.0)	30.1 (13.5)
Partial Crisis	361.4 (139.6)	442.6(141.3)	492.8(150.3)	32.2 (15.7)
<i>About the presence of crisis during tests</i>				
Absence of crisis	327 (99.7)	431.6 (146.7)	502.2 (185.9)	29.2 (14.1)
Presence of crisis	438 (193.8)	521.3(266.5)	548.5 (179.7)	39.0 (16.3)
Memory	Words recognition	Pictures recognition	Test of Corsi	
<i>About the epilepsy type</i>				
Absence of epilepsy	17.0 (3.9)	12.9 (3.5)	4.6 (1.1)	
Idiopathic generalized epilepsy	10.2 (5.9)	8.6 (3.7)	3.5 (1.1)	
Cryptogenic partial epilepsy	12.5 (6.2)	8.9 (3.8)	3.8 (1.4)	
Symptomatic partial epilepsy	8.7 (5.3)	7.9 (4.1)	3.7 (1.4)	
<i>About the crisis type</i>				
Generalized crisis	11.7 (6.0)	9.2 (4.0)	3.8 (1.4)	
Partial Crisis	11.2 (6.2)	8.6 (3.9)	3.8 (1.4)	
<i>About EEG discharges frequency</i>				
EEG discharges <1%	13.5 (6.0)	9.8 (3.7)	4.2 (1.4)	
EEG discharges >1%	8.9 (5.5)	7.2 (3.8)	3.2 (1.2)	
<i>About the presence of crisis during tests</i>				
Absence of crisis	13. 8(5.1)	10.0 (3.4)	4.4 (1.0)	
Presence of crisis	6.4 (5.0)	6.0 (3.5)	2.5 (1.2)	

^a score > to the media for the age school.^b score for the dominant hand

We found an effect when we considered the EEG discharges as covariates with the type of epilepsy (F , 2.371; $P=0.03$). The “post hoc” analysis showed that we had significant values in the two tests of knowledge elaboration: BCRT (F , 5.383; $P=0.02$) and CVST (F , 7.481; $P=0.007$). The “post hoc” t test showed that, for each cognitive test, results depended on the lower score in the group of patients with EEG discharges (no EEG discharges group versus EEG discharges groups, $P<0.001$; EEG few discharges group versus EEG many discharges; $P<0.05$). With the BCRT we had 0.5 SD, while with CVST we had 1.2 SD. When we factored the presence of crisis with the type of epilepsy as a co-variable we found a significant effect (F , 3108, $P=0.007$). The “post hoc” analysis showed that we had significant values with auditive tests (F , 4.197; $P=0.04$) and with the CVST (F , 6.185; $P=0.01$). The “post hoc” t test showed that, for each cognitive test, results depended on a lower score in the group of patients with crisis during the cognitive tests with regards to the control group (epileptic patients without crisis during tests; $t<0.01$). The effect fluctuated from 0.9 SD (ART) to 1.4 SD (CVST). The memory functions were analysed with the Corsi test, using as dependent variables the number of exact answers in the test of recognition of phrases and pictures. Therefore, we had a correlation with the type of epilepsy (F , 2.512; $P=0.02$) but not with the type of crisis (F , 1.566, $P=0.20$), or with the relationship between these factors (F , 0.686, $P=0.56$). The association of EEG discharge effects, as covariance, to the type of epilepsy did not show statistical significance (F , 2.308, $P=0.08$). On the other hand, the association between the crisis parameter and the type of epilepsy had a significant effect (F , 21.087; $P\leq 0.001$). The unvaried “post hoc” test showed that the consequence related to the kind of epilepsy is caused by only one test, that of recognition of words (F , 4.702; $P=0.01$).

The “post hoc” t test for the differences among groups showed that lower scores in the group affected

by partial symptomatic epilepsy influenced the effect (Table 2: middle score 8.7 versus 17.0 for the control group; effect range: differential score divided by SD calculated on the whole sample = 1.8). The effect of the presence of crisis during tests was statistically significant for all the tests: recognition of words (F , 41.396; $P<0.001$), recognition of figures (F , 20.586; $P<0.001$) and the Corsi test (F , 52.232; $P<0.001$). The “post hoc” t tests showed that meaningfully lower values (values of $P<0.01$) in the group with crisis influenced the effect in all the tests analyzed. The range of this effect is within ± 2.4 SD for the word recognition test, ± 2.0 SD for the figure recognition test and ± 1.8 SD for the Corsi test. A further ANOVA was performed, using the crisis factor during cognitive tests as the single dependent variable, because no such factor had been used in the analysis as a moderate variable (yes or no).

We postulate that the number of crises and their length could influence the effect on cognitive function. Therefore, the number of crises and their duration (median duration expressed in seconds) have been inserted as co-variables. No statistically significant effects have been found for the most permanent of the cognitive functions: vocabulary tests and drawing in Wechsler blocks and reading/arithmetic tests (number of crises: F , 0.799; $P=0.53$; crisis length: F , 0.554; $P=0.70$). However, reaction times showed an effect related to the number of crises (F , 2.539; $P<0.02$). Memory tests also showed effects related to crisis length (F , 5.255; $P<0.002$), but not for their number (F , 2.174; $P=0.10$). Likewise, further ANOVA for EEG episodes, using as covariates the lateralization and the localization of the crises (multifocal, generalized, absence of epileptiform activity, frontotemporal, occipitalparietal) was performed. Neither the lateralization (F , -0.214; $P=0.97$) nor the localization (F , 1.882; $P=0.09$) had effects on cognitive tests (for Wechsler’s test and reading/arithmetic tests, F , 0.447; $P=0.77$; F , 0.513; $P=0.73$; for reaction times, F , 0.113; $P=0.99$; F , 1.126; $P=0.35$; for the tests on

mnesic function, F , 0.736; P = 0.53; F , 0.690; P = 0.56).

Results of the parents' answers to the CBCL are indicated in table 3. Forty percent of the epileptic patients had a T score > 60 (borderline or behaviourally disordered) versus 16% for the control group (P < 0.02). Table 4 shows that behavioural problems are particularly found in the areas of attention and social problems (social isolation, integration difficulties with peers). The highest mean CBCL scores are 63.6 for social problems and 64.6 for attention deficits.

Discussion

The risk of cognitive decline over time seems to be increased for children with epilepsy. Cognitive characteristics of epileptic children depend exclusively on illness factors, or if epileptic electroencephalogram discharges during the crisis contribute to these changes.

The results of our study are important in agreement with the methods used by Cohen (where > 0.80 SD is a very significant effect).³⁷ The effects observed in this study are in most cases > ± 1 SD. The model we used shows that 3 factors have a predominant effect on cognitive function. The type of epilepsy influences the stable effects, which are those functions which require the accumulation of knowledge over long periods of time. Patients affected by focal epilepsies have a low educational level, which cannot be explained easily by other factors. This effect was evident in the 0.9-1.9 SD, where 1.9 SD represented a learning delay of about 2 years in normal school education. According to the effect exerted by a type of epilepsy on stable cognitive function, the IQ was basically lower in the group affected by symptomatic focal epilepsy (76.9). This explains the substantially lower IQ observed in the global group of patients affected by epilepsy than in the control group (82.6 vs 97.6; P > 0.05). The type of epilepsy also influences mechanistic cognitive processes, memory or the effect caused by the alteration of the verbal memory, observed in patients af-

TABLE3. CBCL T percentage > 60

CBCL T > 60	Epileptic patients	Control group	P
Total	40%	16%	2

TABLE 4. Mean CBCL scores

CBCL	Epileptic patients	Control group
Internalising problems	597	568
Externalising problems	548	524
Social problems	636	607
Thought problems	585	550
Attention problems	646	612

ected by symptomatic focal epilepsy. This effect is also significant (1.8 SD). We should emphasize that verbal memory is the one most closely linked to the stable aspects of knowledge, which depend on cognitive function associated with acquired vocabulary. The effect of the paroxysmal epileptiform activity on cognitive functions mainly influences the mechanistic cognitive processes. The short non convulsive crises can alter the alert status and the speed in information elaboration. This effect varies from 0.9 to 1.4 SD and is significant. Other studies have shown the effect on attention and vigilance status during the frequent daily crisis.⁴¹

Short-term memory is the second cognitive process which seems to be altered in the presence of crisis. It has an important effect in terms of differential scores, with values from 1.8 to 2.4 SD. Subsequent analysis showed that the number of crises was associated with an altered alert status and/or to a mental slowing, while the length of the crisis (the critical period) was associated with memory alterations.⁴²⁻⁴⁶ Again, only in the presence of frequent crises is there an alteration of mental alertness, and only during a crisis of threshold duration can we observe a deterioration of memory, as found in other studies.^{11,16} The effect of the crisis is not independent of the type of epilepsy. Consequently, partial epilepsies may be responsible for this effect.

According to Aarts et al.¹⁵, the EEG discharges have a further effect, similar to those provoked by the

crisis (that is on attention processes and on the speed of information elaboration). This effect is limited to those patients with frequent EEG discharges relatively moderate compared with other effects analyzed by the study (0.5-1.2 SD). Consequently, the concept of “subclinical epileptic activities” or EEG discharges could be associated with “transient cognitive alterations”, albeit mainly transient mechanistic ones. This effect occurs only if the discharges are inserted as co-variables therefore it is not independent of the type of epilepsy, because it mainly happens in patients affected by partial epilepsy. Since no certain type of crisis was selected in this study, we expected that the majority of patients with difficult to identify crises would have an absence of crises.⁷ On the contrary, it was observed that crises with short partial onset are associated with a high risk of unsuccessful clinical identification and represent the predominant type of crisis in our study.

Regarding psychobehavioural disturbances, our study confirms the high prevalence of behaviour problems in youths with epilepsy, even when the crises are short and difficult to identify. Hermann³⁵ showed that biological, psychosocial, demographic and medication factors contribute to behaviour disorders. With regard to the epilepsy, a high frequency and an early onset of seizures have been identified as the most important risk factors.⁴⁷ We have noted that after successful antiepileptic treatment the behavior problems diminished. This suggests that the behavioural improvements are due to biological factors rather than to psychosocial effects because we assume that important social factors, such as family status, remained stable within the short interval.

Evidence for this “biological hypothesis” comes from the study by Dunn³⁶ that found a high prevalence of behavioural disorders in children and youths even before their first seizures. Therefore, the epileptic focus may directly influence behavioral problems which can be improved by efficient antiepileptic medication.

Conclusion

All of the cognitive models make a distinction between acquired knowledge, such as vocabulary, on one hand and mechanistic processes independent from knowledge such as attention, on the other hand.¹⁸⁻¹⁹ In our study, this distinction between stable aspects, which mainly depend on acquired knowledge, such as educational level, and the mechanistic cognitive processes as attention or speed of information elaboration. Our results show dissociation between the characteristic directly dependent on epilepsy particularly the type of epilepsy, on stable cognitive functions, such as the progress in school, and the effect of paroxysmic anomalies or the immediate effect of crisis and EEG discharges on the mechanistic cognitive processes. Because the crisis effects and the discharges are not independent on the type of epilepsy, a model is suggested in which the effect of epileptiform paroxysmal activity on transient cognitive mechanisms can be accumulated over time. If the general frequency of crises is high, the crises are protracted or there are frequent EEG discharges. In these cases the acquisition of information can be blocked with progressively devastating effects on the more stable aspects of cognitive function, such as intelligence and school progress. This interpretation agrees with clinical data which show a cumulative effect of the crises on cognitive function.^{7,19, 46-50} This can further explain the cognitive deterioration that sometimes happens during the period preceding the control of the crises.^{13,36,51} Our study shows that the risk associated with extended crises and the subsequent progressive cognitive deterioration, is especially important in some types of epilepsy, which originates from an epileptogenic process localized to a known or unknown aetiology. Adopting this model, which combines EEG recordings and cognitive tests, will provide important information about the point at which critical activity interferes with cognitive function. Cognitive deterioration is not entirely dependent on etiological factors. The clinical importance for this approach is in the recognition of the

cognitive impairment of the critical activity and the consequent treatment which may prevent the devastating effect on the cognitive and learning progress.⁷ Our study shows that the concept is also valid for partial epilepsy, especially the cryptogenic type.

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Correspondence to:

Prof. Giuseppe Sabatino
Neonatal Intensive Care Unit
University "G. D'Annunzio"
Via dei Vestini
Chieti, Italy
e-mail: gsabatino@unich.it