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MENTAL DETERIORATION IN EPILEPTIC CHILDREN*

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Epileptic attacks of all sorts are common in children but probably less than a third of them are associated with serious psychological difficulties (Pond and Bidwell, 1960). Of this latter group a much smaller proportion actually deteriorate by the loss of already acquired intellectual and personality functions as the condition progresses. The concept of deterioration in epileptic adults has often been elaborated and the clinical picture is well known (Pond, 1957). Much less is known about children, and the present paper attempts to define the clinical problem as it presents and to investigate the factors apparently related to deterioration. The paper is not concerned with epileptic children who have always been of idiot or imbecile level but with those who have achieved a certain amount of intellectual progress that later is lost. Also excluded are the rare but well recognized neurological degenerations such as the lipidoses.

Material and Methods

The patients for this study were obtained from the Children's Department of the Maudsley Hospital and from the Fountain Hospital for Mental Defectives, Table I shows the basic data in the two groups of cases. Mental deterioration was assessed in the first place by intellectual deterioration as shown by drops in intelligence test scores during the follow-up studies. In addition, there was a social deterioration in almost all cases as shown by a change in the social responsiveness of the children to their surroundings that was noticed by parents, teachers, and employers. A preliminary study clearly showed that all these patients had evidence in one way or another of brain damage. For purposes of a control group, therefore, children from the two hospitals were taken who also had evidence of brain damage, and were matched for age and sex with the deteriorated group. The following two case histories from the Maudsley and Fountain Hospitals respectively illustrate the clinical histories of cases with deterioration.

TABLE I

BASIC DATA OF MAUDSLEY AND FOUNTAIN GROUPS

		Maudsley		Fountain	
		Cases	Controls	Cases	Controls
Total		20	20	8	8
Males		13	10	5	3
Females		7	10	3	5
Age when first seen	M	9 yr. 9 mth.	9 yr. 6 mth.	4 yr. 6 mth.	6 yr.
	r	5-16 yr.	5-18 yr.	2-8 yr.	2-13 yr.
Initial I.Q.	M	85	90	50	45
	r	50-120	62-115	35-75	20-61
Final I.Q.	M	55	85	25	50
	r	40-80	60-112	10-60	22-68
Number of years of follow-up	M	5 yr. 3 mth.	6 yr. 6 mth.	8 yr.	6 yr.
	r	1-12 yr.	1½-10½ yr.	5-12 yr.	2½-8½ yr.

M = Mean; r = range.

Case 1.—P.V., born on June 27, 1943, has been seen in the clinic since 1952. His parents are highly intelligent but very schizoid. There is one younger normal sibling. Paul's birth and early development were normal. Epileptic attacks, at first only major seizures, occurred twice at the age of 2 years, then again twice at 7 years, and then with increasing frequency from the age of 10 years. As well as major seizures and occasionally status, he now suffers from frequent minor attacks, usually brief automatisms or long periods of confusion and inattention. He had the usual childhood diseases without sequelae but no other illnesses.

From 1948 to 1953 he attended ordinary schools but became increasingly disturbed and backward, especially in the last three years. His intelligence test scores were as follows: in 1951, 103 (Binet); in 1953, 90 (Binet); in 1954, 75 (Binet); and in 1957, 67 (Wechsler). Since the last test he has been in a mental deficiency colony and his behaviour precludes adequate intelligence testing. He has become withdrawn and bizarre, with long periods of apathetic staring alternately with aggressive outbursts.

There have never been any abnormal physical signs and the A.E.G. is normal, but E.E.G.s have always been abnormal, showing diffuse, irregular slow spikes and waves and various cortical foci of spikes as illustrated in Fig. 1.

Case 2.—A.P. was born on May 26, 1942, and has been in the Fountain Hospital since 1947. He was a breech delivery after several attempts at version. Although his

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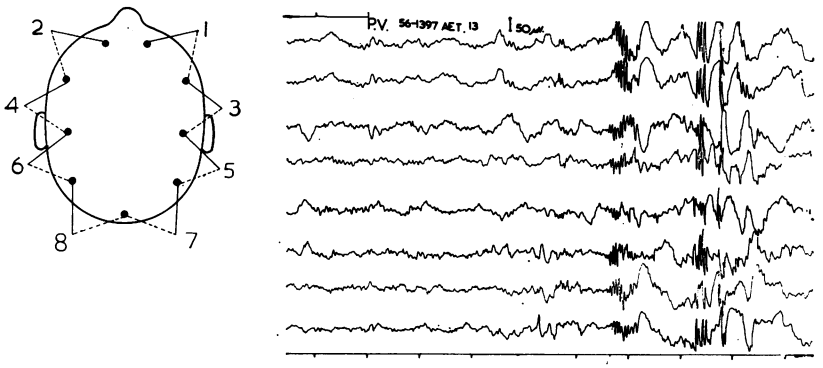

FIG. 1a.—Sleeping record showing normal background activity of sleep interrupted by a symmetrical burst of spikes, fast waves and slow waves.

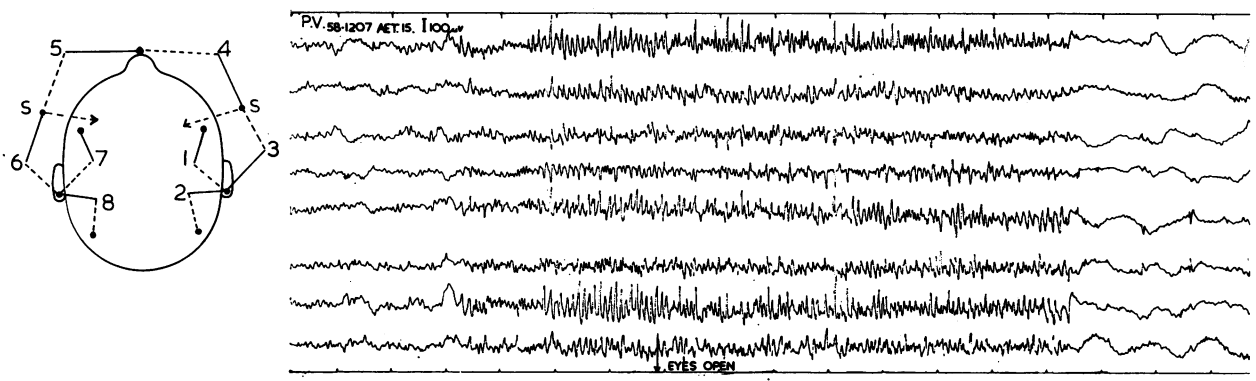

FIG. 1b.—A sphenoidal recording under pentothal showing an unusual generalized brief epileptic attack accompanied by only a minimal change in level of consciousness. Following the seizure spikes are seen at the left base (between channels 5 and 6).

early milestones were said to be normal he was found to be clearly very backward by the age of 4 years when the Binet I.Q. was 45. Up to 1951 he showed slow progress remaining quick, cheerful, but occasionally violent, though able to feed and clean himself. However, he then began to deteriorate, becoming ataxic, perseverative, and incontinent so that he has not been able to attend the hospital school since 1956.

Major epileptic attacks began at 3 months and have continued at about the same frequency of three to four a month in spite of increasing anticonvulsant medication. The abnormal physical signs were doubtful bilateral extensor plantars. An A.E.G. was normal but the E.E.G.s showed at first spikes mixed with generalized irregular slow activity and then later only slow activity.

Results

A variety of psychological tests (Wechsler-Bellevue intelligence scale, Cattell infant intelligence scale, Merrill-Palmer pre-school scale, matrices, and U.S.E.S.) were administered by the psychologists of the two hospitals to these children but the commonest were the revised Stanford-Binet scale and the Wechsler intelligence scale for children. The basic test data are set out in Tables II to V.

Tables VI to IX set out the results of various factors that were investigated and found different or not different between the cases and the controls in the Maudsley and Fountain materials.

The site and extent of brain damage are clearly very difficult to estimate while the patient is still alive. Electroencephalographic (E.E.G.), pneumoencephalographic (A.E.G.), and clinical neurological findings all contribute. In general the E.E.G. shows up only the site of an epileptogenic focus without much being known of its extent. Even pneumoencephalography may show no more than a slight asymmetry of ventricles in cases where subsequent neurosurgery has clearly shown gross pathological changes in the brain. An A.E.G. was done in 11 of the deterioration group and eight of the control group. Diffuse or focal atrophy was found in seven of the deteriorated cases and four of the control

TABLE II

MAUDSLEY CASES

Series No.	Name	Initial Social Status and I.Q.	Later Social Status and I.Q.
1	M.W.	Normal till 6 years 1947 I.Q. = 63	The global intellectual falling off was accompanied by social deterioration (I.C.)
2	P.A.	1954 I.Q. = 100-101	Deteriorated since 1956 (I.C.)
3	R.L.	Normal	1956-57 I.Q. = 60-70 (I.C.) 1957—Temporal lobectomy, no more drop, (still I.C.)
4	O.M.	Normal till 11 years	1956 I.Q. = 50-60 (I.C.) 1958 still needs institutional care
5	E.F.	Normal till 5 years (E.S.N.)	1957 I.Q. = 40-60 (I.C.)
6	M.G.	1955 I.Q. = 105-110	1956 I.Q. = 70-80. Intellectual deterioration between May, 1955, and January, 1956, was noticeable
7	H.J.	Special schooling till 9 years	Ineducable (I.C.) 1958—Temporal lobectomy more S.D.
8	W.M.	1951 I.Q. = 55-70 (E.S.N.)	1952 I.Q. = 45-65. S.D. (I.C.)
9	A.M.	1953 I.Q. = 107	1956 I.Q. = 40-60. Defective (I.C.)
10	M.M.	1946 I.Q. = 79	1952 I.Q. = 46 1954 (I.C.) 1958 further deterioration, (still I.C.)
11	P.B.	1952 I.Q. = 112	1955 I.Q. = 99 1956 (I.C.)
12	G.C.	Normal till 6 years	1950-52 I.Q. = 40-50, Ineducable and psychotic
13	C.R.	1947-49 I.Q. = 105-110	1952 I.Q. = 82, Hopeless work record 1958 still unemployable
14	E.M.	Normal, rather an intelligent girl	1954 I.Q. = 65 1958 I.Q. = 40, Ineducable
15	P.V.	1951 I.Q. = 103	1953 I.Q. = 81, 1954 I.Q. = 75, 1957 I.Q. = 67
16	S.W.	1950 I.Q. = 90	1951 I.Q. = 71, 1953 I.Q. = 52, 1955 (I.C.)
17	L.W.	1947 I.Q. = 82	1949 I.Q. = 67, 1950 I.Q. = 62
18	M.W.	1949 I.Q. = 80-100	1950 I.Q. = 65-70, 1958 Unemployable
19	G.W.	1955 I.Q. = 120	1958 Ineducable, problem for special school
20	L.W.	1948 I.Q. = 105	1952-53, S.D.—considerable decline in social interest

E.S.N. = educationally subnormal.
S.D. = social deterioration
I.D. = intellectual deterioration
I.C. = institutional care (epileptic colony, mental defective colony and special schools)

TABLE III

MAUDSLEY CONTROLS

Series No.	Name	Initial Social Status and I.Q.	Later Social Status and I.Q.
1	B.B.	Normal I.Q. = 75-95	Same range of I.Q. 75-95
2	D.B.	I.Q. = 65-75	I.Q. = 60
3	L.C.	1950 Social Q. = 92	1952 I.Q. = 76, 1953 I.Q. = 73, 1958 Good school report
4	C.C.	I.Q. = 80-90	I.Q. = 80
5	J.D.	1951 I.Q. = 98	No change
6	N.G.	1952 I.Q. = 100 (dull for her family)	No change
7	H.A.	Intelligent girl	Remains intelligent
8	S.M.	I.Q. = 126	No deterioration
9	D.H.	1950 I.Q. = 95	1953 I.Q. = 85
10	D.H.	1952 I.Q. = 62	1958 I.Q. = 64, better at school
11	D.K.	I.Q. = 100-110	Remains normal
12	J.A.	I.Q. = 104	No deterioration
13	S.P.	I.Q. = 77	No change
14	B.P.	1948 I.Q. = 99	1949 I.Q. = 105, 1951 I.Q. = 95. No further change
15	D.R.	1951 I.Q. = 94	Normal
16	A.B.	1955 I.Q. = 70-80	Normal
17	E.S.	1947 I.Q. = 103	1952 I.Q. = 90-110
18	R.S.	1948 I.Q. = 107	1957 I.Q. = 100-110
19	K.O.	1950 I.Q. = 115	1955 I.Q. = 105-110
20	C.W.	Bright child	Remains bright

group. The differences are slight and not significant. In the E.E.G. bilateral foci were seen in half the cases of both groups. Almost all the unilateral changes were in the temporal areas and these again were distributed equally in the two groups and equally between right and left sides in the two groups. Neurological signs were only slightly more common in the deteriorated group than in the controls. In one-third of the patients the histories did not give sufficient evidence to be sure when the brain was damaged. In the other cases the presumed time of damage was at or after birth or later in the same proportions in both cases and controls.

The age of onset of seizures was likewise not different in the two groups which is at first sight surprising since several authors, *e.g.*, Collins and Lennox (1947), have found that the earlier the age of onset of fits the greater the chance of mental backwardness. However, these series include a large number who were defective from the very beginning of their illness as well as those who deteriorated.

TABLE IV

FOUNTAIN CASES

Series No.	Name	Initial I.Q. Level	Later I.Q. Level
1	D.C.	1947-50 I.Q. = 65-75 ESN	1953-54 I.Q. = 50-60 High-grade imbecile
2	A.P.	1947-50 I.Q. = 40-50 High-grade imbecile	1951-54 I.Q. = 20-25 Low-grade imbecile
3	P.B.	1949-51 I.Q. = 35 High-grade imbecile	1956 I.Q. = 20 Low-grade imbecile
4	M.G.	1953 I.Q. = 40 High-grade imbecile	1956 I.Q. = 30 Idiot level
5	E.B.	1953 I.Q. = 40 High-grade imbecile	1956 I.Q. = 14 Idiot level
6	L.K.	1951 I.Q. = 52 High-grade imbecile	1952 I.Q. = 9 Idiot level
7	R.S.	1951-52 I.Q. = 65-75 ESN	1955 I.Q. = 49 1957 I.Q. = 34 1958 Low-grade imbecile
8	M.E.	1951 Normal	1957 Institutional care

TABLE V

FOUNTAIN CONTROLS

Series No.	Name	Initial I.Q. Level	Later I.Q. Level	
1	C.G.	1954 I.Q. = 20	1956 I.Q. = 24	1957 I.Q. = 32
2	D.B.	1955 I.Q. = 41	1956 I.Q. = 47	1957 I.Q. = 49
3	L.P.	1955 I.Q. = 45	1958 I.Q. = 47	
4	S.B.	1954 I.Q. = 25	1955 I.Q. = 29	1957 I.Q. = 22
5	G.A.	1956 I.Q. = 50	1957 I.Q. = 56	
6	R.H.	1949 I.Q. = 61	1951 I.Q. = 65	
7	A.A.	1951 I.Q. = 45	1952 I.Q. = 60	
8	A.W.	1951 I.Q. = 58	1954 I.Q. = 67	1958 I.Q. = 68

The importance of anticonvulsants in producing mental changes in epileptics has been stressed by a number of authors in the past, for example, Stauder (1938) but denied by others, *e.g.*, Loveland, Smith, and Forster (1957). Patients in both cases and the control series had all been on various drugs for various periods and a rating scale had, therefore, to be devised for purposes of comparison. Daily doses of 60 mg. phenobarbitone, or 100 mg. phenytoin, or 0·5 g. of primidone were regarded as approximately equivalent and rated at one point each, larger doses being rated proportionally. Up to three points was classified as a moderate dosage, three to five as high, and above five as very high. Table VI shows that there is no significant difference in the level of dosage of drugs in the controls and the cases, nor is there any difference in the number of years of medication.

A wide variety of emotional and behaviour disorders were seen in both groups but the most important concerned temper tantrums and other aggressive manifestations. These were equally common in both cases and controls.

Finally Table VI shows that there are no significant differences in the family histories of epilepsy in the cases and controls.

Table VII shows similar factors investigated and found not different in the cases of the Fountain

TABLE VI

FACTORS INVESTIGATED AND FOUND NOT DIFFERENT BETWEEN MAUDSLEY CASES AND CONTROLS

	Cases	Control
1 *Site and extent of brain damage*		
Unilateral	9	10
Bilateral	11	10
2 *Presumptive age when brain was damaged*		
Conditions during birth	5	6
Conditions after birth	7	6
Unknown	8	8
3 *Age at onset of seizures*		
Before 6 months	2	1
6 months to 4 years	8	9
After 4 years	10	10
4 *Amount and duration of drugs*		
Amount: Slight or moderate	6	8
Amount: High	13	8
Amount: Very high ($\lambda^2 = 3{\cdot}278$, not significant)	1	4
Duration: 2 to 5 years	4	6
Duration: 5 to 15 years	16	14
5 *Emotional and behavioural problems*		
Disturbance of function	13	13
Conduct disorders	2	—
Nervous traits	1	1
Normal	4	6
6 *Incidence of epilepsy and related diseases (e.g., migraine, mental illness, mental deficiency) in families*		
Epilepsy	6	6
Others	3	3
Negative	11	11

TABLE VII

FACTORS INVESTIGATED AND FOUND NOT DIFFERENT BETWEEN FOUNTAIN HOSPITAL CASES AND CONTROLS

	Cases	Control
Age at onset of seizures		
Before 6 months	3	1
6 months to 2 years	3	6
2 years to 4 years	2	1
Amount and duration of drugs		
Amount: Slight	5	5
Amount: Moderate	3	3
Duration: 2 to 5 years	1	1
Duration: 5 to 15 years	7	7
Incidence of epilepsy and related diseases (e.g., migraine, mental illness, mental deficiency) in families		
Epilepsy	2	1
Others	2	2
Negative	4	5

Hospital material. As would be expected, the cases in this hospital all had an earlier onset of epilepsy than in the majority of the Maudsley cases. Both groups of cases had been on lower anticonvulsant dosage than in the majority of the Maudsley patients and again there was no evidence of any difference between the cases and the controls. In the same way, the family history of epilepsy was not different in the two groups.

Table VIII shows factors investigated in the Maudsley material which show significant differences between the cases and the control groups. The frequency of seizures refers to the time before the patient had been on any regular anticonvulsant treatment. As regards major seizures, infrequency was regarded as not more than one seizure a month;

TABLE VIII

FACTORS FOUND DIFFERENT BETWEEN MAUDSLEY CASES AND CONTROLS

	Cases	Controls	λ^2
Number and type of seizures			
Status or very frequent (M_1 and M_2)	7	2	6·76
Frequent M_1	1	1	
M_1 and M_2	6	3	P < 0·05
M_2	3	4	
Infrequent M_1	2	4	
M_1 and M_2	1	6	
Results of medication on seizures			
Controlled (all types)	3	12	15·0
Controlled partially	—	3	P < 0·001
Reduced	15	5	
Unaffected	2	—	
Type of E.E.G. abnormality			
Focal	4	15	12·92
Focal and general	13	5	P < 0·01
General	3	—	
Previous mental status			
Feeble minded	7	1	5·664
Dull average	3	5	0·1 < P < 0·05
Average or above	10	14	
Environmental and social factors			
Disturbed environment	14	8	3·636
Normal	6	12	0·1 < P < 0·05

M_1 = major seizures. M_2 = minor seizures.

frequent attacks from two to 10 a month; and very frequent more than 11 a month. In the case of minor seizures, infrequent attacks were less than two seizures a week; more frequent attacks from two seizures a week to 10 a day, and very frequent attacks, more than 11 seizures a day. The difference between the cases and the control groups is significant at the 5% level; the effect of medication on the seizures shows an even more definite difference. In the case of the control groups, all types of attack became very infrequent, but in the majority of the cases the attack frequency was merely reduced.

The E.E.G.s of both the Maudsley and the Fountain cases were done at the Maudsley hospital and reported on by one of us (D.A.P.). They were divided up into the three groups as far as possible without knowledge of the clinical findings. The E.E.G.s were classified as focal only if they showed against a normal background focal disorders, whether these were spikes, sharp waves, slow waves, or combinations thereof. The focal and generalized abnormalities showed not only the focal changes already mentioned, but also generalized irregular disorders such as slow spike-and-wave complexes or an excess of very slow activity of whatever form. Table VIII clearly shows that the patients much more often had a combination of focal and general changes in contrast to the controls, who had mostly focal abnormalities only.

The previous mental status refers to the state that the patient showed before deterioration occurred. It shows that there is a tendency for the already dull child to deteriorate more than those who are average or above. As a group, of course, both the cases and the controls show a range of intelligence well below average since both groups have cases of epilepsy with brain damage.

Lastly, there seems to be some evidence that the social and family environments of the deteriorated child are more disturbed than those of the control cases.

Table IX shows the data on the Fountain Hospital material as regards effects of number and types of seizure, the results of medication, and the type of E.E.G. abnormality. The numbers in these cases are

TABLE IX

FACTORS SUGGESTIVE OF DIFFERENCES BETWEEN FOUNTAIN HOSPITAL CASES AND CONTROLS

	Cases	Control
Number and type of seizures		
Status and very frequent M_1	5	2
M_1 and M_2	2	—
Frequent M_1 and M_2	1	3
M_2	—	1
Infrequent M_1 and M_2	—	2
Results of medication of seizures		
Controlled	1	3
Reduced	6	5
Unaffected	1	—
Type of E.E.G. abnormality		
Focal	—	3
Focal and general	3	1
General	5	4

M_1 = major seizures. M_2 = minor seizures.

small and no significant statistical analysis can be made. However, the figures show the same general tendencies that were found in the Maudsley material.

Not brought out in these tables are the various behavioural changes that accompany the deterioration. Confusional periods lasting up to hours are sometimes seen, with or even without preceding major or minor attacks, so that these episodes do not seem to be typically post-ictal. There is also a progressive withdrawal of interest so that the child appears dull, unresponsive, and even at times frankly autistic, with mannerisms typical of the psychotic child. Violent tantrums and rages may alternate with these periods of apathy. Speech often becomes slow and monotonous in delivery with a progressive loss of vocabulary and responsiveness.

Discussion

Before commenting on the significance of this material, some relevant neuropathological findings should be mentioned. Three of the cases and one patient in the control group have had a temporal lobectomy by Mr. M. A. Falconer, in the Guy's-Maudsley Neurosurgical Unit. The neurological data kindly supplied by Dr. J. P. Cavanagh, of the Department of Neuropathology at the Maudsley,

showed diffuse microscopic lesions in all four cases. The changes are similar to those found in other temporal lobectomy cases at various ages and cannot be regarded as in any way specific to deterioration. How far out from the temporal lobe area the diffuse damage spreads cannot, of course, be told from biopsy specimens without full post-mortem studies.

The close correlation with the frequency of seizures and particularly with the efficacy of their control by anticonvulsants makes it tempting to consider that the fits themselves may cause deterioration as has been put forward by a number of authors, particularly Scholz (1951). However, in at least two cases there is clear evidence that the process of deterioration may be reversed, in the one case, following a temporal lobectomy. As this reversibility is an important point, the two cases are briefly described here.

C.F., born on May 14, 1944, was the daughter of a professional man. Pregnancy and birth were normal although she showed considerable feeding difficulties for the first three months of life that were shared with the other children. The milestones of sitting up, walking, and talking were quite normal, but at 2½ years of age she was regarded as abnormally restless. Shortly before her third birthday, a month after the arrival of the new baby, of whom she was obviously jealous, major seizures began and rapidly became daily. The fits often started in the right arm and leg only. At this time she had a temporary right flexid hemiplegia and aphasia. The fits continued to recur in groups affecting sometimes right and left sides only. Subsequently she began to have frequent minor attacks of a kinetic type and she became abnormally tearful and fidgety. After a few weeks her behaviour at home became an acute problem and she was wilful and screaming with any frustration. Psychological tests at this time gave her provisional Binet I.Q. as 72. In spite of full doses of anticonvulsants the fits continued as did the mental deterioration and she had to be admitted at the age of 5 to an epileptic colony. For the next few years the fits diminished in number, and she then became free of fits for several years. By 1958 she was able to leave the colony and her I.Q. on testing was about 93. She was just able to hold her own in an ordinary school though still somewhat emotionally unstable.

The second case, D.L. born on January 26, 1943, had a normal birth and early development. At the ages of 2 years, 3½ years, and 4 years he had groups of fits but then the fits became much more frequent and he was admitted to the Maudsley Hospital at the age of 10 years. Up to this time he had managed to hold his own in an ordinary school but with increasing difficulties, especially as regards behaviour. He was discharged from the Maudsley to an epileptic colony, but his stay lasted only two months when his violent behaviour necessitated his admission to a remand home. An I.Q. test in 1951 gave a score of 93 on the W.I.S.C. but by 1954 the score had dropped to 80. E.E.G.s showed several epileptogenic areas, the most active of which was in the left temporal region. The A.E.G. was normal and there were no neurological signs except for a right homonymous hemianopia. A left temporal lobectomy of standard type was carried out on July 27, 1954. Following the operation his fits were controlled but for many months his behaviour remained difficult and his I.Q. test scores continued to fall. However, he was then sent to a special residential school for maladjusted children and began to make good progress so that by 1958 his I.Q. score had returned to 94 and he was able to go home and start a normal job free of fits. It is of interest that at this time his E.E.G. was relatively normal as regards background rhythms and absence of foci but he had a few bursts of generalized spikes and waves of centrencephalic type which have been in one or two other adult temporal lobectomy cases (Hill, personal communication).

The reversibility of mental change is in contrast to the lack of progress shown by infants with so-called hypsarrhythmia (Gibbs, Fleming, and Gibbs, 1954) or infantile spasm (Illingworth, 1955). This latter condition differs from the present series of cases in showing an earlier and simultaneous onset of fits and deterioration. Like the present series it is probably only a clinical syndrome with a variety of possible causes.

This investigation thus stressed the comparative unimportance of certain factors in the production of deterioration: the presumptive age of brain damage; the number of years fits have continued; the quantity of anticonvulsants and adverse social and psychological factors do not seem very important. On the other hand, the close correlation with the number of attacks and the apparent reversibility of deterioration when the attacks are brought under control would suggest that there may be some sort of 'subclinical' epilepsy which causes deterioration.

Such a concept has long been upheld by older authorities, and terms such as 'epileptic equivalent' or 'epileptoid psychopath' imply a similar mechanism. However, to be effective such discharges must be of a certain type or affect certain areas and not others, since, for example, patients with pure petit mal and frequent generalized symmetrical three-per-second spike and wave do not appear to suffer any ill effects from such discharges. Several authors have pointed out the resemblances between the behavioural disturbances and learning difficulties seen in epileptics and the effects in animals of epileptogenic lesions in the rhinencephalic regions or visceral brain loosely so-called.

It is probable that the patients in this series had lesions in these regions but so probably had some of the control cases and we are still ignorant of the reasons why some lesions seem so much more disturbing than others.

Summary

Twenty-eight epileptic children from the Maudsley and Fountain Hospitals who showed intellectual and social deterioration were compared with a control group of epileptics who had similar evidence of brain damage but no deterioration.

Significant differences were found in the increased frequency of seizures, the poor effects of medication on the seizures, and the incidence of focal plus generalized E.E.G. abnormalities in the cases.

Insignificant differences were found in the site and extent of brain damage, the presumptive age when the brain was damaged, the age of onset of seizures, the amount and duration of anticonvulsants, and the associated emotional and behavioural problems.

Attention is drawn to certain cases which improve after long periods of apparent deterioration and a hypothesis is put forward that some form of 'subclinical' epilepsy may be partly responsible for deterioration which is not a true dementia.

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