

THE INCIDENCE OF TEMPORAL LOBE EPILEPSY AMONG A HOSPITAL POPULATION OF LONG-STAY FEMALE EPILEPTICS*

By

J. H. MARGERISON

and

D. W. LIDDELL

INTRODUCTION

THERE are some 23,000 long-stay epileptic in-patients in Britain. Most of these patients are in hospitals for the mentally ill or subnormal or in independent colonies. There are, however, only two hospitals within the National Health Service which cater specifically for the long-term treatment of epilepsy, St. Faith's Hospital, Brentwood, for females, and St. David's, Edmonton, for males. This communication is concerned with the incidence of temporal lobe epilepsy among the long-stay female epileptics at St. Faith's.

Belinson (1947) reported that 26 per cent. of a group of epileptic in-patients showed psychomotor type seizure discharges in their EEGs. However, most of these patients had been admitted with the primary diagnosis of mental deficiency and EEG information obtained from single 20-minute standard recordings. Roger and Dongier (1950) analysed the clinical and EEG features in 50 mental hospital epileptics and found EEG evidence of involvement of the temporo-hippocampo-perifalciforme area in 84 per cent. of the 38 cases in which admission was because of outbursts of impulsive and/or aggressive behaviour. Liddell (1953) reported a temporal focus in 50 per cent. of the epileptic patients in a mental hospital. There does not appear to have been any previous survey of the incidence of temporal lobe epilepsy among those long-stay epileptics who are supposed to have been hospitalized primarily on account of their epilepsies.

Jackson's (1899) term "uncinate group of seizures" was based on a clinico-pathological association in that seizures involving "dreamy states" were found to occur in patients in whom autopsy studies subsequently revealed gross lesions of the temporal lobe. He was careful to point out, however, that "the discharge lesions in these cases are made up of some cells, not of the uncinat group alone, but of some cells of different parts of a region of which this gyrus is part—a very vague circumscription, I admit—the uncinat region."

In recent times and with the advent of EEG, circumscriptions have become more rather than less vague, so that comparison between different studies of temporal lobe and/or psychomotor epilepsy is rarely possible. Lennox (1960), in a chapter entitled Psychomotor (Temporal: Psychic) Epilepsy, did not offer any operational definition nor did one emerge from the Second International Colloquium on Temporal Lobe Epilepsy (1957). Bingley (1958) referred to this

* This work was carried out at Runwell Hospital, Wickford, Essex, with monies provided by the Dowager Lady Peel Trust and under the auspices of the Mental Health Research Fund from whom one of us (J.H.M.) held a research fellowship.

problem and himself elected to classify on two criteria, namely, the presence of epileptic seizures and the presence of a temporal EEG focus and/or a verified temporal lobe glioma. As Northfield (1958) pointed out, "there is much evidence that forms of epilepsy which we associate with temporal lobe disorder can arise from lesions outside the temporal lobe, but little evidence that these highly complicated seizures are exclusively disturbances of temporal lobe function." Fulton's (1953) suggestion that the group of seizures in question "might more properly be designated as seizures involving the limbic lobe" is an attractive way out of the dilemma but to date has not proved testable in man.

Estimates of the incidence of temporal lobe epilepsy involve the primary assumption that such a condition exists. If there is reasonable doubt about the specificity of diagnostic criteria then the survey should be carried out in such a way that others can repeat the work and results be compared. For the purposes of the present study concordant EEG and clinical data, separately assessed and without cross contamination between the two sources of evidence, was the operational basis upon which a diagnosis of temporal lobe epilepsy was to have been made.

METHOD

The entire population of 270 females was surveyed in the following way:

(a) *Clinical Information*

1. Existing hospital records of each patient were summarized.
2. Interviews were carried out by one of us (J.H.M.). In most cases they lasted for several hours and involved first obtaining a free history and thereafter asking questions deliberately slanted with a view to completing a questionnaire. The questionnaire had been designed to cover as widely as possible the symptomatology of epilepsy in general and of the clinical concept of temporal lobe epilepsy in particular.
3. A fit report was made in respect of each patient by a nurse familiar with the usual seizure pattern.
4. Neurological examinations were carried out by one of us (D.W.L.) on all patients. No other evidence, clinical and/or EEG, was made available to the examining clinician.

(b) *EEG Information*

EEGs were taken with an eight-channel Edison Swan machine. The electrode positions used were the same as those adopted by Hill at the Maudsley Hospital, London, and have been described by Pampiglione (1956). An important difference from the 10-20 system is the use of an additional sylvian electrode. The position of the electrodes with respect to underlying cerebral cortex was determined on autopsy specimens at the start of the survey (Darby, Lettich and Margerison, 1958).

In nearly all cases a minimum of three EEGs were carried out, a resting record with hyperventilation (3 minutes) and photic stimulation, a quinalbarbitone induced sleep tracing and a sphenoidal EEG under thiopentone. The technique for sphenoidal recordings followed that described by Pampiglione and Kerridge (1956) being based on earlier work by Jones (1951) and by Kerridge (1952). An additional refinement was the use of wire electrodes threaded through the sphenoidal needle and left *in situ* after withdrawal of the needle, following again the routine practice in the EEG Department of the

Maudsley Hospital. Children (patients below the age of 16) and the elderly infirm were in general excluded from the sphenoidal investigation and where the EEG data was incomplete patients were put into a "not enough evidence" category for the purpose of assessment of incidence figures.

EEGs were interpreted by one of us (J.H.M.) without reference to other information and this was achieved by adopting a number coding for the patients on the survey. The criterion used for EEG evidence in favour of temporal lobe epilepsy was the occurrence of spikes or sharp waves in the anterior and/or middle and posterior temporal regions. In order to check the value of spike counting as a procedure an inter-rater reliability correlation study was subsequently carried out by three members of the EEG Department of Runwell Hospital and correlation coefficients of 0.97 or better established between these raters in 66 counts.

In view of the observations of Kennedy and Hill (1958) on the prognostic significance of localized diminution in barbiturate-induced fast rhythms between the sphenoidal and ear electrodes on the side of the focus in cases of temporal lobe epilepsy, this was made an additional factor in EEG analysis and will be considered separately.

(c) *Assessment of the Information*

All the clinical information was collected and summarized. It was then read out in summary form to us and we attempted independently to classify the patients with respect to the presence or absence of temporal lobe epilepsy on clinical grounds. It is not possible to lay down in precise terms the criteria for clinical diagnosis because while most clinicians specializing in the subject are in general agreement as to the reality of the symptom complex there is no such agreement as to what may be included or must be excluded. Furthermore, it is not yet known what differential weighting should be applied to the multiplicity of symptoms which may be encountered. Gastaut's (1953) long list of symptoms of psychomotor epilepsy, "perhaps too inclusive" according to Lennox (1960), was for instance known to both of us. We were also familiar with Penfield and Jasper's (1954) classification. In general, the basis for clinical diagnosis was that described by Hill (1953). "Objective or subjective vertigo", however, when described as an aura was not considered of itself to point towards a diagnosis of temporal lobe epilepsy. If vertigo, however, was preceded, accompanied or followed by some other temporal lobe symptom as, for instance, in combination with an epigastric sensation and/or a feeling tone of fear, then it was considered to point to such a diagnosis.

After the clinical and EEG information had been separately collected and analysed, correlations were sought between the clinical diagnoses of the two clinicians and between EEG and clinical data. Incidence figures based on these assessments were then calculated.

Arrangements were made at the start of the survey in 1956 for autopsy material to be collected and neuropathological evidence is gradually being accumulated by Dr. J. A. N. Corsellis. Reference will be made to the provisional findings and a cross correlation study is to be carried out between clinical, EEG and neuropathological data.

RESULTS

(a) GENERAL

Figure 1 shows the age distribution of the total population of 270. The median value for age is 49 years (52 years if the 24 children are excluded).

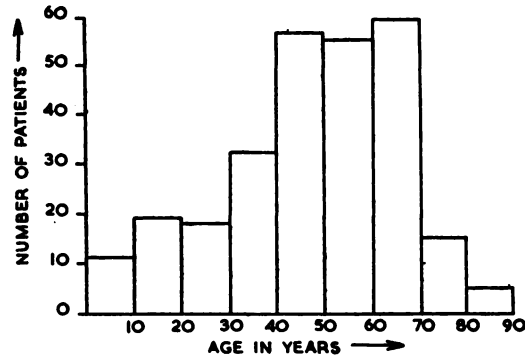


FIG. 1.—St. Faith's. Age distribution (270 patients).

In general, the patients come from large families with an average of 5 or 6 children and are of low social status. They are nearly all single (212 out of 246 adults) and the adults usually enter hospital when about 30 years old. They appear to be admitted at this time in their lives not so much because of worsening fits or deteriorating behaviour as for social reasons, quite often the death of the mother. The average time spent in hospital for the whole population is about 16 years (17·3 years if children are excluded).

(b) PHYSICAL FINDINGS

These are summarized in Table I. About one-third of the patients suffer

TABLE I
St. Faith's
Physical Findings (Gross)

	No. of Patients	Percentage No. of Patients
Paralyses	77	28·5
Other physical illness	26	9·6
No physical findings (gross)	167	61·9
Total	270	
Paralyses:		
Hemiplegia—Slight disability		
Right	22	
Left	16	
Moderate disability		
Right	5	
Left	7	
Severe disability		
Right	6	
Left	2	
Paraplegia	3	
Quadriplegia	3	
Other paralyses	13	

from physical incapacity and this includes 77 with one form of paralysis or another. There is a high incidence of hemiplegia and, although this can to some extent be accounted for by the fact that many of the patients are elderly, there

are also those in whom the hemiplegia either preceded or coincided with the onset of fits during infancy. Lack of documentary evidence precluded more detailed analysis but the assumption is that some of these cases are probably examples of the syndrome of hemiconvulsions, hemiplegia and epilepsy described by Vigouroux (1958) and by Gastaut, Poirier, Payan, Salamon, Toga and Vigouroux (1960).

(c) INFORMATION ON SEIZURES

In 37·2 per cent. of cases one or more relatives was thought to have had at least one seizure (Fig. 2). If only parents and siblings with a history of convulsions are considered then a "positive family history" is found in 26·3 per cent.

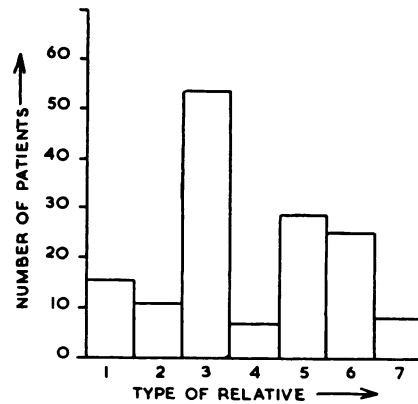


FIG. 2.—St. Faith's. Family history of epilepsy (270 patients).

1. Mother.
2. Father.
3. Siblings.
4. Children of siblings or patients.
5. Maternal relatives.
6. Paternal relatives.
7. ?Maternal or ?paternal relatives.

The total number of patients with a family history of fits is 100, or 37·2 per cent.

Figure 3 shows the age at the time of the first reported seizure. In nearly one-fifth of the patients this was during the first year of life.

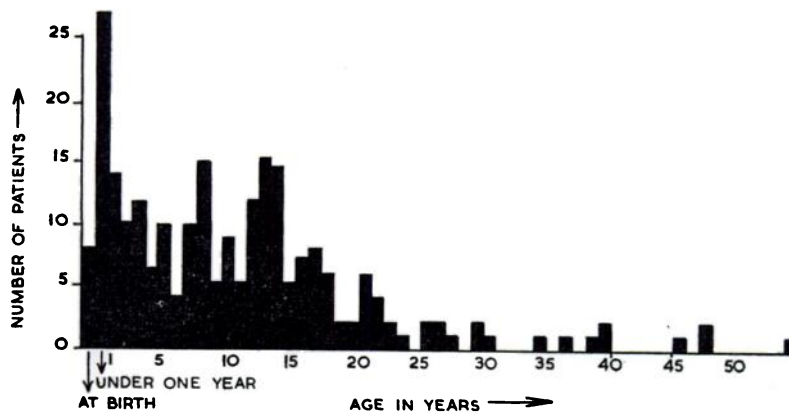


FIG. 3.—St. Faith's. Age of first seizure (270 patients).

Incidence of prodromata was based on statements by the nursing staff in respect of those patients of whom they felt it regularly possible to predict some hours previously that a fit would take place. There are nine cases not considered upon whom no nursing report was available but prodromes were described in 93 out of the remaining 261 (270–9). The different types of prodrome reported are given in Table II. Most commonly patients were noted as “irritable”.

TABLE II
St. Faith's
Prodromata
(Total population sampled=261)

	No. of Patients
Frightened	1
Feels funny	3
Irritable	54
Sad	4
Wants to be alone	2
Quiet	2
Excited	2
Sings	1
Happy and laughs	2
Happy and sings	1
Irritable and sad	12
Irritable and wants to be alone	4
Irritable, wants to be alone and sad	3
Irritable and excited	1
Wants to be alone and sad	1
Total	93

Initial phenomena are listed in Table III. Of the patients with prodromata, 70 described “auras” and 23 denied them. Although Lennox (1960) and others,

TABLE III
St. Faith's
Initial Phenomena
(Total population sampled=270)

	No. of Patients
Sensory (mainly viscerosensory)	94
Psychical	14
Autonomic	1
Motor	10
Sensory and Psychical	22
Sensory and Autonomic	17
Sensory and Motor	20
Psychical and Autonomic	1
Motor and Autonomic	1
Sensory, Motor and Autonomic	1
Sensory, Psychical and Autonomic	1
Total	182

including the authors, use the term aura to embrace both sensory and motor events, the phrase initial phenomena has been used, following Penfield and Jasper (1954) in the absence of general agreement on nomenclature. This also avoids treating localizing motor disturbances as ictal while sensory and other happenings are regarded as somehow pre-ictal, any such distinction being considered unreal. One hundred and eighty-two patients reported initial phenomena, 74 denied any such and there were 14 from whom accounts could not be obtained.

Post-ictal phenomena of various types are shown in Table IV. Sleepiness

TABLE IV
St. Faith's
Post Ictal Phenomena
(Total population sampled=270)

	No. of Patients
Headache	153
Sleepy	171
Sick	16
Automatism	88
Difficulty in speech	36
Motor disability	22
Other phenomena	11
Mood swing	62

and a dull bi-fronto-temporal headache were by far the commonest post-ictal complaints. Most patients denied being incontinent during fits and tongue biting was likewise infrequent. Post-ictal automatism was fairly common as also was mood-swing, the latter usually towards increased cheerfulness. Most of the patients with post-ictal mood swings reported that they did not "feel right" until after they had had a "proper fit". The motor disabilities referred to in the table appeared in most cases to be based on a post-ictal paralysis but hardly any were examined immediately after fits so that a more ambiguous heading has been preferred.

(d) TEMPORAL LOBE EPILEPSY

Classification from Clinical Findings

Of the 270 patients, 24 children were not considered for classification purposes and from the 246 adults, 42 were eliminated on the grounds of inadequate clinical and/or EEG data. In the remaining sample of 204 adult female epileptics, 159 patients were classified as temporal lobe epileptics and 23 as not temporal lobe epileptics by both clinicians. There was disagreement in respect of 22 cases. This measure of agreement between the clinicians is significant at the 0.0005 level ($\chi^2=72.98$ for one degree of freedom).

If clinical criteria of assessment are adopted for the purpose of compiling incidence figures then 77.9 per cent. of the sample of 204 may be described as temporal lobe epileptics and comprise those patients concerning whom both clinicians are in agreement with respect to such a diagnosis.

Classification from EEG Data

If Bingley's (1958) criteria are accepted and classification based upon the EEG findings in epileptics without regard to the clinical details of the fit patterns, then 152 out of the 204 show EEG evidence in favour of temporal lobe epilepsy, i.e. the occurrence of focal spikes or sharp waves in the anterior and/or middle and posterior portions of the temporal lobe. The incidence figure based on this EEG criterion is 74·5 per cent.

Classification Based on Combined Clinical and EEG Information

The fact that incidence figures, whether derived from clinical or from EEG assessments, are so similar when expressed as percentages must not be assumed to support either or both forms of classification. The measure of agreement between the clinical findings and the EEG evidence for or against temporal lobe epilepsy was therefore tested and found to be significant at the 0·05 level ($\chi^2=7\cdot1$ for 2 degrees of freedom). Concordant clinical and EEG data in favour of temporal lobe epilepsy was found in 124 out of 204 cases. The incidence figure based on clinical and EEG agreement is correspondingly reduced to 60·8 per cent.

Finally, those cases in which in addition to focal temporal spikes or sharp waves, a localized reduction in barbiturate induced fast activity was seen between the sphenoidal and mid-temporal electrodes on the same side as the spike focus and in which also a clinical diagnosis of temporal lobe epilepsy was made independently by both clinicians, amount to 33·8 per cent.

All the above incidence figures have been set out in Table V.

TABLE V

*St. Faith's**Incidence of Temporal Lobe Epilepsy According to Different Criteria*

(Expressed as percentages of the "enough evidence group" of 204 adult females)

	Percentage No. of Patients
1. Both clinicians agree on a clinical diagnosis of T.L.E.	77·9
2. EEG data compatible with T.L.E.	74·5
3. Concordant clinical and EEG evidence of T.L.E.	60·8
3a. Patients in group 3, whose EEGs also show localized diminution in barbiturate induced fast activity	33·8

(e) NEUROPATHOLOGICAL DATA

Since the start of the project five years ago autopsy material has become available on about 30 patients from St. Faith's Hospital. Only provisional data are available and, although lesions of the uncus and hippocampal region are found in some 30–40 per cent. of cases, greater numbers will be required before much reliance can be placed on the incidence figure. Ammon's horn sclerosis, when present, is almost always unilateral. Small focal macroscopic lesions as described by Cavanagh (1958) have not been found to date.

DISCUSSION

Corsellis and Meyer (1958) in a neuropathological review of epilepsy and the temporal lobe pointed out that "this is a subject in which considerable activity is taking place and while so much remains unknown the constant supply

of information must inevitably result in a state of flux". The problem of how to supply useful information during this "state of flux" is not easily solved but an attempt has been made to follow the approach laid down by Symonds (1954) in his outstanding contribution on the classification of epilepsies. Symonds stated that "the necessity should be admitted for different categories of data" and suggested that presentation should be such that correlations might be established between the different categories but that the arrangement should also "provide opportunities for recording differences as well as similarities, the differences being equally important".

It was intended to base the incidence figure for temporal lobe epilepsy in this patient population on concordant clinical and EEG data separately collected and independently assessed. This gives a figure of 60·8 per cent. which, it is suggested, would generally be regarded as a conservative estimate. The question arises whether figures of 77·9 per cent. (combined clinical assessment) and/or 74·5 per cent. (EEG assessment) are to be preferred as more comparable with those of other series in which clinical, EEG, or mixed clinical and EEG criteria have been used. More important is the fact that different values have been found within the same study, dependent upon the different categories of evidence admitted.

The data which have been presented show that, while two clinicians may reach a high level of agreement with respect to independent clinical diagnosis and while there is apparently a relationship between clinical and EEG categories, the amount of concordance is indeed limited. There seems a parallel here with attempts to decide upon what clinical basis a diagnosis of syphilis may be placed on a positive Wassermann reaction in the absence of clinical signs or symptoms. In so far as clinical opinion is concerned, it is clear that some cases are more confidently diagnosed than others and the degree of confidence depends on the differential weighting applied to the different clinical findings. Gowers (1901), for instance, elected to accept an olfactory aura as the only basis for the diagnosis of uncinat epilepsy. Such a view would now be generally regarded as much too limited, but most clinicians would rightly be more impressed by the localizing value of an olfactory aura than by that of subjective vertigo or even by that of an epigastric sensation. Some would go further, and Penfield and Jasper (1954) state that "olfactory auras are comparatively rare and, in our experience, have often been associated with tumours of the temporal lobe in an anterior and inferior position".

There appear to have been two opposing tendencies, the one ever widening the notion of temporal lobe epilepsy in the direction of Fulton's physiological concept of limbic lobe epilepsy, and the other trying to break down the multiple symptoms and to allocate anatomical specificity to the several seizure components. Fuster (1953) made a considerable contribution in the latter direction. The fact is, however, that there is no great measure of agreement between different studies. Williams (1956) has for instance reported that fear "when appearing as an emotional experience and as part of the attack" was found in 70 per cent. of 50 patients with *anterior* temporal foci, but Gibbs (1958) has suggested that "attacks of fear, though rare, are more or less peculiar to patients with a *mid-temporal* focus".

The problem of the specificity of diagnostic criteria for temporal lobe epilepsy is not only academic. The practical consequences of such a diagnosis may be radical in so far as the patient is concerned because temporal lobectomy is often carried out as the treatment of choice on patients diagnosed as suffering from temporal lobe epilepsy and in whom anti-convulsant therapy has failed

to control seizures. The fact that excellent therapeutic results have been obtained in a significant proportion of cases, while gratifying, does not of course confirm the diagnosis any more than response to electroconvulsive or other therapy confirms a diagnosis of depression.

The particular value of the Maudsley series lies in the additional evidence which has been obtained from detailed neuropathological study of those portions of temporal lobes resected at operation. Falconer, Hill, Meyer and Wilson (1958) have pointed out that post-operative improvement was striking in the sub-group of tumours and glial hamartomas and marked in the sub-group of disseminated lesions with Ammon's horn sclerosis but that "results in the sub-group of disseminated lesions without Ammon's horn sclerosis were on the whole much poorer". Pre- and post-operative drug regimes were not described but it is not inconceivable that if anti-convulsant medication is maintained more or less constant any epileptic may benefit to some degree from the removal of a substantial portion of cerebral tissue. An alternative way of interpreting these results is to regard the group of cases, and they amounted to 15 out of 50, in which neither focal macroscopic lesions nor Ammon's horn sclerosis were found, as cases which should not be classified as suffering from temporal lobe epilepsy. Specificity of diagnostic criteria would, by these tokens, be dependent on the correlation between clinical, EEG and neuropathological evidence. This admittedly involves a return to the Jacksonian concept of a clinico-pathological association to which should now be added at least the third dimension of EEG findings. In our present state of ignorance, and given that the therapeutic implications of the diagnostic label "temporal lobe epilepsy" may involve a neurosurgical operation, it seems not unreasonable to test both EEG and clinical criteria against the neuropathological findings. This involves accepting the limitations of present neuropathological techniques but avoids the more dangerous assumption that failure to find localized pathology is due to these limitations. In course of time the biochemist may come to our aid.

Kennedy and Hill (1958) established a predictive value for the observation of unilateral localized reduction in barbiturate-induced (intravenous thiopentone) fast activity from sphenoidal to ear electrodes in patients from the Maudsley series. Specimens of temporal lobe tissue from those patients in whose pre-operative EEG records such an observation was made were likely to show lesions in the uncus and Ammon's horn on the same side as that upon which reduction of fast activity had been noted. The fact is, however, that barbiturate-induced fast activity can show local diminution in amplitude following a focal seizure and probably also in association with any considerable amount of focal spike activity even in the absence of a recent clinical seizure. In serial records from one patient it is possible to observe less barbiturate-induced fast activity on one side than the other in the temporal regions on one occasion but not on another. It is quite clear that a great deal still requires to be known about the significance and standards of measurement of localized reduction in barbiturate-induced fast activity. However, in view of Kennedy and Hill's (1958) findings, and notwithstanding the limitations which have been mentioned, it seems reasonable to treat the observation of reduced fast activity as an additional EEG criterion and indeed as an essential part of EEG evidence in favour of temporal lobe epilepsy. If this is done, then the incidence figure for temporal lobe epilepsy in the present series is further reduced to 33·8 per cent. It is of interest that this figure is of the same order as that for the observation of temporal lobe lesions in those cases which have come to autopsy.

The progressive narrowing of diagnostic criteria has been accompanied by a parallel decrease in incidence figures. Which figures are to be accepted is clearly a matter of opinion, but when the criteria are strictest the findings are more likely to be in keeping with the neuropathological evidence. It is not suggested that wider concepts are erroneous, and it is appreciated that they may ultimately prove more fruitful. On the other hand it must be recognized that the clinical diagnosis of temporal lobe epilepsy, as commonly made, represents a rather vague formulation. Patients with this form of epilepsy, however defined, may show a rhythmicity of seizure incidence, a greater tendency to have a "positive" family history than is seen in the general population and a liability for fits to be made better or worse by emotional and metabolic factors which must call seriously into question the view that these seizures necessarily start in the temporal lobe, or within the limbic system.

In conclusion it must be emphasized that among this population of long-stay female epileptics a fairly large group are considered likely to have unilateral temporal lobe lesions and might therefore be expected to benefit from temporal lobectomy. A controlled study on the effects of the operation is required in order to establish this. A long-term hospital population might well prove most suitable for such a study.

SUMMARY

The results of a survey into the incidence of temporal lobe epilepsy among a hospital population of long-stay female epileptics are presented. The incidence figures are shown to be different according to the categories of clinical and/or EEG evidence admitted. It is suggested that there is a lack of specificity of diagnostic criteria and reasons are given for proposing that the term temporal lobe epilepsy should at present be confined to describe those cases in which there is reason to suspect an association between clinical, EEG and neuropathological findings. It is further argued that if EEG classification is made stricter by admission of an additional category of data the possibility of correlation between EEG and neuropathological evidence may be increased as suggested by Kennedy and Hill (1958). Figures for the incidence of lesions of the temporal lobe, based on provisional autopsy findings, are offered in support of this view.

ACKNOWLEDGMENTS

Grateful thanks are due to the patients and to the medical and nursing staff of St. Faith's Hospital, Brentwood. Dr. J. A. N. Corsellis has given generously of both his time and material, and helpful advice has been received from many other colleagues. We are indebted to Dr. Ström-Olsen, Physician-Superintendent of Runwell Hospital, for permission to carry out this work and for his encouragement. Finally, we are very grateful to Mrs. C. E. Darby without whose drive, enthusiasm and persistent hard work, as EEG recordist and in many other capacities, the survey could not have been done.

REFERENCES

- BALDWIN, M., and BAILEY, P. (eds.), *Temporal Lobe Epilepsy*, 1958. Proceedings of Second International Colloquium. Springfield, Illinois: C. Thomas.
- BELINSON, L., *Amer. J. Ment. Defic.*, 1947, **52**, 9.
- BINGLEY, T., "Mental Symptoms in Temporal Lobe Epilepsy and Temporal Lobe Gliomas", *Acta Psychiat. scand. Suppl.*, 1958, **120**, v. 33.
- CAVANAGH, J. B., *Brain*, 1958, **81**, 389.
- CORSELLIS, J. A. N., and MEYER, A., in: *Recent Progress in Psychiatry*, 1958, **3**, 160.
- DARBY, C. E., LETTICH, E., and MARGERISON, J. H., *Proc. E.P.T.A.*, 1958, **7**, 3.
- FALCONER, M. A., HILL, D., MEYER, A., and WILSON, J. L., in: *Temporal Lobe Epilepsy* (eds. Baldwin, M., and Bailey, P.), 1958. Springfield, Illinois: C. Thomas.
- FULTON, J. F., *Epilepsia*, 1953, **2**, 77.

920 INCIDENCE OF TEMPORAL LOBE EPILEPSY IN FEMALE EPILEPTICS

- FUSTER, B., *ibid.*, 1953, 2, 77.
GASTAUT, H., *ibid.*, 1953, 2, 59.
Idem, POIRIER, F., PAYAN, H., SALAMON, G., TOGA, M., and VIGOUROUX, M., *Epilepsia*, 1960, 1, 418.
GIBBS, F. A., in: *Temporal Lobe Epilepsy* (eds. Baldwin, M., and Bailey, P.), 1958. Springfield, Illinois: C. Thomas.
GOWERS, W. R., *Epilepsy and Other Chronic Convulsive Diseases*, 1901. London: J. & A. Churchill.
HILL, D., *Proc. roy. Soc. Med.*, 1953, 46, 965.
JACKSON, H. J., and STEWART, P., *Brain*, 1899, 22, 534.
JONES, D. P., *Electroenceph. clin. Neurophysiol.*, 1951, 3, 100.
KENNEDY, W. A., and HILL, D., *J. Neurol. Neurosurg. Psychiat.*, 1958, 21, 24.
KERRIDGE, J. C., *Electroenceph. clin. Neurophysiol.*, 1952, 4, 254.
LENNOX, W. G., *Epilepsy and Related Disorders*, 1960. Vol. 1. London: J. & A. Churchill.
LIDDELL, D. W., *J. Ment. Sci.*, 1953, 99, 732.
NORTHFIELD, D. W. C., *Proc. roy. Soc. Med.*, 1958, 51, 607.
PAMPIGLIONE, G., *Proc. E.P.T.A.*, 1956, 7, 20.
Idem and KERRIDGE, J., *J. Neurol. Neurosurg. Psychiat.*, 1956, 19, 117.
PENFIELD, W., and JASPER, H., *Epilepsy and the Functional Anatomy of the Human Brain*, 1954, London: J. & A. Churchill.
ROGER, A., and DONGIER, M., *Rev. neurol.*, 1950, 83, 593.
SYMONDS, C., *Arch. Neurol. Psychiat.*, 1954, 72, 631.
VIGOUROUX, M., *Rev. neurol.*, 1958, 99, 39.
WILLIAMS, D., *Brain*, 1956, 79, 29.

J. H. MARGERISON, M.B., Ch.B., *Lecturer, Institute of Psychiatry, Maudsley Hospital, London*

D. W. LIDDELL, M.B., M.R.C.P., D.P.M., *Department of Psychological Medicine, King's College Hospital, London*