INTELLECTUAL FUNCTIONS OF PATIENTS WITH CHILDHOOD-ONSET EPILEPSY

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There is a considerable amount of literature to indicate that epileptic children have more learning difficulties in school than age-matched controls or those with other chronic disabilities. This has been extensively documented by the senior author (Rodin 1968) and by Rutter *et al.* (1970), Stores (1978, 1981) and Bourgeois *et al.* (1983). Most authorities agree that the IQ of epileptic children who are not overtly brain-damaged lies in the average range, but there are indications that they tend to cluster towards the lower end and that their scores are slightly lower than those of their siblings (Needham *et al.* 1969, Rodin 1978, Bourgeois *et al.* 1983). Although the findings may not reach statistical significance, it has also been observed, especially with the Wechsler Intelligence Scale, that the performance scores tend to be lower than the verbal scores (Angers 1958, de Moura Simoes 1967, Wagner 1969). The most important factors contributing to lowered IQ scores were early age at onset of the illness, longer duration, persistence of seizures in spite of adequate treatment and possible CNS depressant effects from anticonvulsant medication (Freudenberg 1968, Hung 1968, Rodin 1968, Tchicaloff and Gaillard 1970, Vogt and Schlack 1974, Thompson and Trimble 1983).

Most of the studies have a serious drawback, however, in that they present cross-sectional data and in many cases only one evaluation was done. Even when there were two evaluations the time between tests tends to be rather short, and since Patterson and Fonner (1928) and Barnes and Fetterman (1938) have pointed out that the IQ of epileptic children can fluctuate markedly, a single significant gain or loss cannot be taken as an indication of the patient's permanent intellectual functions. Serial studies are quite rare, and a review of the literature has not yielded any data on long-term outcome. The most recent prospective study by Bourgeois *et al.* (1983) involved an average follow-up of four years. Their study also used, in part, two different IQ measures, so possible differences between verbal and performance areas could not be evaluated.

Since we had observed previously that the Wechsler IQ results for young adult epileptic patients had shown a small but statistically significant decline (especially in the performance area) over five years (Rodin 1968), and comparable data were lacking for children, this investigation was undertaken. The goal was to assess whether childhood-onset epilepsy has a deleterious influence on some mental functions, as sampled by the Wechsler test and, if this were the case, to delineate some of the responsible factors.

Material and method

The charts from the Epilepsy Center of

Michigan for children who were seen for multidisciplinary evaluation between the years 1958 and 1978 were reviewed. Patients who fulfilled the following criteria were then selected for the study: (1) definite diagnosis of epilepsy, defined as a minimum of three epileptic seizures which conformed to one of the types in the International Classification; (2) age at initial evaluation between five and 16 years; (3) Wechsler IQ rather than Stanford-Binet or other assessment of intellectual level at the initial evaluation; (4) one or more subsequent Wechsler IQ tests, with the last one performed at least five years after the first. Patients who had suffered only from febrile convulsions or who had an active concomitant CNS disorder were excluded from consideration. This yielded 64 charts. The clinical and laboratory information on the charts was then abstracted on forms for computer processing. Statistical evaluation was carried out through the Clinical Research Center of the University of Michigan, as well as Wayne State University's AMDAHL 470 V6, using the statistical package MIDAS developed by the University of Michigan. Paired t-tests were used to establish the significance of changes between evaluations. Pearson product moment correlations and χ^2 tests with Yates' correction for small numbers were used when indicated to establish significance of relationships. All findings were evaluated at the alpha = 0·05 level.

The descriptive statistics of the patients are presented in Table I. It is noteworthy that the neurological examination was entirely normal for 70 per cent of the children. 25 per cent had 'soft signs', consisting mainly of mild difficulties with co-ordination, inattention or hyperactivity. Two patients had mild hemiparesis: it was right-sided in one and was probably related to prematurity; the other had developed a left-sided paralysis after the first seizure at 10 months of age and still had slight residual signs. One other patient had cerebral anoxia at birth and showed definite features of infantile hemiparesis, with underdevelopment of left upper and lower extremities.

Results

By the time of last follow-up (mean 9·6 years, range five to 33 years) 50 per cent of the patients had been seizure-free for at least two years. The anticonvulsant regimens had become less complex because the mean number of anticonvulsants per patient had decreased from 2·3 to 1·6. Four patients' anticonvulsants had been discontinued for between one and six years. EEG background slowing had improved slightly and the EEG seizure discharges had lessened significantly ($p < 0·005$).

These generally positive developments, however, were not fully reflected in the IQ measurements. When the total group was compared for Verbal, Performance and Full-scale IQ, there was a slight but not statistically significant decrease between the first and last evaluations on all three measures (VIQ 93·2 *vs.* 90·3; PIQ 92·5 *vs.* 91·4; FSIQ 91·6 *vs.* 90·2) (Table II). The sample was split into remitted and unremitted (N = 32 *vs.* N = 31). One patient was eliminated from consideration because the history of current seizure frequency was unreliable. The findings are shown in Tables III and IV. Patients who continued to have seizures had small losses of function in all measured areas. It was statistically significant for the PIQ and vocabulary, as well as picture arrangement subtests. The remitted group had a slight increase in function for PIQ and FSIQ, as well as on five subtests. There was no change in three of the subtests and a slight decrease in another three. The VIQ had also decreased slightly, but not to the same extent as in the unremitted group. None of the changes in the remitted group was sufficiently large or consistent to reach statistical significance.

It seemed possible that the decreased scores of patients with active seizures might be due to etiological factors also responsible for the epilepsy, so an additional analysis was carried out of those for whom there was no evidence of prenatal or perinatal difficulties, head injury, cerebral infection or other potential external cause, and who had entirely normal neurological examinations. There were 33 such patients, 70 per cent of whom were female. There was a family history of epilepsy in 33 per cent. The same trend of decreased scores for the 15 patients who still had seizures within the previous two

TABLE I
Description of sample

	N	%
Sex: Male	33	51·6
Female	31	48·4
Race: White	57	89·1
Black	7	10·9
Seizure type		
Tonic-clonic	43	67·1
Petit mal absence	14	21·8
Partial simple	4	6·2
Partial complex	8	12·5
Myoclonic	4	6·2
Other*	5	7·8
1 type only	50	78·1
2 types	13	20·3
3 types	1	1·5
History of status epilepticus	1	1·5

Etiological	*None*		*Questionable*		*Definite*	
	N	%	N	%	N	%
Pre/perinatal injury	41	64·0	19	29·6	4	12·5
Postnatal head-injury	56	87·5	8	12·5	—	
Cerebral infection	60	93·7	4	6·2	—	
Other external causes	—		1	1·5	—	
Family history of epilepsy or febrile convulsions	20	31·2				
Neurological examination						
Normal	45	70·3				
'Soft signs'	16	25·0				
Abnormal	3	4·6				
EEG background						
Normal	46	71·9				
Slight slowing	11	17·2				
Definite slowing	7	10·9				
EEG seizure pattern						
None	12	18·8				
Questionable	7	10·9				
Definite	45	70·3				
Behavior						
Essentially normal	43	67·1				
Mildly disturbed	16	25·0				
Markedly disturbed	5	7·8				
School performance						
Below average	33	58·9				
Average	22	39·3				
Above average	1	1·8				
Seizures in remission	8	12·5				

	Mean	*SD*	*Range*
Age (yrs)	10·0	3·1	5–16
Duration of epilepsy (yrs)	3·7	3·4	<1–11·7
Frequency of seizures			
Tonic-clonic	4/yr	2–6/yr	<1/yr–>1/wk
All others	1–2/mth	2/yr–1/wk	<1/yr–>1/wk

Anticonvulsant regimens and dosages

	Initial				*Follow-up*			
	N	Mean	SD	Range	N	Mean	SD	Range
Phenytoin	47	205	106·8	32–500	40	266	102·7	60–575
Phenobarbital	41	63	28·3	15–120	25	88	20·8	30–200
Primidone	14	612	325·5	125–1000	8	768	107·4	150–1000
Carbamazepine	5	560	219·0	200–800	3	866	205·8	600–1200
Valproate	10	1050	752·7	500–2500	9	1388	875·0	500–2500
Ethosuximide	17	741	310·8	100–1250	12	841	322·7	100–1500
Others	17				11			
Not on anticonvulsants	8	2·8 yrs (<1–6 yrs)			6	3·1 yrs (<1–7 yrs)		
N anticonvulsants/pt	2·3				1·6			

*Ill-defined or poorly described episodic symptoms in addition to at least one of the above.

TABLE II
Initial vs. final IQ values of total group (N = 64)

	Value	*Difference*	*SD*	*p*
Verbal IQ	92·3/90·3	−2·0	12·6	NS
Performance IQ	92·5/91·4	−1·1	11·0	NS
Full-scale IQ	91·6/90·2	−1·4	10·5	NS
Information	8·2/7·9	−0·3	3·0	NS
Digit span	8·3/8·0	−0·3	2·5	NS
Vocabulary	8·4/7·6	−0·8	3·0	<0·03
Arithmetic	8·8/8·2	−0·6	2·8	<0·05
Comprehension	8·8/8·3	−0·5	3·9	NS
Similarities	8·9/9·0	+0·1	3·6	NS
Picture completion	9·3/9·1	−0·2	2·7	NS
Picture arrangement	9·3/8·8	−0·5	4·4	NS
Block design	8·7/9·5	+0·8	4·8	NS
Object assembly	8·9/9·4	+0·5	4·8	NS
Digit symbol	9·5/8·1	−1·4	7·5	NS

TABLE III
Initial vs. final IQ values of non-remitted patients (N = 31)

	Value	*Difference*	*SD*	*p*
Verbal IQ	92/90	−2·0	12·8	NS
Performance IQ	89/85	−4·0	11·3	<0·05
Full-scale IQ	90/87	−3·0	10·3	NS
Information	8·1/7·3	−0·8	2·9	NS
Digit span	8·5/8·3	−0·2	2·6	NS
Vocabulary	8·6/7·0	−1·6	3·4	<0·01
Arithmetic	8·9/8·5	−0·4	2·8	NS
Comprehension	9·0/8·2	−0·8	2·8	NS
Similarities	8·9/8·2	−0·7	3·8	NS
Picture completion	8·4/8·1	−0·3	2·6	NS
Picture arrangement	9·5/7·7	−1·8	4·2	<0·02
Block design	8·5/8·3	−0·2	2·6	NS
Object assembly	8·9/8·2	−0·6	4·5	NS
Digit symbol	8·9/7·4	−1·5	6·6	NS

TABLE IV
Initial vs. final IQ values of remitted patients (N = 32)

	Value	*Difference*	*SD*	*p*
Verbal IQ	92/91	−1·0	11·6	NS
Performance IQ	95/97	+2·0	10·2	NS
Full-scale IQ	92/93	+1·0	10·2	NS
Information	8·3/8·6	+0·3	2·9	NS
Digit span	8·0/8·0	0	2·5	NS
Vocabulary	8·3/8·3	0	2·4	NS
Arithmetic	8·8/8·0	−0·8	2·8	NS
Comprehension	8·6/8·6	0	4·7	NS
Similarities	8·9/9·8	+0·9	3·4	NS
Picture comprehension	10·2/10·1	−0·1	2·9	NS
Picture arrangement	9·2/9·9	+0·7	4·4	NS
Block design	9·0/10·6	+1·6	6·3	NS
Object assembly	8·9/10·6	+1·7	5·0	NS
Digit symbol	10·3/9·0	−1·3	8·5	NS

years was again observed (VIQ 93·7 *vs.* 91·0; PIQ 94·2 *vs.* 90·9; FSIQ 93·4 *vs.* 90·4). The differences were not statistically significant, again probably due to the small numbers involved. A rise in PIQ was observed for the 18 patients who were in remission (97·8 *vs.* 99·7), but that also was not of statistical significance; VIQ and FSIQ were unchanged.

Since scaled scores carry an age-correction factor, the decrease in IQ points could have come about either through actual loss of previously acquired abilities or slowed mental growth, thus creating a discrepancy between chronological and mental age. A decrease in the raw scores suggests a degenerative process, while an insufficient increase in raw scores argues in favor of slowed development. Inasmuch as raw scores of the WAIS and WISC cannot be directly compared, this aspect of the investigation was limited to the 33 patients who had at least two WISC evaluations. It was apparent that the raw scores had increased, even for the patients with decreased scaled scores, and the reported IQ differences therefore were due to slowed mental growth.

While these findings established direction of change, they masked the magnitude because of fluctuations in test-retest scores. A decrease of 10 or more points in the verbal area had occurred for 19 patients and in the performance area for 13 of the total group. An increase of the same magnitude was observed in the verbal area for nine and the performance area for 10 patients. These fluctuations occurred at any IQ level. The maximum increase in VIQ was 27 points (92 to 119) and for PIQ 26 points (93 to 119). The maximum decreases were 31 points for VIQ (114 to 83) and 29 points for PIQ (114 to 85). Verbal and performance changes did not necessarily occur in the same direction. The patient who had the largest gain in the verbal area had a one-point loss in Performance IQ, while the patient who had gained 26 points on Performance IQ had gained only 11 points on the verbal portion (90 to 101). The patient who had lost 31 points on the verbal scale had lost 21 on performance (100 to 79), and the patient who had a performance loss of 29 points had a verbal drop of 18 points (105 to 87).

The relationship between significant loss or gain of IQ points (defined as a difference of at least 10 points between first and last evaluations) and clinical state is shown in Tables V and VI. It can be seen that, in general, a minimum gain of 10 points occurred in the performance area only when the patient was in remission. However, clinical remission was not necessarily associated with PIQ gain, and occasionally there were even considerable losses, as will be discussed later. VIQ changes of the magnitude under discussion occurred regardless of clinical state.

Test-retest findings showed considerable fluctuations for the 28 individuals who had at least three follow-up evaluations. A consistent downhill course leading to a minimum 10-point loss was observed for five (17·8 per cent) in VIQ, for seven in PIQ (25 per cent) and for eight in FSIQ (28·5 per cent). A consistent rise of the same magnitude occurred for three in VIQ (10·7 per cent), one in PIQ (3·5 per cent) and five in FSIQ (17·8 per cent).

An attempt to define factors from the first evaluation that would predict IQ gain or loss was largely unsuccessful. There were no signficant relationships between age, sex, presence or absence of etiological factors by history, neurological examination, EEG observations, seizure type, seizure frequency or duration of seizure disorder and VIQ, PIQ or FSIQ. The only significant relationship was with higher initial Verbal IQ (VIQ 98 *vs.* 88, $p<0·01$), and the seizure disorder had started at an earlier age in the patients who had lost 10 or more PIQ points (51 months *vs.* 100 months, $p<0·02$). Since there were no other significant relationships, regression or discriminant function analysis could not be carried out.

The data on anticonvulsant levels are incomplete because determinations had been performed only during the past 10 years; also, since the study deals with retrospective information, blood samples had been obtained only infrequently on the day of psychological evaluation. This was the case in 31 instances for phenytoin, 29 for phenobarbital, eight for valproate, seven for ethosuximide, six for primidone and four for carbamazepine. The mean levels and ranges are shown in Table VII. Because of small sample size the IQ relationships were investigated only for

TABLE V

Relationships of minimum 10-point gain or loss for VIQ

	Gain	*Loss*	*Total*
Non-remitted	3	9	12
Remitted	6	10	16
Total	9	19	28
	$\chi^2=0\cdot08$, NS		

TABLE VI

Relationships of minimum 10-point gain or loss for PIQ

	Gain	*Loss*	*Total*
Non-remitted	1	9	10
Remitted	9	4	13
Total	10	13	23
	$\chi^2=6\cdot0$, $p<0\cdot02$		

TABLE VII

Anticonvulsant levels at time of psychological testing (μg/ml)

	N	*Mean*	*SD*	*Range*
Phenytoin	31	12·3	7·2	5·2– 29·8
Phenobarbital	29	20·5	11·2	5·6– 49·3
Primidone	6	10·4	4·5	2·8– 14·7
Carbamazepine	4	4·6	1·5	3·3– 6·8
Valproate	8	43·9	2·1	16·2– 78·9
Ethosuximide	7	47·8	3·8	56·0–105·3

TABLE VIII

Significant correlations of phenobarbital level with IQ measures (N=29)

	r	*p*
Performance IQ	–0·46	<0·01
Full-scale IQ	–0·38	<0·03
Similarities	–0·39	<0·03
Block design	–0·37	<0·04
Digit symbol	–0·45	<0·01

phenytoin and phenobarbital. No significant correlations were observed between phenytoin levels and IQ measures (VIQ r –0·07, PIQ r –0·19, FSIQ r –0·01). Phenytoin levels which would be considered in the 'toxic' range had occurred only twice (26·8 and 29·8μg/ml). The statistically significant relationship between phenobarbital levels and IQ measures are shown in Table VIII. The correlation was not significant for the VIQ (r –0·16). When the sample was split into groups of 14 patients whose phenobarbital levels had been less than 20μg/ml versus those whose levels had been greater, the mean PIQs were 90·7 *vs.* 78·7 ($p<0\cdot02$). 10 patients had had more than one determination of phenobarbital level in conjunction with IQ testing, but a marked fluctuation in levels had occurred only once, and phenobarbital levels above 40μg/ml had been encountered in only two instances (41·1 and 49·3μg/ml).

Discussion

The results of this investigation confirm that IQ levels of epileptic children tend to be slightly below the mean, and that they can fluctuate markedly on retests in a positive as well as negative direction. Therefore a single evaluation can not be used to establish a long-term prognosis and it can be misleading, especially if anticonvulsant levels are not measured close to the time of testing. Elevated levels, especially of phenobarbital and occasionally of phenytoin, may not result in obvious clinical toxicity and therefore can be missed unless specifically tested for. Yet they can interfere significantly with cognitive abilities, especially in the areas of new learning.

Phenobarbital but not phenytoin levels were significantly inversely correlated with IQ, probably because phenobarbital levels had been significantly higher ($p<0\cdot005$), which resulted from the assumption that levels up to 40μg/ml are within the 'therapeutic' range (Pippenger *et al.* 1978, Kutt and Paris-Kutt 1982). The data presented here, as well as those of Reynolds and Travers (1974), Bourgeois *et al.* (1983), and Reynolds (1983) suggest that this is not necessarily justified and that revision of the upper limit to between 20 and 25μg/ml may be advisable.

A steady rise in IQ was observed mainly for patients whose seizures were controlled, while a drop in IQ occurred mainly among those whose seizures were not controlled. This agrees with previous findings for adults (Rodin 1968, Seidenberg *et al.* 1981) and for children (Bourgeois *et al.* 1983). The precise percentage of patients with major IQ loss (10 points or more) depends not only on the composition of the sample, *i.e.* how many patients still have seizures or are in remission, but also on length of follow-up. 11·7 per cent of the patients in Bourgeois and colleagues' study had deteriorated intellectually, compared to 28 per cent in our sample. This discrepancy may well be due to longer follow-up of our cases, and the fact that 25 of 63 (39·6 per cent) still had seizures within the last year of follow-up. In the Bourgeois *et al.* study only 13 of 72 (18 per cent) were regarded as 'difficult to control', but the term was not defined further. They also gave the impression that a discriminant function formula based on age at onset of seizures and frequency of toxic levels of anticonvulsants allowed accurate classification of 71 per cent of their patients with regard to prognosis for intellect. However, careful reading of the data showed not only that the 'difficult to control' group had a similar degree of IQ decline to the 'toxic' group, as was pointed out by Shafer (1984), but also that the 71 per cent figure is clinically not very meaningful. When one calculates the percentages presented in the paper, the formula classified correctly 93·6 per cent of the patients whose IQ remained within 10 points, but only 42·8 per cent whose IQ had decreased and none whose IQ had increased.

Neither Bourgeois and colleagues' nor our study yielded a reliable set of criteria which would allow an accurate prognosis of patients' future intellectual functions at the time of first evaluation. Prognostication improves when one follows the course of the illness. Early onset of seizures tends to be associated with some intellectual decline, as can persistently high levels of sedative anticonvulsant drugs, but these factors do not fully explain the clinical picture in all instances. We had patients in our series whose seizures were in remission, who were either off anticonvulsants or carried low levels, yet had decreased intellectual abilities.

We found no factors which could be regarded as etiological for the patient's epilepsy and which might have caused a cerebral degenerative process. This raises the question of whether there is a subgroup of epileptic children whose seizures and EEG manifestations improve, but the as yet unknown cerebral metabolic disturbance which predisposes to epileptic seizures persists and reasserts itself later in life. Neurologists who see mainly adults are familiar with patients who had 'outgrown' their seizures in childhood and are seen because of recurrence decades later. One wonders, therefore, whether IQ measurements could have predictive value, in that a failure for IQ to rise when seizures cease and anticonvulsants are withdrawn may have negative prognostic implications. This will require further long-term studies, as will the question of the relationship of anticonvulsants to specific aspects of mental function. Since patients with intractable seizures tend to receive higher doses of anticonvulsant medications, cumulative effects could be expected. Nevertheless, there is a suggestion in our results that the Wechsler subtests may make it possible to distinguish between changes resulting from epilepsy and those due to phenobarbital. Digit symbol, block designs and similarities were significantly related to phenobarbital levels, while vocabulary and picture arrangement appear to have been influenced by continued seizures. These findings, however, should be regarded as tentative.

Studies of this type require a long-term commitment but they can be facilitated if physicians recognize the problem, and if IQ testing of the patient who has just experienced an epileptic seizure were to be carried out as routinely as the EEG is. This would provide a baseline against which future progress or lack of it can be measured. A test which allows separate assessment of verbal and performance functions is preferable to one which gives only global data. The verbal and performance areas of IQ are only moderately correlated for children with epilepsy (r 0·44 for first and 0·61 at last evaluations) and they need not necessarily change in the same direction to the same

extent during the course of the illness. Periodic IQ re-evaluation, combined with determinations of anticonvulsant levels, would then establish the direction of a child's cognitive development. Slowed mental growth then could be detected early and remedial action taken whenever possible.

The long-term social prognosis, in contrast to the medical prognosis, is not good for the majority of patients with childhood epilepsy, as the studies by Sillanpää (1973) and Harrison and Taylor (1976) have shown. They tend to end up in the lower socio-economic brackets of society, which in all probability is due to decreased scholastic achievements which did not prepare them adequately for competitive employment. It needs to be recognized, therefore, that treatment of childhood epilepsy involves more than the seizures themselves. The physician has to be aware of the potentially fluctuating nature of the patient's cognitive abilities and this should be integrated in the total treatment approach.

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SUMMARY
The intellectual functions of 64 epileptic patients who had had an initial evaluation between five and 16 years of age, including the WISC, were re-evaluated after a period of at least five years. In general the seizure states had improved, and 50 per cent were in remission for between two and eight years. All but four were still taking at least one anticonvulsant drug. WISC IQ estimates showed a slight decrease. Verbal and performance areas could be differentially affected, and a gain in one could be offset by a loss in the other, so the Full-scale IQ might not be a reliable measure of day-to-day performance. Those whose seizures remained uncontrolled had a statistically significant decrease in performance IQ, whereas in general it was stable or increased for patients in remission. There was evidence that decreased IQ indicated slower mental growth rather than loss of previously acquired function. Phenobarbital but not phenytoin levels were inversely correlated with IQ, suggesting that the upper limit of the 'therapeutic range' of phenobarbital may already be toxic with regard to learning abilities. To optimize an epileptic child's functioning in school and to prevent long-term intellectual problems, it is advisable that IQ testing should be part of the routine initial evaluation, and that drug levels should be checked at regular intervals.

RÉSUMÉ
Les fonctions intellectuelles de 64 épileptiques dont l'intelligence avait été évaluée entre cinq et 16 ans, notamment par le WISC, ont été remesurées après une période d'au moins cinq ans. En général l'état des crises s'était amélioré et 50 pour cent des sujets étaient en rémission pour des périodes allant de deux à huit ans. Tous les sujets sauf quatre prenaient encore une médication anticomitiale. Les QI au WISC montraient une légère perte. Les échelles verbale et performance pouvaient être modifiées de façon différente et un gain dans l'une des échelles pouvait être compensé par une perte dans l'autre, si bien que la pleine échelle de QI pourrait ne pas être une mesure fiable des performances journalières. Les sujets dont les crises demeuraient non contrôlées présentaient une baisse statistiquement significative à l'échelle de performance, alors que cette échelle était en général stable ou en gain pour les sujets en rémission. Des signes indiquaient que la baisse de QI était davantage liée à un ralentissement de la croissance mentale qu'à une perte des fonctions antérieurement acquises. Les taux de phénobarbital mais non de phénytoine étaient inversement corrélés au QI, suggérant que la limite supérieure de "l'étendue thérapeutique" du phénobarbital peut déjà être toxique en ce qui concerne les aptitudes d'apprentissage. Pour optimiser l'activité scolaire d'un enfant épileptique et pour prévenir des problèmes intellectuels à long terme, il est souhaitable que des mesures de QI fassent partie des évaluations initiales de routine et que les taux de médication soient contrôlés à intervalles réguliers.

ZUSAMMENFASSUNG
64 Patienten mit Epilepsie, die zwischen fünf und 16 Jahren erstmals untersucht und beurteilt—einschließlich WISC—worden waren, wurden nach mindestens fünf Jahren nachuntersucht. Im allgemeinen hatte sich das Anfallsleiden gebessert und 50 Prozent waren seit zwei bis acht Jahren in der Remission. Alle, außer vier Patienten, nahmen noch mindestens ein Antikonvulsivum. WISC IQ-Schätzungen zeigten eine leichte Verschlechterung. Verbale-und Leistungs- Bereiche konnten unterschiedlich betroffen sein und eine Verbesserung in einem Bereich konnte durch eine Verschlechterung in dem anderen relativiert werden, so ist der Gesamt-IQ nicht unbedingt ein verläßliches Maß für die tägliche Leistung. Die Patienten mit unkontrolliertem Anfallsleiden zeigten eine statistisch signifikante Verschlechterung des Leistungs-IQ, während er bei Patienten in der Remission im allgemeinen stabil war oder besser wurde. Es fanden sich Hinweise dafür, daß ein abfallender IQ eine verlangsamte geistige Entwicklung anzeigte und nicht einen

Verlust von zuvor erworbenen Funktionen. Phenobarbital, jedoch nicht Phenytoinspiegel korrelierten umgekehrt mit dem IQ, was vermuten läßt, daß der obere therapeutische Bereich von Phenobarbital schon im Hinblick auf die Lernfähigkeit toxisch sein kann. Um die Schulleistungen eines Kindes mit Epilepsie zu verbessern und um spätere intellektuelle Probleme zu vermeiden, ist es ratsam, IQ-Testungen als Teil der routinemäßigen Erstbeurteilung durchzuführen und in regelmäßigen Abständen Medikamentenspiegel zu kontrollieren.

RESUMEN

Las funciones intelectuales de 64 pacientes epilépticos que habian pasado una primera evaluación entre los cinco y los 16 años de edad, incluyendo el WISC, fueron reevaluados después de un periodo de al menos cinco años. En general la incidencia de ataques había mejorado y el 50 por ciento se hallaban en remisión de dos a ocho años. Todos excepto cuatro tomaban todavía un fármaco anticomicial El CI en el WISC mostraba un ligero descenso. Las áreas verbales y de manipulación podían afectase diferentemente, de forma que la mejoría en un campo podía ser desvirtuada por un descenso en el otro, por lo que la escala global de CI podía no ser una medición fidedigna de la realización dia a dia. Aquellos cuyas convulsiones permanecían sin control tenían una disminución estadísticamente significativa en el CI manipulativo, mientras que en general permanecía estable o aumentaba en los pacientes en remisión. Esto evidencia que el descenso en el CI indicaba un crecimiento mental más lento, más bien que una pérdida de una función previamente conseguida. Los niveles de fenobarbital, pero no los de fenitoina, estaban en correlación inversa con el CI, lo que sugiere que el límite superior del margen terapéutico del fenobarbital puede ya ser tóxico con respecto a las capacidades de aprendizaje. Para optimizar el que un niño epiléptico vaya bien en la escuela y para prevenir problemas intelectuales a largo plazo, se aconseja que la medición del CI forme parte de la rutina de exploración inicial y que la determinación de los niveles en sangre de los fármacos se haga a intervalos regulares.

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