ii. Physical Aspects

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The psychiatric aspects of the investigation of these patients having been covered in the first of the papers of this series, it is the purpose of the present paper to provide details of physical factors, especially those relating to the epilepsy.

Past Medical History

The relevant data of the past medical history of the 69 patients are shown in Table 1. This shows that in an impressive proportion of cases events have occurred which could well have been productive of an organic lesion, which might have been responsible for the epilepsy. In all, only 29 of the 69 patients were entirely without such a history. In five patients the history suggested birth trauma, with instrumental delivery and marking of the head in 4 cases; in one case (*8*) there was breech presentation with labour lasting 40 hours. One of these birth trauma patients (*21*) also had encephalitis accompanying pneumonia at the age of two.

In 18 cases there was a history of head injury occurring later in life. In 7 of these the injury seems unlikely to have been serious, although in one case (*30*) in which the injury occurred at the age of two the fits began shortly afterwards. In 7 cases injury was of a more material kind, with a history of concussion and fits following in close sequence in 4 cases. In one of these (*68*) there were three head injuries, with periods of unconsciousness of 0, 15 and 30 minutes. This patient also had a history of mastoid disease. In another case (*20*) a blow on the back of the head, which had not caused concussion, had been followed by rhinorrhoea; epilepsy did not begin until the age of 40.

Four patients had suffered severe head injuries. In one case (*6*) this was at the age of 17, and the patient was kept in hospital for two months; the fits may have begun soon afterwards, but were not prominent until six years later. Another patient (*27*) sustained a fractured skull at the age of 38, and was unconscious for two days and confused for three weeks; the fits came on in the following year. In another case (*41*) epilepsy may have preceded the head injury; but at the age of 15 the patient fell and fractured the left temporal bone and developed a large extradural haematoma. In the fourth of these cases of severe head injury (*58*), at the age of 22 the patient sustained an injury which resulted in unconsciousness lasting fifteen hours; however, he had suffered from fits from the age of 11.

Infections of the middle ear and mastoid bone were reported in six cases (*2*, *16*, *32*, *65*, *67*, *68*). In one of these (*2*) a mastoid operation at 27 was followed by a temporal lobe abscess, and fits began in the following year. In case *32* the patient suffered from bilateral otitis media at the age of 16; his epilepsy started at the age of 23, but it is reported that he had four grand mal seizures at the age of two. In case *65*, there was otitis media in early childhood, and onset of fits at 14. Another patient (*67*) had mastoiditis at the age of six and was not operated on. Her fits may have begun at the age of 15, but were not prominent until 29 after the onset of the psychosis. The head-injured patient (*68*) already referred to had a mastoidectomy at 23 followed by epilepsy at 25.

Encephalitis and meningitis are recorded in six cases (*3*, *11*, *21*, *37*, *39*, *59*). In one of these (*3*) meningitis at the age of four was followed by epilepsy at 11. Another patient (*11*) had scarlet fever at the age of 13 accompanied by delirium; fits began at 21. Another patient (*37*) had meningitis at the age of 10 months, which kept her in hospital for over a year. There were some fits accompanying the disease, but no more until they began again at the age of 10. Another patient (*39*) had meningitis at the age of 10 which left her with hearing affected on the left side; epilepsy began at 19. The last patient in this group (*59*) had some febrile illness at the age of nine days, and may have had encephalitis;

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the epilepsy began at two.

There are two other cases which have to be mentioned. In one of these (*40*), a cerebral thrombosis at the age of 18 months left the patient with an upper motor neurone hemiplegia; the fits began shortly afterwards. Another patient (*62*), a miner, suffered from coal gas poisoning at the coal face at the age of 45. It is not known how long he was unconscious. His epilepsy began at 49.

There is, therefore, a total of 28 cases in which the history suggestive of a cerebral lesion is thought to be significant. The distribution of these cases according to clinical group is shown in Table I. It will be seen that four out of 11 cases in Group A and 23 out of 46 cases in Group B, i.e., nearly half of the cases in these groups, show a positive history. On the other hand only one of the 12 patients in Group C had such a history of possible brain trauma. The difference between the C Group and the other groups taken together is highly significant. By the exact test, the probability of making this finding or a more extreme one by chance is less than 0·011.

The History of Epilepsy

We have analysed the history of the patients' epilepsy from several standpoints:—

(*a*) the age of onset of the epilepsy, and in relation to it the age of onset of the psychosis;

(*b*) the quantity of medication taken before the onset of the psychosis;

(*c*) the severity of the epilepsy, as judged by the frequency of the grand mal attacks, and by the number of grand mal attacks sustained before the onset of psychosis;

(*d*) the extent to which the grand mal attacks were controlled by anti-convulsant medication;

(*e*) the clinical diagnosis of the type of the epilepsy;

(*f*) whether the grand mal attacks were predominantly nocturnal or diurnal.

It will be seen that so far as the features (*c*), (*d*) and (*f*) above are concerned, we have dealt only with grand mal attacks. These, of course, do not form a homogeneous group from the point of view of electro-encephalography, physiology or aetiology. Although there is a multitude of varieties of epileptic attack, broadly speaking they can be divided into four types clinically, and each of these includes one or more electro-encephalographic groups:—

1. Grand mal without an aura. This clinical presentation may have as its cause either a centrencephalic grand mal or a focal attack originating in a silent area. If the attack is recorded on the EEG, these are easily distinguished in the majority of cases.

2. Petit mal, a transient loss of consciousness without convulsion or premonitory symptom. This is centrencephalic in origin and in the great majority of cases the EEG is pathognomonic.

3 and 4. Focal or Jacksonian epilepsy. This may manifest itself in one of two ways: aural symptoms (i.e. symptoms occurring as part of

Table I

History of	A	B	C	Totals
Birth trauma		5 8 34 50	21†	5
Head injury, moderate	51	19 20 42 43 46 68*		7
Head injury, severe	6	27 41 58		4
Middle ear and mastoid disease	16 32	2 65 67 68*		6
Encephalitis		3 11 37 39 59	21†	6
Cerebral thrombosis		40		1
Coal gas poisoning		62		1
Totals	4	23	1	28

Case numbers of patients with histories suggesting brain damage, in the three clinical groups.

*, † These cases had evidence of two types of brain damage in the history.

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the epileptic aura) followed by a grand mal, or aural symptoms which are not followed by a grand mal. From the physiological point of view these are equivalent. When considering the localization of the lesion, it is immaterial whether the epileptic discharge spreads more generally and produces a grand mal, or whether the discharge remains localized to the focus with only aural symptoms produced. Admittedly there are differences from the point of view of the patient, and perhaps, if the grand mal attacks have a deleterious effect on the brain, from the prognostic viewpoint. We would include amongst aural symptoms all the epileptic equivalents.

It is, of course, recognized that this is a rough and ready classification; that many patients exhibit more than one variety of fit; and that the varieties of attack which are associated are of considerable significance. Petit mal is often associated with grand mal in the same patient; in these cases the grand mal is usually part of a centrencephalic epilepsy. In other cases aural symptoms or equivalents occur in association with grand mal attacks, or may precede them by many years; in these cases the grand mal attacks are usually focal in origin. It is also not forgotten that fits are often not to be placed unambiguously into one category or another. This is particularly the case with focal epilepsy, where the patient may have aural symptoms and a few twitches not amounting to a convulsion.

It is also well recognized that grand mal attacks which are originally associated with an aura may cease to be so associated after a few years, i.e., the aura disappears. This may be due to the fact that the fits are originally focal, but there is an increase in the number of foci as the epileptic history lengthens. One of our cases (*46*) illustrates this. Originally he had an aura "as if a lot of people were shouting 'yes, you do it, no you don't' ". After about eight years the aura became less and less frequent. The EEG 12 years after the onset showed multiple independent spike foci at left sphenoidal, right sphenoidal, right frontal and right mid-temporal areas; there was moreover a gross excess of generalized slow activity.

For the purposes of (*c*), (*d*) and (*f*) above, we have concerned ourselves only with grand mal attacks, whether preceded by an aura or not. We have not considered petit mal attacks or aural symptoms, which may be of any degree of complexity from a momentary experience of fear to complex automatic behaviour or a mood change lasting for days, unless these have been followed by a definite spontaneous convulsion. There is justification for this in that from our experience of pyknolepsy it is generally accepted that petit mal attacks do not leave any sequelae in the way of permanent brain damage; and the same is probably true of aural symptoms not followed by a convulsion—though of course when the aural symptoms are complex the psychological reactions may be profound.

a. The age of onset of the epilepsy in relation to the age of onset of the psychosis.

The relevant data on this aspect are shown in Table II.

From this table it will be seen that there are marked differences between the three groups in respect of age of onset of epilepsy, the biggest difference being between Group C and the others; that the difference between groups is smaller in respect of age of onset of psychosis; and that there is practically no difference between the three groups in respect of the difference between these ages, i.e., the duration of the epilepsy before the appearance of the psychosis.

An analysis of variance was carried out on the raw ungrouped data to test the significance of these findings. In the case of age of onset of epilepsy the ratio of between-group variance to within-group variance was 4·24, $\cdot 05 > P > \cdot 02$. With age of onset of psychosis the non-significant value of F of 1·72 was obtained. In the case of duration of epilepsy, with an F value of 0·24, between-group variance was only a fraction of the within-group variance.

It appears, therefore, that the groups differ significantly in age of onset of epilepsy, but not in the other respects. Whatever the age of onset of epilepsy, the psychosis tends to come on at a relatively standard time thereafter. Calculation of the correlation between ages of onset of epilepsy and of psychosis provides a correlation coefficient of 0·58. The suggestion arises that it is the duration of the epilepsy, rather than either

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age of onset, which plays an aetiological role in determining the psychosis.

Some further commentary is needed. Rather different figures would have been obtained if, for age of onset of epilepsy, we had taken the age at which the first grand mal occurred. In case *49*, the patient had *déjà vu* and temporal lobe experiences from the age of 10, but the first grand mal at 29; in case *52* automatic behaviour of the temporal lobe type occurred from the age of 11, but the first grand mal was at 44; in case *55* either petit mal or temporal lobe attacks occurred from the age of three (the EEG shows both centrencephalic and temporal lobe origins), the first grand mal occurring at 15; in case *29*, Group C, petit mal began at five, grand mal at seven. In five cases (*12*, *18*, *35*, *63*, *66*) there were no grand mal attacks.

The age of onset of the epilepsy was usually easy to determine. The main difficulty was to assess the significance of a few convulsions without localizing features, sometimes associated with teething or a febrile illness during the first few years of life, after which the patient had no further fits for a number of years. The occurrence of fits under these circumstances might not be without significance, since they could mark an illness in which damage to the brain had occurred, or could be an indication of a genetical predisposition. It would, however, be misleading to accept them as showing the time of origin of a syndrome requiring treatment and having its own natural history. They have therefore been disregarded in fixing the age of onset, which has been taken as that age at which fits became definitely established.

Five patients had infantile convulsions followed by an interregnum of a few years before grand mal attacks started again: in Group B, *37*, a few convulsions in infancy associated with meningitis, grand mal attacks at 10; *46*, a few convulsions, cause unknown, when a few weeks old, grand mal attacks from 15; *61* a few convulsions in infancy, cause unknown, grand mal from 10; in Group C, *44*, two convulsions when aged two, cause unknown, grand mal from 15; *57*, one convulsion at two-and-a-half when cutting molars, petit mal from four, grand mal from 16.

There was an unusually short duration of epilepsy before onset of psychosis in six cases; in four of these the duration was less than one year, (*27*, *41*, *60*, *66*), and in two others less than two years (*11*, *56*). In some of these cases the relationship between the two onsets is dubious. Thus in Case *11*, the patient was first admitted to hospital at the age of 32 with a history of having had both fits and ideas of reference for the past 10 years. In Case *27*, the patient came

TABLE II

	Onset of Epilepsy				Onset of Psychosis				Difference			
Age	A	B	C	All	A	B	C	All	A	B	C	All
0– 4	—	7	3	10	—	—	—	—	1	8	2	11
5– 9	1	2	3	6	—	—	—	—	2	10	3	15
10–14	3	16	3	21	—	4	1	5	2	10	1	13
15–19	2	11	3	17	1	3	5	9	3	5	2	10
20–24	2	2	—	4	—	9	3	12	2	4	3	9
25–29	2	1	—	3	3	8	1	12	—	6	1	7
30–34	—	1	—	1	1	8	1	10	1	2	—	3
35–39	1	4	—	5	1	5	1	7	—	—	—	—
40–44	—	1	—	1	3	1	—	4	—	—	—	—
45–49	—	1	—	1	1	5	—	6	—	1	—	1
50–54	—	—	—	—	1	1	—	2	—	—	—	—
55–59	—	—	—	—	—	2	—	2	—	—	—	—
Total	11	46	12	59	11	46	12	69	11	46	12	69
Means	20·5	16·3	9·3	15·7	36·1	30·3	22·1	29·8	15·6	14·0	12·8	14·1
S.D.'s.	8·7	10·3	5·9	9·9	9·9	11·8	7·7	11·6	9·0	10·2	8·5	9·7

Distribution of ages of onset of epilepsy and psychosis, and of the duration of the epilepsy before onset of psychosis, in the three clinical groups and in the material as a whole; with means and standard deviations

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under observation at the age of 53, with a history of having had a fracture of the skull in an explosion at Woolwich Arsenal at the age of 38; within a year his eventual epilepsy was showing itself in periodic nocturnal incontinence and automatisms, and his psychosis in suspicions of his wife's fidelity. In Case *41* it is impossible to date the onset of the epilepsy with certainty. At the age of 12 the patient began to have inexplicable attacks of temper which would end in a deep sleep, and also at times to fall from her pony. In one of these falls at the age of 15 she sustained a subdural haematoma; and after this came attacks of unconsciousness without convulsion, and periodic rages and moods of depression.

In the remaining cases of exceptionally short duration of epilepsy, the epilepsy and the psychosis can be dated more accurately. In Case *60*, at the age of 19 the patient had a single convulsion which passed off into a prolonged confusional episode with disorientation and hallucination. She misidentified people, was emotionally labile, noisy and aggressive. The psychotic state persisting, she was eventually diagnosed as suffering from schizophrenia, and given electro-shock and insulin coma treatments. In Case *66*, the patient had her first fit, clinically of the temporal lobe variety, at the age of 16. She then began to develop wild rages, and over the years became increasingly psychotic. In Case *56*, the patient developed normally until the age of 11. He then had rheumatic fever, and this was followed by a personality change in the direction of irritability, laziness and apathy. He had his first fit at the age of 17. When he was 18 he developed symptoms of a florid schizophrenia, which over the years has become less florid, so that he now presents the picture of a chronic deteriorated schizophrenic. One cannot exclude the possibility that the pre-psychotic personality change might have been the first manifestation of an insidious psychosis.

b. Quantity of medication.

On this subject there is a voluminous literature, in which arguments have been raised for and against the hypothesis that the mental changes associated with epilepsy are due to medication with anti-convulsant drugs. It is not usually possible to determine in any accurate way how much anti-convulsant medication a patient has received over a period of many years, during which the drugs have been changed from time to time. We have not attempted to assess the quantity of individual anti-convulsants used with each patient, but have had to content ourselves with a rough classification. Nearly all the patients have been on phenytoin sodium and phenobarbitone for long periods, with or without other anti-convulsants; and an average amount would be phenytoin sodium 300 mgm. with phenobarbitone 100 mgm. daily, or their approximate equivalent of other anti-convulsants. This dosage has been designated **, significantly more than this as ***, and less as *. If the patient had no anti-convulsant medication, he was placed in the category 0. Though this is but a crude classification, it should show the effects of any existing relationship between the psychosis and intake of drugs. The data are shown in Table III.

TABLE III

Group	Quantity of medication				
	0	*	**	***	Total
A	2	2	7	0	11
B	4	16	24	2	46
C	0	6	6	0	12
Total	6	24	37	2	69

Quantity of anticonvulsants administered. **=a level of medication average by ordinary clinical standards, ***=significantly more than this, *=significantly less.

It will be noted that as many as six of the patients received no medication between the onset of the epilepsy and the psychosis. Of the two patients in Group A, one (*32*) had only six grand mal attacks before the onset of psychosis. Though he had four grand mal attacks at the age of two (which have been disregarded), he had only six grand mal attacks from the age of 23 when he began to have "minor" attacks with temporal lobe auras and 35 when the psychosis became permanent; after some of these six grand mal attacks he had confusional episodes lasting a few days, with a florid religiose content. The other patient in group A (*60*) had a confusional

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episode following a single first fit at the age of 19, two months after a confinement. However, this episode was of such duration and severity that she was diagnosed as suffering from schizophrenia and given 18 ECT and a course of 30 insulin comas. In Group B there are four cases (*14*, *20*, *25*, *67*); the first three of these had respectively one, three and 10 grand mal attacks in periods of ten, seven and two years between the onset of epilepsy and psychosis. In the last case, grand mal attacks occurred only after the onset of the psychosis.

From the table it seems clear that an excessive quantity of medication is not a factor in the production of a schizophrenic-like psychosis. 37 of the patients had what we would regard as an average level of medication, judging by ordinary clinical standards. 30 patients had less than that (six of them having none at all). Only two patients had what we would regard as a significantly heavier level of medication than is needed for the average patient.

c. The severity of the epilepsy.

There are those who believe that the cause of the mental changes in epileptic patients is the cumulative ill effects of the fits, perhaps through the anoxic stress imposed on the brain during a major convulsion. We therefore considered both the frequency of fits in our patients and the number of grand mal attacks undergone before the onset of psychosis. With our data it was not possible to classify the patients in respect of frequency of fits in any well defined way. Apart from the fact that information about the remote past is not wholly reliable, the frequency of fits in an individual varies from month to month, and generally shows a tendency to diminish as the patient gets older. We have arbitrarily regarded 12 grand mal attacks per year as an average fit frequency, and have rated this frequency as **. Significantly greater and lesser frequencies have been rated as *** and * respectively. In those patients with a long history of epilepsy, whose fits diminished greatly in course of time, either spontaneously or as the result of treatment, we have taken the fit frequency as that of recent years rather than earlier ones. The data obtained in this way are shown in Table IV.

TABLE IV

Group	Frequency of the Grand Mal Attacks				Totals
	0	*	**	***	
A	—	6	3	2	11
B	3	18	11	14	46
C	2	5	2	3	12
Totals	5	29	16	19	69

Severity of the epilepsy, as judged by frequency of grand mal attacks: ** signifies about 12 attacks per annum.

The table shows three patients in Group B and two patients in Group C with zero frequency of attacks. These are the patients (*12*, *18*, *66* in Group B, *35*, *63* in Group C), previously mentioned, who had no grand mal attack before the onset of the psychosis.

It seems from this table that our psychotic epileptics are not distinguished from the generality of epileptics by the severity of their epilepsy, if our criterion of 12 fits per annum is accepted as a modal figure. In practically half of our patients the fit frequency was substantially less than this, while little more than a quarter of them had a substantially higher frequency. Allowing for the small numbers of patients in Groups A and C, there does not seem to be any difference between one Group and another in respect of fit frequency.

A rough estimate could also be made of the total number of major fits sustained by each patient before the onset of psychosis. This was done by considering in each case the frequency of the fits at various stages in the illness, and multiplying number of fits per annum by number of years over which that frequency was maintained with final summation of the products. In five cases the data were insufficient for this calculation. As the scatter was so large, the distribution of cases by total grand mal attacks sustained, as shown in Table V, has been drawn up on a logarithmic scale.

The table does not suggest that total number of fits sustained plays a major role in precipitating the schizophrenia-like psychosis. While about one-third of the patients have had a large number of fits, about 40 per cent. have had a very small number, 31 or less. There are no conspicuous differences between the three

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TABLE V

No. g.m. attacks	Clinical Groups A	B	C	Totals
0	—	3	2	5
1– 3	2	4	2	8
4– 10	1	8	1	10
11– 31	1	1	2	4
32– 100	—	3	—	3
101– 316	3	8	1	12
317–1000	3	9	2	14
1001–3162	—	4	—	4
3500–5000	—	3	1	4
Not known	1	3	1	5
Totals	11	46	12	69

Case distribution of total numbers of grand mal attacks sustained (logarithmic scale).

clinical Groups. The most striking feature of the table is the wide scatter.

d. The control of the epilepsy by medication.

The concept of control by medication is not easily defined in any satisfactory way; we took as an adequate degree of control, rated as **, a reduction by medication of the spontaneous tendency to have fits to the point at which there was about one major attack per month. This was considered appropriate because there are two divergent views on the possible aetiological significance of control of the fits. On the one hand complete control by drugs may require such a quantity of drugs that the patient is made dull and retarded, and both mental and physical health are impaired. On the other hand some authorities consider that the fits themselves have a therapeutic effect against mental changes, such as irritability and depression, and even psychosis. There is a small group of epileptics in whom a convulsion may certainly have a good effect: these are the patients who have a prodromal period, perhaps of several days, with altered mood, aggressiveness and malaise, which is at once terminated by a spontaneous fit and may also be terminated by an artificially induced one. However, these patients constitute only a small fraction of the epileptic population.

Classifying the degree of control in our patients along lines which are now familiar, we reach the figures of Table VI. This table is,

TABLE VI

Group	Not applicable	*	**	***	Totals
A	2	4	5	—	11
B	7	18	19	2	46
C	2	2	6	2	12
Totals	11	24	30	4	69

Degree of control of grand mal attacks by medication; ** signifies a degree of control which reduces the frequency of grand mal attacks to about one per month.

unfortunately, not a very reliable one, as the frequency of fits before the patient was put on medication was often difficult to determine. The frequency of fits observed when a patient who has been on anticonvulsants ceases to take them for any reason cannot be regarded as an indication of the innate frequency. However, it was thought that the data were sufficiently reliable to show any outstanding relationship between control by drugs and mental changes, if it were there to be discovered.

The concept of control of grand mal attacks is not applicable in 11 cases. Of the two cases in Group A, in one (*32*) there were only six grand mal attacks, and in the other (*60*) only two. Of the seven patients in Group B who fall into this category, three (*12*, *18*, *66*) had no grand mal attacks, and the remainder (*14*, *20*, *25*, *67*) had only one, three, 10 and "a few" attacks respectively. Two patients in Group C (*35*, *63*) had no grand mal attack.

There is no evidence from these figures that over- or under-control of the fits by medication is a factor in the causation of the schizophrenic type of epileptic psychosis. About half of the patients, 30 in number, have been subjected to an average or moderate degree of control; 24 patients have been less rigidly controlled, and four have been more rigidly controlled. Again there is no conspicuous difference between any group and the others.

e. Clinical diagnosis of type of epilepsy.

The EEG diagnosis of the focus of origin of the epilepsy is probably more reliable than the clinical diagnosis, though of course there should be a certain degree of agreement, and the degree

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of correlation is a measure of the acumen of the clinical observer and the thoroughness of the EEG investigations. We have classified clinical diagnoses into four groups: (1) grand mal without an aura; (2) petit mal; (3) focal epilepsy originating in the temporal lobe; (4) focal epilepsy originating in other sites. The diagnosis of temporal lobe epilepsy was on the basis of the well recognized aural symptoms, and one or more of the following were regarded as indicating a focus in the temporal lobe:—hallucinations of smell, taste, hearing or vision, fear, masticatory movements, illusions, *déjà vu* or *jamais vu* experiences, epigastric sensations, and depersonalization or derealization. Automatic behaviour or dysphasia as part of the fit were also given the same significance. We did not analyse the clinical diagnoses by laterality, because none of the aural symptoms apart from dysphasia are of value for this purpose. It has been stated that *déjà vu* as an aural symptom is associated with a focus in the non-dominant hemisphere, but our data do not support this. The frequency of the various aural symptoms and their correlations with EEG findings are reported in the section on EEG findings.

TABLE VII

Group	Grand mal without aura	Petit mal	Focal temporal lobe	Focal other	Total
A	2	—	9	—	11
B	11	1	30	4*	46
C	1	4	6	1†	12
Totals	14	5	45	5	69

Clinical diagnosis of focus of origin on basis of aura. *2 in right sensory area, 1 in right and 1 in left motor area. †1 in right motor area.

It is obvious from this clinical assessment that temporal lobe epilepsy dominates the picture, especially in Groups A and B. However, as the EEG diagnosis is more precise than the diagnosis on clinical data, this point will not be pursued further here.

f. Timing of attacks in relation to sleeping period.

In his communication Pond stressed the association of the paranoid psychosis of epilepsy with nocturnal rather than diurnal fits. Our data relating to this point are shown in Table VIII.

TABLE VIII

Group	Timing of Epileptic Attacks: Diurnal	Nocturnal	Either	Total
A	1	7	3	11
B	13	5	28	46
C	5	1	6	12
Totals	19	13	37	69

Timing of attacks, "nocturnal" being those attacks which occurred during the sleeping period.

The striking point about this table is that of the 13 patients with only nocturnal epilepsy seven fell into the group of 11 patients in Group A, whose histories were characterized by the repeated occurrence of confusional states. The probability of this distribution or a more extreme one arising by chance is less than 0·001. Furthermore, 10 of the 11 patients in this group had some of their attacks at night. This finding is perhaps not entirely surprising. If a nocturnal fit is more likely than a diurnal one to be associated with a confusional state, a plausible explanation would be that during sleep the external stimuli which provoke a waking response are then largely absent, and even in the normal individual the nervous system is then more than ordinarily predisposed to fantasy and hallucination.

FINDINGS ON EXAMINATION

a. Clinical neurological findings.

The clinical neurological findings were, as a rule, negative. When positive signs were found they were nearly always minimal in degree, such as to demand special skill and experience for recognition with confidence. In two cases (*9*, *59*) an extensor plantar response was obtained, and in one case (*61*) one plantar response was ambiguous. In all there were 12 cases in which there was evidence of a pyramidal disturbance in the form of a difference in the reflexes or in a relative weakness of one side of the body. In five of these cases the weakness was most noticeable in the lower part of the face. In

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all but one (*14*) of these cases the functional impairment was shown on the left side. There was one case (*5*) in which there was impaired sensation on the right side of the body; one case (*10*) showed aphasia; and in one case (*25*) impaired sensation accompanied the motor deficiency. In all these cases showing neurological signs the EEG abnormality was found in one or both temporal lobes; in six the abnormality was bilateral, in three right-sided, and in five left-sided. It is noteworthy that in two of these last cases, abnormality in the left temporal lobe was associated paradoxically with a left-sided pyramidal disturbance.

We have to conclude, therefore, that clinical neurological signs when found provide highly significant evidence; but in the great majority of cases the neurological examination gives no help.

b. Psychiatric findings indicative of organic change.

Special attention was paid to findings in interview which might indicate organic personality change. Such changes are difficult to define, to assess and to classify, as the personal or subjective factor cannot be excluded. Nevertheless there is a good deal of consistency in independent judgments made by different examiners of experience on any single case; and both at the National Hospital and at the Maudsley all these patients were seen by more than one experienced clinician. The working classification eventually used was as follows:—

1. lack of spontaneity, slowness, dullness, retardation (34 cases);
2. impaired memory, forgetfulness, vagueness (29 cases);
3. stickiness and tendency to perseveration (22 cases);
4. stubbornness (21 cases);
5. tendencies in conversation to be concrete, ponderous, circumstantial, pedantic, long-winded (33 cases);
6. egocentricity, smugness or superiority, unctuous or grandiose utterances (23 cases);
7. religiosity (26 cases);
8. irritability (11 cases).

Despite the subjectivity of these observations, there is little doubt that the group of patients as a whole showed changes in these directions. Only 13 patients, or 14 if irritability is disregarded, are marked as free from any of these indications of organic change; the remainder showed from one up to seven. When they were brought into relation with one another, these findings showed a good deal of internal correlation, as is shown in Table IX. The traits numbered 1 to 4 were correlated more closely than the rest, and appear to form a nuclear cluster. Those numbered 5, 6 and 7 were also correlated with each other and with the first four traits, but less closely, i.e., with lesser statistical significance, and appeared therefore to be more independent. The last of the symptoms noted, irritability, was quite independent of all the others. This independence fits in with clinical impressions, since in the single case irritability varies greatly from time to time, and seems to be a manifestation of temporary and functional disturbance rather than of any permanent change.

The case material was also examined for indications of any correlation between the seven more significant personality changes, i.e., with the exclusion of irritability and other features of the illness. There was a connection between the duration of the epilepsy and the extent to which personality changes were shown. Thus, of

TABLE IX

Symptom of organic personality change	1	2	3	4	5	6	7	8
1. Lack of spontaneity, retardation	−	+	+	+	+	o	o	o
2. Impaired memory, vagueness	+	−	+	o	+	o	o	o
3. Stickiness, perseverativeness	+	+	−	+	+	+	+	o
4. Stubbornness	+	o	+	−	o	+	o	o
5. Concreteness, circumstantiality	+	+	+	o	−	+	+	o
6. Egocentricity, unctuous utterance	o	o	+	+	+	−	+	o
7. Religiosity	o	o	+	o	+	+	−	o
8. Irritability	o	o	o	o	o	o	o	−

Intercorrelations of signs of personality change; + signifies χ^2 significant at 0·05 level.

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the 36 patients whose epilepsy had lasted less than 14 years before the onset of the psychosis 11, or less than a third, showed as many as three or more of the named changes; but this was true of 18 out of 33, or more than half, of the patients whose epilepsy had lasted 14 years or longer before psychosis. This difference is significant at the 0·05 level ($\chi^2=4\cdot06$). Looking at the material in another way we find that the 26 patients with prepsychotic duration of epilepsy of 0 to 9 years had a mean number of personality changes of 1·9; the 23 patients with duration 10 to 19 years had a mean number of 2·2 personality changes; the 20 patients with duration in excess of 20 years had an average of 2·9 personality changes. However, memory impairment and irritability were not related to duration; and the degree of personality change was not related to severity of epilepsy or to amount of medication, and other correlations were not observed.

c. The electro-encephalographic investigation.

Electroencephalographic studies were made on all patients, and the majority had a series of recordings. The majority were also investigated by sphenoidal recordings with pentothal activation, and some were activated with bemegride, a wire being inserted into the sphenoidal region. In all there were 228 straight recordings and 134 sphenoidal recordings, 117 with pentothal and 17 with bemegride. On an average, therefore, there were about four straight and two sphenoidal recordings per patient. In each case the series of EEG recordings was surveyed by Dr. Cobb and his colleagues at the National Hospital, and by Dr. Hill and his colleagues at the Maudsley Hospital, apart from the present author. On the basis of all the EEG evidence available an EEG diagnosis was made in every case. These diagnoses are set out in Table X.

The EEG was classified into centrencephalic (grand mal and petit mal), secondary subcortical, cortical (temporal lobe, dominant, non-dominant and bilateral, and other) and generalized abnormality when the EEG showed a very considerable amount of dysrhythmia but could not be placed into any of the other categories.

The most striking feature of the series is the preponderance of cases with temporal lobe abnormality. An interesting negative finding is that it does not seem to be important whether the focus is in the dominant or the non-dominant temporal lobe, or whether both are affected. EEG foci were found in 16 patients in the dominant temporal lobe, and in 12 patients in the non-dominant temporal lobe, and 20 patients had bilateral temporal lobe foci; in 48 out of 69 patients, or nearly 70 per cent., the main abnormality consisted of one or more temporal lobe foci. In contrast to this, we see that only twice were foci found outside the temporal lobe.

Further examination of the data shows that the proportion of patients with EEG evidence of temporal lobe damage is even higher than this. For the purpose of Table X we took a single EEG diagnosis, and where a series of EEGs suggested, say, "mainly generalized

TABLE X

Group	Normal	Centrencephalic		Secondary subcortical	Cortical				Generally abnormal
					temporal lobe			Other	
		GM	PM		dom.	non-dom.	bi-lat.		
A (11)	—	—	—	—	3	1	4	—	3
B (46)	3	—	2	—	11	9	14	2*	5
C (12)	—	1	4	1	2	2	2	—	—
Totals (69)	3	1	6	1	16	12	20	2*	8

EEG diagnosis. *1 right (non-dominant) posterior frontal focus (33); 1 right (non-dominant) frontal pole focus (42).

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abnormality with occasional left temporal spikes", the diagnosis would be "generalized abnormality". If we consider all cases with evidence of temporal lobe involvement, we find that a further four patients had EEG evidence of temporal lobe abnormality, i.e., 75 per cent. of all cases showed evidence of a temporal lobe focus. The preponderance is particularly marked in groups A and B; temporal lobe foci were seen in only half of the 12 cases in group C.

It was earlier said that there should be some correlation between the clinical diagnosis and the EEG diagnosis. The EEG diagnosis is necessarily more precise in that, for example, clinical investigation cannot distinguish between centrencephalic grand mal and focal epilepsy starting in a silent area, unless the march of the fit suggests that one is in fact dealing with a focal epilepsy. Moreover, secondary subcortical epilepsy is an EEG diagnosis for cases in which the characteristics of the fit would not usually suggest a focal origin, the fit appearing to be centrencephalic.

With these considerations in mind, we examined our data for the degree of correspondence between the EEG diagnosis and the clinical diagnosis of temporal lobe epilepsy. The results are shown in Table XI. Combining the clinical and EEG evidence, we see that 55 patients out of the total of 69, or 80 per cent., had some evidence of temporal lobe epilepsy. In 38 out of these 55 cases there was agreement between clinical and EEG diagnoses; in seven there was clinical evidence of a temporal lobe focus which could not be demonstrated on the EEG; and in 10 cases there was EEG evidence of a temporal lobe focus, although the clinical history had been in no way distinctive. The fact that thorough EEG investigation may reveal a temporal lobe focus not suggested by the history in such a sizeable proportion of cases is a point of great importance. Some of these foci are, of course, amenable to surgery; and they would not be found if temporal lobe epilepsy were not looked for very carefully even in the clinically not very promising case.

TABLE XI

Group	Clinical and EEG	Clinical not EEG	EEG not clinical	Neither	Total
A	7	2	1	1	11
B	26	4	8	8	46
C	5	1	1	5	12
Total	38	7	10	14	69

Correlation between clinical and EEG diagnoses of temporal lobe epilepsy.

TABLE XII

	No. of cases	Localization of EEG abnormality							
		Centrencephalic		Secondary subcortical	Cortical temporal lobe			Other	Generalized
		GM	PM		dom.	nond.	bilat.		
Olfactory hallucinations	2	—	—	—	2	—	—	—	—
Gustatory hallucinations	3	—	—	—	1	2	—	—	—
Auditory hallucinations	5	2	—	—	1	—	2	—	—
Visual hallucinations	5	1	—	—	3	—	1	—	—
Feeling of fear	5	1	1	—	—	2	1	—	—
Motor accompaniments (chewing, etc.)	14	—	1	—	3	4	6	—	—
Déjà vu	8	1	—	—	—	1	5	—	1
Epigastric sensations	24	—	—	—	8	5	9	1*	1
Derealization	1	—	—	—	1	—	—	—	—
Dysphasia	15	—	—	—	7	3	4	—	1
Automatic behaviour	26	—	—	1	4	8	10	1*	2

Analysis of relationship of aural symptoms to EEG findings. * focus at r. frontal pole.

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The aural symptoms experienced by these patients were classified and correlated with the EEG findings; the results of this analysis are shown in Table XII. The findings are not greatly at variance with what might have been expected on the basis of generally accepted views. However, there are points of interest in the relative frequency of these symptoms, and the extent to which they are and are not specific of a temporal lobe focus. Epigastric sensations and automatic behaviour are much the commonest aural symptoms, and when found were shown almost exclusively by the patients with a temporal lobe focus. Motor accompaniments (chewing, etc.) and dysphasia are the next commonest symptoms. The latter was found in 14 patients with temporal lobe epilepsy, seven with focus in the dominant lobe, four with bilateral foci, and surprisingly three with focus in the non-dominant temporal lobe. In these three patients there was no reason to suspect a switch of laterality.

d. Air encephalography.

Air encephalography was carried out at least once in 56 cases. In one case it was twice attempted unsuccessfully, and in a few cases it was not done because of insufficient cooperation by the patient. In the Maudsley patients air encephalography was carried out as a routine procedure in every case. In the National Hospital patients, some of the early cases were missed; it was not until we were some way through the investigation that we began to realize the high incidence of organic lesions in these patients, and to appreciate that the AEG was a measure which should be carried out whenever possible in patients of this type. Table XIII shows the results.

Apart from the two cases where the AEG indicated a neoplasm, we classified the AEG abnormalities into:—(1) those cases with dilatation of one or both temporal horns, the rest of the ventricular system being normal; (2) cases with dilatation of the lateral ventricles, the temporal horns being normal; (3) cases with generalized dilatation of the ventricular system, either unilaterally or bilaterally; (4) cases in which the atrophic process affected the cortex rather than the white matter.

In 17 cases the AEG was regarded as normal. We have the impression that there was a slight difference between our radiological colleagues of the National Hospital, and the radiologists of the Guy's-Maudsley Neurosurgical Unit and Mr. Falconer and Dr. Hill, in the latitude allowed to "normality". However that may be, a judgment of "normality" is only provisional for another reason, since the picture may change over the course of years. In 11 cases repeat AEGs were carried out after various intervals, and in six cases progressive changes were shown. In one case (*14*), the AEGs in 1945 and 1956 were negative; but in 1957 there were indications of the neoplasm in the right hemisphere from which the patient eventually died. In five other cases atrophic changes were found in later examinations, previous findings having been

TABLE XIII

	Not done	Normal	Dilatation or atrophic process												Other change
			t. horn			l. vent.			vent. genrl.			Cortex			
			R	L	B	R	L	B	R	L	B	R	L	B	
Group A (11)	2	2	4	1	—	1	1	—	—	—	—	—	—	—	—
Group B (46)	9	12	1	2	—	1	1	1	1	5	4	1	1	5	2*
Group C (12)	2	3	—	—	—	—	2	3	—	1	—	—	1	—	—
Totals (69)	13	17	5	3	—	2	4	4	1	6	4	1	2	5	2*

Findings on air encephalography. Changes classified as showing on right, left or both; and as affecting the temporal horn, the lateral ventricle but not the temporal horn, the ventricular system generally, and the cortex.

* findings of space occupying lesion, 1 of neoplasm of right frontal pole, 1 with deformity of the right temporal horn in the supracornual cleft.

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negative. Futhermore, in two cases in which the AEGs were negative, temporal lobectomies revealed in one case (*15*) a small astrocytoma, and in another (*18*) a dermoid cyst. Too much weight, therefore, should not be put on purely negative information from the AEG.

With a few exceptions the nature of the AEG abnormality was that of an atrophic process. We may consider the exceptions. One patient (*16*) showed the scar of an old abscess of the right temporal lobe in a dilatation of the right temporal horn, together with a diverticulum. A patient (*5*) who had an angioma showed only the atrophy caused by deep X-ray treatment. In two cases (*14, 53*) the findings suggested a space-occupying lesion. The former patient, mentioned above, had a frontal glioma. In the latter case, the AEG showed a deformity of the right temporal horn in the supracornual cleft, suggesting a tumour occupying the anteromedial part of the right temporal lobe. This diagnosis was consistent with his epilepsy, which was Jacksonian in type. He would probably have come to surgery; but he had to be discharged prematurely on grounds not connected with his epilepsy or his psychosis. When seen two years later, in June 1960, there was no change in his psychosis, but his epileptic attacks were less frequent; there was no evidence of progression to support the diagnosis of a tumour.

In the remaining cases, 36 in number, the findings were all of an atrophic process, sometimes localized and sometimes generalized. In the majority of cases the central white matter was affected, in some cases the cortex, and in some cases both.

In summary, 11 patients showed generalized dilatation of the ventricular system including the temporal horns; 10 patients showed dilatation of the lateral ventricles, the temporal horns being normal; 8 patients showed dilatation of one temporal horn, the rest of the ventricular system being normal; and in 8 cases there was pooling of air over the cortex as the principal abnormality, with the abnormality limited to one side in 3 of them. Of the 29 patients who had atrophy of the white matter, 7 also showed pooling of air over the convexity; these 7 patients with both cortical and white matter atrophy showed a relatively high incidence of personality change. The table also shows that in 19 cases there was dilatation of one or both temporal horns, either as part of a generalized ventricular dilatation (11 cases) or alone (8 cases).

There is no indication of differences of distribution of the abnormal findings in each of the Groups A, B and C. In all groups, considering only the cases where the examination was made, about 30 per cent. of the patients showed no AEG abnormality and 70 per cent. showed an abnormal AEG. Where the AEG was abnormal, in 95 per cent. of cases the abnormality was that of an atrophic process.

e. Otological findings.

Otological investigation was carried out on 13 of the National Hospital patients; a clinical examination was made of the ear, nose and throat, and vestibular and cochlear functions were tested for signs of peripheral and central lesions. The method of examination of the central connections of the VIIIth nerve has been described by Carmichael, Dix and Hallpike (1954, 1961). In no case did clinical examination of ears, nose and throat reveal any abnormality. In 12 cases cochlear functions was normal; one patient showed a slight degree of deafness. In 5 of the 13 cases vestibular functions to the caloric tests were normal; and in the other 8 cases the responses suggested that one or other of the temporal lobes was affected. In 8 of the 13 cases there was agreement between otological and other findings, clinical, EEG, etc.; and in five cases there was some measure of disagreement.

Taking first the cases in which there was agreement, there were six patients in whom vestibular tests indicated a lesion of one or other temporal lobe, in conformity, both in respect of the existence of a lesion and of laterality, with all the other evidence available. There were two cases in which vestibular tests were normal, and there was no other evidence of a temporal lobe lesion.

The cases in which there was disagreement consisted of three cases in which the vestibular tests were normal, but in which there was other evidence of temporal lobe abnormality. The conflict of evidence in these cases is more

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apparent than real, since the vestibular tests relate to a possible lesion of the posterior part of the temporal lobe rather than the temporal lobe in its entirety. There were also two cases in which vestibular tests indicated temporal lobe abnormality, not supported by the other evidence. However, in both these cases the EEGs showed evidence of widespread abnormality, and this may well have involved the temporal lobe; temporal lobe epilepsy, however, was not the clinical or the EEG diagnosis in these cases.

These figures suggest that otological methods of diagnosis appear to be accurate and reliable; and that tests of vestibular function, when positive, are a valuable contribution to the diagnosis of temporal lobe epilepsy. It is a matter for regret in retrospect that we did not arrange for otological investigation in a more systematic way.

f. Psychometry

Psychometric testing was as a rule uninformative. Indications of intellectual impairment of a general kind were found in eight patients; and in one of these there were suggestions of a frontal lobe lesion. In none of these patients was the impairment severe, nor sufficient to produce a clinical impression of some degree of dementia.

In 5 patients (*6*, *14*, *21*, *44*, *57*) there were findings suggestive of a slight expressive dysphasia or an auditory learning deficit. However this was a less productive source of information than the clinical record. From the past history and the psychiatric clinical examination indications of dysphasia were found, as part of the fit itself in 14 patients (*5*, *14*, *15*, *23*, *25*, *32*, *37*, *38*, *41*, *44*, *48*, *54*, *64*, *69*) and as occurring between fits in eight patients (*10*, *15*, *21*, *23*, *24*, *37*, *44*, *65*). Table XIV shows the numbers of patients showing dysphasia in these various ways, the extent to which these 20 patients were found to be dysphasic by more than one method, and whether or not the EEG showed temporal lobe epilepsy. In fact, 17 of these 20 patients had temporal lobe epilepsy on the EEG, and three showed widespread general abnormality.

The most accurate indicator of a temporal lobe epilepsy seems to be the appearance of dysphasia as an ictal phenomenon. All of the 14 patients with ictal dysphasia were temporal lobe epileptics; whereas one out of the five patients with psychometric dysphasia, and two out of the eight patients with interictal dysphasia clinically, had generalized abnormality.

Neurosurgical operations

Eleven of our 69 patients were subjected to temporal lobectomy. Seven of them have been reported by Serafetinides and Falconer (1962), and need not be further discussed. The cases numbered III, VI–XI in their paper are respectively the cases numbered *64*, *32*, *30*, *18*, *26*, *9* and *15* in our series. The other 4 patients who had temporal lobectomies (*5*, *8*, *41*, *23*) together with 3 other patients subjected to other neurosurgical procedures, are reported in a tabular form, similar to that used by Serafetinides and Falconer (see Table XV).

In all these four patients, the family history

TABLE XIV

Dysphasia indicated by	Electroencephalographic Findings: Temporal lobe abnormality	Generalized abnormality	Total
Psychological testing, clinically ictal and interictal	1	—	1
Psychological testing, clinically ictal only	1	—	1
Psychological testing, clinically interictal only	1	—	1
Psychological testing only	1	1	2
Clinically ictal and interictal	3	—	3
Clinically, ictal only	9	—	9
Clinically interictal only	1	2	3
Totals	17	3	20

Numbers of patients with one or more indications of dysphasia, correlated with EEG findings.

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TABLE XV

Case No.	Sex	F.H.	Possible acquired aetiological factors	Age at onset Epilepsy	Age at onset Psychosis	Type of epilepsy Psycho-motor	Type of epilepsy G.M.	Type of psychosis	Pathological findings	Results of operation Epilepsy	Results of operation Psychosis	Follow-up period	Age and type of operation
5	F	Neg.	Difficult forceps delivery, marked L. forehead. Racemose venous angioma L. hemisphere	7	25	Yes	Yes	Confusional episodes, paranoid psychosis with aggressiveness	Racemose angioma L. hemisphere. Temporal lobe showed diffuse capillary angioma in cortex	Improved	Unchanged	1.5/12	13 Craniotomy for racemose angioma L. hemisphere, followed by D.X.R. 27 L. anterior temporal lobectomy
8	F	Neg.	Breech delivery. 40 hours labour	11	13	Yes	Yes	Paranoid psychosis	Nerve cell loss and gliosis with distortion of cortical architecture and additional amygdaloid damage	Improved	Unchanged	4.1/12	19 R. anterior temporal lobectomy
41	F	Neg.	Neg.	12	12	Yes	Yes	Chronic paranoid-hebephrenic schizophrenia	Some increase in astrocytes in the cortex	Much improved	Improved	4.10/12	23 L. anterior temporal lobectomy
23	M	Neg.	Neg.	17	21	Yes	Yes	Hebephrenic psychosis	Normal	Improved	Unchanged	5.11/12	31 R. anterior temporal lobectomy
42	M	Neg.	Moderate head injury aged 17	19	45	Yes	Yes	Paranoid psychosis	General circumscribed area of tuberose sclerosis	Much improved	Progressed rapidly	2.4/12	46 R. frontal lobectomy
65	F	Neg.	Left otitis media since childhood	14	24	Yes	Yes	Confusional episodes, chronic paranoid psychosis with depression	Normal	Unchanged	Improved	1.4/12	55 Bimedial leucotomy
14	M	Neg.	Neg.	39	49	No	Yes	Paranoid psychosis	Astrocytoma Grade III	Died 15 months after operation		1.3/12	51 Ventriculography and biopsy of L. frontal region

Salient features of cases submitted to neurosurgery, other than those previously reported.

was negative for psychosis or epilepsy. Two patients had had a possible birth trauma, and one of them had also had a racemose venous angioma of the left hemisphere, which had been treated by deep X-ray therapy at the age of 13. All the patients had both psychomotor and grand mal epilepsy. The psychosis was in all cases schizophreniform, hebephrenic in one case (*23*) and paranoid in the three others.

A follow-up survey in 1960, which is reported by Glithero and Slater in the fourth paper of this series, shows the following findings. The first of these patients (*5*) was then in a mental hospital, but coming home for weekends. Since the operation epileptic fits have continued, but have been less severe, though they cause post-ictal confusion. In addition the patient is liable to aggressive outbursts, and accuses the nurses and the male patients of persecuting her; but she is cheerful, can be friendly, and is active and a good worker. The second patient (*8*) retains her old paranoid ideas, though they are less severe. She is difficult, surly and uncommunicative, and often negativistic to her parents. She cannot stick at any job for more than a few minutes. Her epileptic fits are so slight as to be no problem. The third patient (*41*) is out of hospital and at work in a domestic post. She has lost all her old psychotic symptoms, but is giggly and explosive in speech, and talks in a loud voice in rather a childish fashion. She has had no fits since the operation. In Case *23* the patient in 1960 was living at home unemployed; he was free from fits, but would have occasional minor attacks in which things went blank for a couple of seconds. All psychotic symptoms have passed off, but he is often aggressive in manner, argumentative, quick to take offence. He is very slow in anything he does, and shows the thought disorder of epileptic personality change.

In these four patients, accordingly, it would seem that epilepsy has been the symptom responding best to operation, in one patient relieved entirely and in two others reduced to being no handicap. Schizophrenic and paranoid symptoms are very greatly benefited in two cases, improved in one. On the other hand organic personality changes are evident in all four cases and are a serious handicap.

Of the other three patients, one (*42*) had a right frontal lobectomy because repeated EEGs showed a clearly defined focus at the tip of the right frontal lobe. Another patient (*65*) was leucotomized for chronic depression, which responded temporarily to ECT. The third patient (*14*) had his first grand mal at the age of 39; but investigations then and subsequently were repeatedly negative until he was 51, when a biopsy revealed an astrocytoma from which he died 18 months later.

Summary

In this paper are discussed the physical aspects of a group of 69 epileptics who developed a schizophrenia-like psychosis.

(1) Examination of the past medical history showed that a significant proportion of patients in group A (chronic psychosis coloured by recurrent confusional episodes) and group B (chronic paranoid psychosis) had experienced conditions liable to cause a brain lesion; this was not the case in patients of group C (hebephrenia-like state).

(2) The history of the epilepsy showed that the patients of group C had an earlier age of onset (9·3 years) than those of groups A and B (20·5, 16·3). However, the duration of epilepsy before the onset of psychotic symptoms was nearly the same in all three groups, ranging from 12·8 to 15·6 years, with average for the whole material of 14·1 years. There was a correlation between the ages of onset of epilepsy and psychosis with coefficient value + 0·58. The suggestion arises that duration of epilepsy plays an aetiological role in determining the psychosis.

(3) The quantity of medication, the severity of the epilepsy and the degree of control of the attacks by drugs did not seem to play a part in determining the psychosis.

(4) There was a tendency for the patients in Group A to have all or at least some of their fits during the hours of sleep.

(5) The clinical features of the epilepsy indicated in 45 out of 69 cases an origin in the temporal lobe, and in a further five cases a focal origin in a sensory or motor area.

(6) Neurological examination was usually unhelpful. Altogether there were 12 cases with evidence of pyramidal disturbance.

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(7) Psychiatric examination showed a high incidence of personality changes indicative of organic brain lesions. The degree of personality change was proportional to the duration of the epilepsy, and the association between these two variables was statistically significant.

(8) The electroencephalographic findings substantiated the clinical impression of a high incidence of temporal lobe epilepsy; and there was good agreement between the clinical and EEG diagnoses. However, the EEG using sphenoidal wires and activation revealed ten cases of temporal lobe epilepsy which were not suggested by the history. This figure is considered important, in that the temporal lobe foci so revealed are sometimes amenable to surgery, and on occasion they may indicate a tumour and facilitate its early recognition.

(9) Air encephalography confirmed the impression obtained from the past medical history that many of these patients have an organic brain lesion. The commonest finding, in 37 out of 56 cases, was that of an atrophic process. In only two cases did the AEG indicate a tumour.

(10) Thirteen patients were examined otologically for abnormalities affecting the central connections of the VIIIth nerve. In five of these caloric tests showed vestibular functions to be normal, and in the remaining eight cases the responses suggested that one or other of the temporal lobes was affected. Otological tests would appear to be a valuable method of examination in such cases.

(11) Data obtained by psychometric tests were usually uninformative. In five cases there were findings suggestive of a slight expressive dysphasia or an auditory learning deficit.

(12) Eleven patients were submitted to temporal lobectomies and one patient to a frontal lobectomy. Pathological findings were very various, including neoplasms, a dermoid cyst, circumscribed tuberose sclerosis and atrophic lesions. Some of these patients have been previously reported by Serafetinides and Falconer. As a result of these operations there was usually substantial benefit to the epilepsy, some improvement of the psychosis, but a heavy incidence of organic personality changes.

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