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Epileptic Psychosis:
An Evaluation of PSE Profiles

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Summary: Data are presented on 24 patients with epilepsy and psychosis whose clinical presentation was rated using the Present State Examination (PSE). Seventeen had complex partial seizures and a diagnosis of temporal lobe epilepsy, seven had generalised epilepsy. An association between a CATEGO category of nuclear schizophrenia (NS) and a lesion of the left side was noted. No clear link between depressive symptoms and a right-sided focus was discovered. Affective disorders were noted in both groups of epileptic patients, although paranoid psychoses were commoner in the temporal lobe group. There was also a tendency for the latter to have more delusions of persecution, ideas of reference, and special features of depression. The group rated as NS appear less likely to show evidence of intellectual deterioration than the other psychotic patients; in addition, the interval between the onset of their epilepsy and the onset of their psychosis is shorter. Radiological assessment by CAT reveals few differences between groups, but the psychotic samples do show higher than expected values on a number of variables, in particular the bilateral septum-caudate distance and the size of the third and fourth ventricle.

Slater & Beard (1963) reported 69 patients with epilepsy and a schizophreniform psychosis and concluded, on statistical and clinical grounds, that the two were associated causally rather than by chance; renewed interest in the nature of such a linkage has emerged since then. Flor-Henry (1969), for example, suggested that in temporal lobe epilepsy, the laterality of the lesion was important in determining the type of psychosis which developed, but Bruens (1971) suggested that few or none of such cases were phenomenologically akin to process schizophrenia, and both Kristensen & Sindrup (1978) and Jensen & Larsen (1979) failed to confirm any link between laterality and psychosis.

The pathogenesis of this association has also been the subject of speculation. Pond (1957) suggested that the abnormal experiences associated with temporal lobe epilepsy gradually became integrated into a person's psychic life, thus resulting in psychosis, while Bruens proposed that the organic and psychodynamic factors 'potentiate' each other. Slater & Beard suggested that the psychoses were non-specific organic psychoses, the psychosis being a late product of a disorder of function of which epilepsy is an earlier manifestation. Flor-Henry, noting an inverse relationship of the presence of psychosis with seizure frequency, thought that epileptic psychoses were not organic but were truly epileptic.

In an earlier paper (Perez & Trimble, 1980) we described the mental state of a group of patients with epilepsy and psychosis who were prospectively referred to us and evaluated using the Present State Examination (PSE) (Wing *et al* 1974); a link was demonstrated between temporal lobe epilepsy and symptoms of schizophrenia. In this communication, we present an analysis of the symptom and syndrome profiles of these patients, and further examine the pathogenesis of the schizophrenia-like psychoses of epilepsy noting the evidence, in our series, for the presence of organicity and its relationship to the psychopathology.

Materials and Method

Twenty four patients with epilepsy and psychosis, consecutively referred to the Department of Neuropsychiatry at the National Hospital, Queen Square, were examined. All had been psychotic for at least one month, were psychotic at the time of their assessment, and had psychoses occurring in a setting of clear consciousness. They received a neurological examination and a number of investigations, including routine haematology, assessment of serum anticonvulsant levels, psychometry, electroencephalography, computed axial tomography (CAT), and CSF analysis. Their mental state was rated using the PSE. Eleven non-epileptic psychotic patients, all of whom had been diagnosed as schizophrenic by two psychiatrists, were used as controls.

The epileptic patients had, on average, 3.5 EEGs recorded prior to their admission and at least one during the study. Sphenoidal recordings were undertaken in six patients with complex partial seizures, and one patient had prolonged video-telemetry. The diagnosis of epilepsy was initially made on clinical grounds, but

confirmed by assessment of the EEG data by one of us (NM), who was blind to the psychiatric status of the patients. The latter were based on the CATEGO sub-classes provided from the PSE, in which the classification of nuclear schizophrenia (NS), is based on Schneider's first-rank symptoms. The syndrome profiles of patients with and without epilepsy have been compared, as have those with different types of epilepsy and, with respect to those with temporal lobe abnormalities, to the side of the lesion.

During the investigation, information was gathered from the clinical histories and where possible, an independent relative, of the date of origin of the epilepsy and the length of the psychiatric illness. All patients received psychometric testing, and values for the performance scale,the verbal scale and the full scale of the WAIS were recorded. In 16 patients, there were records of previous psychometry with which their current status could be compared.

CT scans were performed on an EMI 5005, using 13mm cuts at normal resolution; they were imaged at window width 40 and centred at a level of 18 EMI units. Pictures were obtained using an EMI multi-format imager, the magnification factor being 4.54. Radiological assessments included visual inspection of the scan, as well as linear measurements, performed with a transparent ruler to the nearest 0.5 mm. Subjective assessments of the sub-arachnoid spaces were rated on a scale of 0 to 3, inter-observer reliability having previously been established by three radiologists. The width of the anterior horns, septum-caudate distance, cella media distance, and the third ventricle size were performed as described by Gyldensted & Kosteljanetz (1975) and Gyldensted (1977). The Evans Ratio was measured according to the original descriptions (Evans 1942) and assessment of posterior fossa structures, including the fourth ventricle and cistern-brainstem ratios, were performed according to the method of Koller *et al* (1981). Evaluation of the CT scans was carried out blind to the psychiatric diagnosis.

The Fisher Exact Probability Test or 'Students' t-test were used in the statistical analysis.

Results

PSE profiles

Of the 24 patients with epilepsy and psychosis, 17 had complex partial seizures and history of an EEG abnormality compatible with a diagnosis of temporal lobe epilepsy. Specifically, spikes or a slow-wave focus in one or both temporal regions were present. In seven, the seizure type was generalised and a diagnosis of primary or secondary generalised epilepsy made. In the temporal lobe group, ten were males and seven females, with a mean age of 37 years (range 23–58). In the generalised group, there were five males and two females, with a mean age of 37 (range 19–50). The PSE sub-class diagnoses showed that 11 of the temporal lobe group were categorised as NS and six had other forms of psychosis; none of the generalised group had a diagnosis of NS.

The laterality of the focus, as noted from the EEG recordings in the temporal lobe group, was as follows: in 11 patients classified by the CATEGO programme as NS, seven had exclusively a left-sided EEG focus, two had a right-sided focus (one was left-handed), one had bilateral foci with a left-sided predominance (who had exclusively left-sided lesions on previous EEGs) and one had bilaterally independent foci.

In six temporal lobe patients with CATEGO sub-classes other than nuclear schizophrenia, one had a left temporal lobe focus, two had unilateral right foci, two had bilateral foci, one with generalised involvement, and one had no defined focus on the EEG. These data demonstrate a significant link ($P < 0.05$) between a left-sided focus and a classification of NS. There was no clear relationship between a right-sided focus and type of psychosis.

Figure 1 shows a comparison of the PSE syndrome profile of 11 patients with temporal lobe epilepsy (TLE) and a classification of nuclear schizophrenia and nine non-epileptic schizophrenic controls who were also rated as having NS. As previously reported by Perez & Trimble, both were comparable for sex and age distribution. Three of the epileptic and none of the non-epileptic group were married: four of the epileptic and five of the non-epileptic group were employed at the time of study. A positive family history for psychiatric illness was noted in three patients from each group. The two groups were compatible, not only in terms of NS symptoms, but also shared similarities of other symptoms, rendering both profiles almost identical. The two exceptions were delusions of grandeur (GR) and visual hallucinations (VH), both significantly ($P < 0.05$) more common in the non-epileptic group.

In comparison of the profiles of patients with left and right-sided TLE, there was, in addition to the NS association to the former, a non-significant trend towards more ideas of reference (IR) in the left-sided group and a higher scoring of delusions of persecution (PE) and sexual and fantastic delusions (SF).

Thirteen of the total sample of patients with epilepsy and psychosis received a diagnosis other than nuclear schizophrenia; six came from the temporal lobe group. Figure 2 summarises these data. Affective disorders were noted in both groups. All the temporal lobe patients had either a primary or a combined disturbance of affect and in four out of the six, the combination was with a paranoid psychosis. Generalised epilepsy was less clearly related to a disorder of affect and paranoid psychoses in this group were rare (one out of seven). Figure 2 gives the PSE syndrome profiles of these two sub-groups of patients with non-nuclear schizophrenia. It can be seen that the temporal lobe group have more delusions of persecution (PE), delusions of reference (RE) and special features of depression (ED) than the generalised group, the latter showing more tension, sexual fantasies, overactivity, depersonalisation and hypomania.

The EEG findings during the study, in which records were taken when the patients were psychotic, showed 13 to have inter-ictal spike activity, nine having diffuse abnormalities and only two being reported as normal. The epileptic changes were present in eight of the 11 patients with NS and in four of six with TLE and non-nuclear schizophrenia profiles.

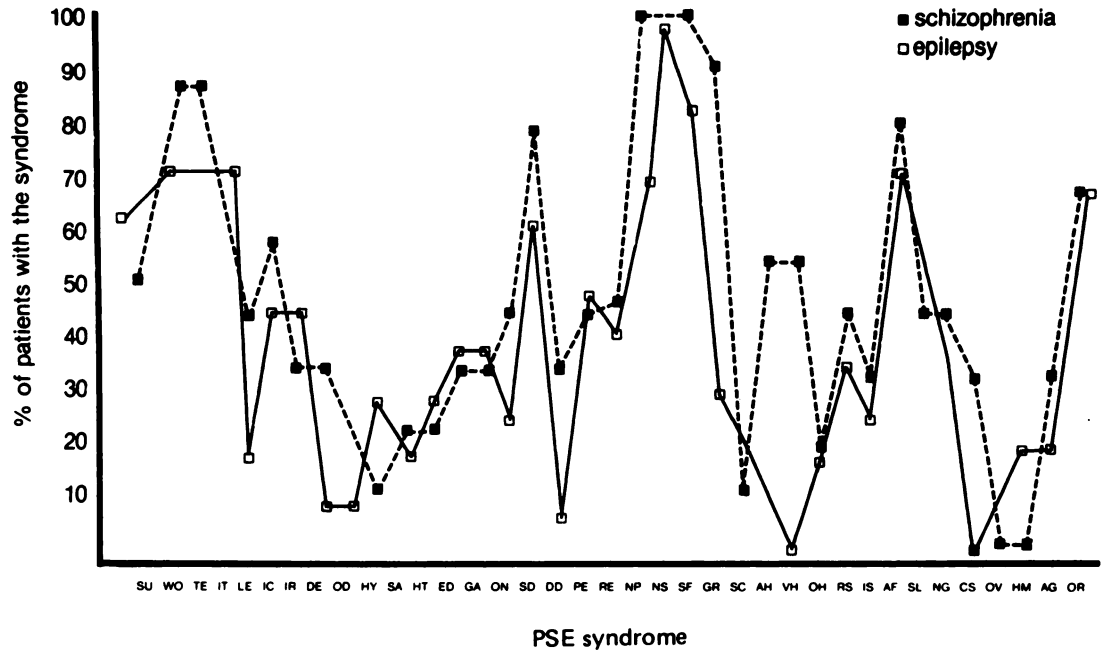

FIG. 1 PSE Syndrome profiles comparing patients with a schizophreniform psychosis of epilepsy to process schizophrenia.

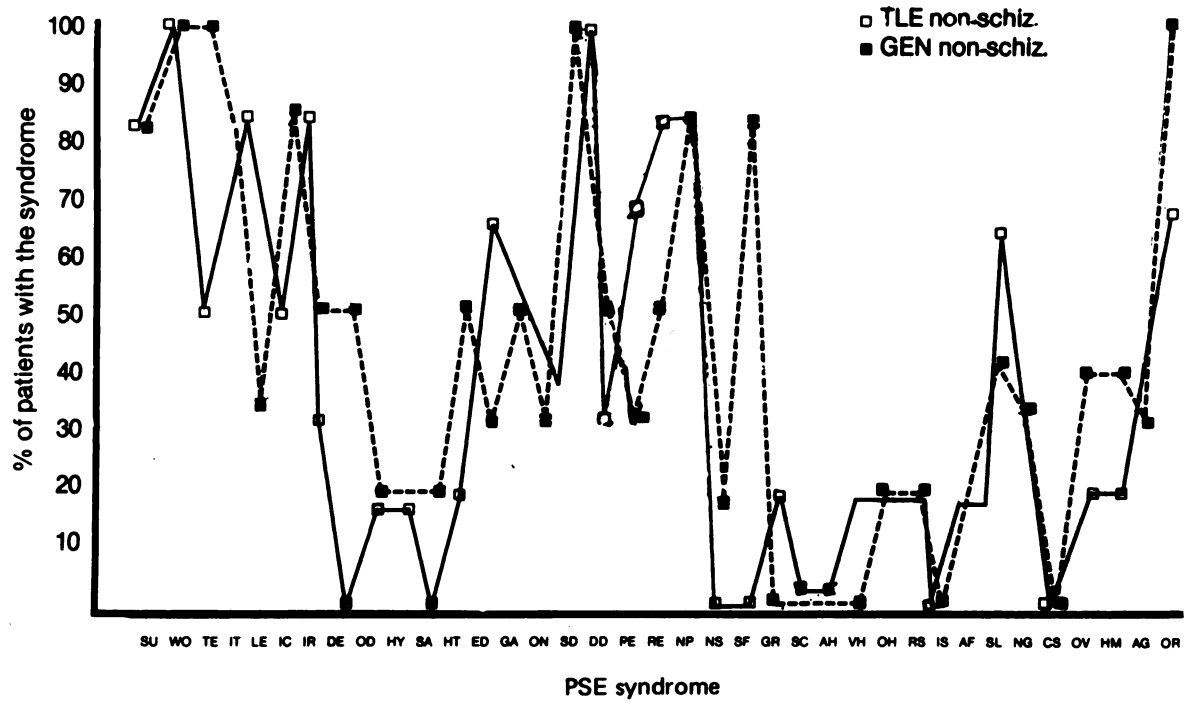

FIG. 2 PSE profile comparison between non-schizophrenic TLE (6) and GEN (7) epileptic non-schizophrenic patients

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Radiological and other data

Using the PSE CATEGO sub-class data on the epileptic sample, the group NS (n = 11) was compared with those patients with temporal lobe (n = 6) and those with generalised epilepsy (n = 7), with other forms of psychosis (the combined group). In addition, data from the 11 non-epileptic patients diagnosed clinically as having schizophrenia were available for comparison.

Values for intelligence testing (IQ) and data with regard to duration of epilepsy, duration of psychosis, and the interval between the two are shown for the epileptic samples in Tables I and II respectively. These show that the mean IQ of the NS group falls within the normal range and that significant differences exist between this group and the other two groups combined, mainly reflected in the lower performance scale of the latter. Generalised impairments were noted on testing in two of the NS group, four of the temporal lobe group and two of the generalised epilepsy group. A significantly shorter interval was noted between the onset of the epilepsy and the onset of the psychosis for the NS group, when compared with the combined temporal lobe and generalised epilepsy group.

CT scans were available for examination in 20 of the epileptic sample; visual inspection revealed gross focal abnormalities in seven, and four were rated as showing mild atrophy. In the NS sample, focal lesions were seen on three scans: one had bilateral low density temporal lobe lesions with greater changes on the left, one had similar lesions with greater changes on the right (the patient had previously undergone a right temporal lobectomy) and one had a left-sided lesion compatible with an earlier resection of an angioma. In the temporal lobe and generalised epilepsy groups, four focal abnormalities were noted: one left-sided lesion occurred in the parietal cortex, two patients had bilateral abnormalities and another a right frontal lesion. In the non-epileptic schizophrenia sample, no focal lesions were noted, but two were reported as having mild atrophy.

Data from the measurement of the scans are shown in Table III. Patients from the NS group are compared to those in the combined temporal lobe and generalised epilepsy group and, for comparison, the non-epileptic schizophrenia group. Finally, normal values provided from the literature (Gyldensted & Kosteljanetz, 1975/77) and Koller *et al* (1981) are given. Statistical evaluation against these normal values has not been attempted.

Table III shows only one significant difference, i.e. the small combined cella media size in the combined group of temporal lobe and generalised epileptics. From the Table, however, it should be noted that the NS sample more frequently has enlargement of the cerebellar sulci and that the psychotic groups generally show larger values for all measurements except the combined cella media size and the Evans Ratio. From the normative data provided, the most interesting discrepancies are the septum-caudate distance, the third ventricle size, and the fourth ventricle ratios.

No difference between the right and left sides of the brain were noted in the patients with epilepsy and a CATEGO diagnosis of nuclear schizophrenia for the width of the anterior horns, the septum-caudate distance, and the size of the cerebral sulci or the cella media distance.

TABLE I

Intellectual Assessment of Epileptic patients with nuclear schizophrenia (NS) and other forms of psychoses, (standard deviations in brackets)

IQ	*Performance*	*Verbal*	*Full Scale*	*% Deteriorated*
Category				
TLE/NS	100 (14)*	98 (13)	100 (13)	18
TLE/other	85 (4)	89 (29)	87 (25)	66
Gen/other	80 (14)	88 (15)	85 (10)	28

*P <0.02 TLE/NS v others

TABLE II

Comparison between length of epilepsy, length of psychosis and the interval in patients with TLE and a category of nuclear schizophrenia means compared with others (standard deviation)

PSE/Epilepsy	*Age*	*Length Epilepsy*	*Length Psychosis*	*Interval*
	(Years)	(Years)	(Years)	(Years)
TLE/nuclear schizophrenia n = 11	37.2	26.2 (11.0)	10.0 (8.2)*	16.2 (8.0)*
TLE/Non-nuclear schizophrenia n = 6	37.3	28.2 (11.0)	2.1 (3.0)	26.7 (9.0)
Gen/Non-nuclear schizophrenia n = 7	36.7	29.9 (10.0)	4.7 (5.0)	25.1 (12.0)

*P <0.05 TLE/nuclear schizophrenia and rest

With regard to anticonvulsant serum levels, nine of the NS group were on either one or two anticonvulsant drugs and only one patient was receiving four drugs. The mean serum levels were phenytoin 48 μmol/litre (range 14–88), carbamazepine 33 μmol/litre (range 23–42) and phenobarbitone 109 μmol/litre (range 74–155). The temporal lobe non-nuclear schizophrenia group (n = 6), contained five on two drugs. Mean serum levels were phenytoin 67 μmol/litre (range 18–118) and carbamazepine 22 (13–34) μmol/litre. The generalised epileptic group had two patients on one drug alone, three patients were on two drugs and one was on three drugs. Mean levels were phenytoin 58 μmol/litre (range 56–61) and phenobarbitone 92 μmol/litre (range 43–159). In the whole series, only one patient, in the temporal lobe non-schizophrenia group, had a toxic level (phenytoin 118 μmol/litre) and had associated clinical signs of toxicity.

Discussion

This study, the first prospective study of psychosis in patients with epilepsy, has attempted accurately to define the psychopathology, and to associate it with both the type of epilepsy and the site of the epileptic focus. With regard to the first, we confirm a link between the presentation of nuclear schizo-

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TABLE III
Radiological data (± S.D.)

Diagnosis:	*(a) Epileptic/nuclear schizophrenia*	*(b) Epileptic non-nuclear schizophrenia*	*(c) Non-epileptic schizophrenia*	*(d) Normal values (5 to 95% in brackets)*
	n = 10	*n* = 10	*n* = 10	
Enlarged cerebral sulci (left)	1	1	1	
Enlarged cerebral sulci (right)	1	1	1	
Enlarged cerebellar sulci (left)	3	1	0	
Enlarged cerebellar sulci (right)	3	1	0	
Enlarged Cerebellar Vermis	4	4	2	
Maximum distance between anterior horns, mm	34.1 (5.9)	35.9 (4.9)	33.8 (4.6)	33.0 (26.4 – 39.6)
Bilateral septum caudate distance, mm	17.7 (3.7)	15.5 (7.8)	16.6 (3.1)	14.9 (10.0 – 24.8)
Combined cella media size, mm	28.1 (5.6)	20.9 (8.7)*	27.5 (3.5)	29.7 (19.8 – 36.3)
Evans ratio	0.26 (0.05)	0.27 (0.03)	0.26 (0.04)	0.26 (0.20 – 0.30)
Third ventricle size, mm	4.08 (1.44)	5.08 (2.75)	3.7 (1.17)	3.3 (1.7 ± 6.6)
Cistern brainstem ratio	0.13 (0.02)	0.17 (0.09)	0.13 (0.05)	0.11 (± 0.01)
Fourth ventricle ratio	0.12 (0.03)	0.11 (0.02)	0.10 (0.01)	0.07 (± 0.003)

*P <0.05 (a v b)

phrenia as defined by the PSE, which predominantly detects Schneiderian first-rank symptoms, and temporal lobe epilepsy. The link between psychosis and temporal lobe epilepsy was first suggested by Gibbs *et al* (1951) and further emphasised by both Hill (1953) and Pond (1957). In Slater's series, 52 out of 69 had a temporal lobe focus, and this was seen in 65% of those with a chronic paranoid state. However, none of the above authors were able to quantify their phenomenology, as has been carried out in this series.

Patients without a temporal lobe focus and classified as generalised epilepsy are seen in this study to have a variety of psychoses and when the non-nuclear schizophrenia group are considered, no clear relationship was found between the type of psychosis and the type of epilepsy. Our data confirm the link, first suggested by Flor-Henry (1969), that patients with a schizophreniform psychosis and epilepsy are more likely to have a left-sided focus, although in this study we were unable to find any link between a right-sided abnormality and any particular presentation. We are aware that surface EEG recordings are not sufficient to rule out contralateral temporal lobe foci,but an attempt was made in this study to classify patients using the EEG, instead of intracerebral recordings, prolonged ambulatory monitoring, or positron emission tomography. By having had our recordings rated blind with regard to the clinical diagnosis and using sphenoidal electrodes when routine scalp recordings were inconclusive, we consider that we have minimised this source of error. It is also important to examine how our data confirm or refute other information drawn from the literature on this point. Table IV gives a list of those authors who have examined a link between a schizophreni-

TABLE IV
Left and right-sided temporal lesions in patients with a schizophreniform psychosis of epilepsy

Author	*N*	*Left*	*Right*	*Bilateral*
Slater and Beard (1963)	48	16	12	20
Flor-Henry (1969)	21	9	2	10
Gregoriades *et al* (1971)	43	43	0	0
Taylor (1975)	13	9	4	0
Hara *et al* (1980)	10	6	4	0
Sherwin (1981)	6	5	1	0
Sherwin (1982)	7	5	2	0
Toone *et al* (1982)	12	4	0	8
Ounsted & Lindsay (1982)	9	7	0	2
Trimble & Perez (1982)	11	8	2	1
Total	180	112	27	41

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form psychosis and the laterality of an epileptic focus in papers from which figures can reliably be drawn. It shows a clear predominance of left-sided lesions.

Of particular interest to this study is the predominance of paranoid syndromes in association with temporal lobe epilepsy, again mainly linking with left-sided abnormalities. Thus, in the epilepsy literature, particularly where a link between personality change or psychosis and epilepsy has been examined, a hint emerges of an increased association between paranoid disorders and temporal lobe abnormalities (for review see Trimble, 1983). Although not all studies have been conclusive, many show raised paranoia scores on, for example, MMPI profiles, which even led to the suggestion that the temporal lobes were somehow involved in the genesis of paranoia (MacLean, 1969). Our data confirm this view, raising important questions regarding the underlying neuropathology of paranoid symptoms and the involvement of the temporal lobes in their development. With regard to the laterality effect, several authors (e.g. Jensen & Larsen, 1979, Kristensen & Sindrup, 1978), have not reported such a finding. However, they have also failed to specify the precise nature of the psychosis; in the case of Kristensen & Sindrup, they only used paranoid ideas and/or hallucinations in a state of clear consciousness as inclusion criteria for their study. It is of interest that the majority of authors who have commented on the laterality effect, including ourselves, have used Schneiderian criteria as a basis for the categorisation of schizophrenia. These findings are in agreement with the extensive literature on the relationship of process schizophrenia to left-sided brain dysfunction which has been detected by a variety of techniques (*inter alia* Gruzelier, 1981), and the literature from patients sustaining head injury, in which again those with left-sided trauma are more likely to develop a psychosis (Hillbom, 1960).

One other group (Toone *et al*, 1982) has recently used the PSE to investigate the profile of epileptic psychosis. Although not reporting on any laterality differences with regard to EEG data, they have also commented on the similarity of profiles between epileptic psychosis and process schizophrenia and, with few exceptions, have provided data similar to those presented here in our prospective study. Using neuroradiological evidence for the assessment of the site of abnormality, they note a trend towards more left-sided lesions with a presentation of a schizophreniform psychosis compatible with our data.

We have tried here to evaluate further the relevance of organic factors to the pathogenesis of epileptic psychoses, emphasising mainly information from case histories, standard intelligence tests, and CT scans. By using the CATEGO diagnostic programme and the PSE we have attempted to minimise a major source of error in investigations in this field, i.e. the use of crude clinical evaluation in diagnosis. We have separated a group of patients who have epilepsy and a CATEGO classification of nuclear schizophrenia from the other psychiatric categories, and have shown a clear link with this presentation and temporal lobe epilepsy. We also show that this group appears to be separated from other patients in the overall sample of epileptic psychosis by showing near normal IQ values with little evidence of intellectual deterioration. The patients with NS and epilepsy are more likely to be married than those with process schizophrenia, perhaps emphasising an important clinical difference between the schizophreniform psychoses of epilepsy and schizophrenia, i.e. the well preserved affect, discussed fully by others (*inter alia* Slater & Beard). In our experience, most patients in this category do not become institutionalised and live reasonably satisfactory lives in the community. It is suggested that this is one reason why surveys of patients in psychiatric hospitals very often fail to show a combined diagnosis of schizophrenia and epilepsy (*inter alia* Stevens, 1980). The radiological data must be viewed in the light of the extensive reports by Slater & Beard and the more recent paper of Toone *et al* (1982). Slater & Beard noted a high frequency of atrophic processes on pneumoencephalograms, affecting especially the central white matter. Toone *et al*, in their comparison of epileptic patients with and without psychosis (rated with the Syndrome Check List derived from the PSE and used in retrospective case history evaluation), noted 44% of the former to have abnormalities (mainly atrophy) with a tendency for a diagnosis of schizophrenia to be associated with left-sided lesions. We have shown that 55% of our total epileptic sample with epileptic psychosis have structural lesions, one-third of these being atrophy. Within the nuclear schizophrenia group, of those three with abnormalities, all had focal lesions on the left side, but in two cases the abnormalities were bilateral. For the others, there was one left-sided non-temporal lesion, two bilateral, and one right-sided abnormality. These figures are too small to allow comment on the relationship of the structural lesions to the clinical presentation of the psychosis, but the presence of abnormalities is of the same order as reported by Flor-Henry and Toone *et al*.

Quantitative assessment of our scans with com-

parisons between groups gives little further information. It is unclear why the combined group of temporal lobe generalised epileptics should show smaller cella media size than the other groups and on this index, the NS group shows similar values to those reported from control studies. It is recognised that the values reported in the latter may not be directly comparable to our patients, but it is notoriously difficult to obtain reliable quantitative values for CT scan measurements in normals, and the samples obtained by Gyldensted & Kosteljanetz (1975/1977) and by Koller *et al* (1981) are widely used for comparative evaluations. The mean age of our samples (NS = 37 years) is compatible with the ages of the normals reported by these authors. In comparison with expected values it is therefore of interest that the bilateral septum-caudate distance is greater in all the psychotic samples and greatest in the nuclear schizophrenia group. In a small sample (n = 5) of epileptic non-psychotic patients, also evaluated radiologically by us, this measurement was 13.6 mm (s.d. 3.8)—again smaller than the psychotic groups. These data therefore suggest an involvement of basal ganglia structures in these psychotic states, especially in the group presenting with symptoms of nuclear schizophrenia. Likewise, the higher third and fourth ventricle size points to periventricular abnormalities, which have been shown to occur in association with both schizophrenia and also epileptic psychosis (Stevens, 1982). The recent reporting of raised third ventricle size and increased caudate density (Dewan *et al*, 1983) and increased bicaudate ratios (Woods & Wolf 1983) in patients with schizophrenia provide clear links between our data and investigations in non-epileptic psychotic patients. We did not detect any significant left/right differences in our quantitative radiological assessments. The data presented by Toone *et al*, in which a trend was seen between left-sided abnormalities and a diagnosis of schizophrenia are quite compatible with our negative findings; they did not use quantitative assessments, and were referring to gross structural changes. As noted above, the structural lesions on our scans were too few in number to provide informative data.

Finally, it should be noted that patients in the NS group have a significantly shorter interval between the age of onset of epilepsy and that of psychosis, when compared with the other groups. This issue was first discussed by Slater & Beard, who suggested that the duration of the epilepsy was important for the development of the psychosis. Several others have confirmed that the psychosis usually suceeds the epilepsy by an interval of from 12 to 22 years (Toone, 1982). We feel that the shorter onset for the NS group not only highlights differences between those patients presenting with a schizophreniform picture, resembling nuclear schizophrenia, and other forms of psychosis, but may have pathogenic significance. Thus, while the other psychotic patients examined in this series tend to have more evidence of organic changes with intellectual deterioration, this does not apply to the NS group. The psychosis in the former may therefore be related to a more generalised disturbance of brain function, whereas the NS group, all of whom have temporal lobe epilepsy, may be developing the psychosis precisely because it is dependent upon epileptic as opposed to more generalised organic processes. One theoretical possibility here relates to the experimental model of kindling; in animal models, if sub-threshold stimuli are given (usually to the amygdala) on a specified schedule, after an interval of time the animal develops behaviour changes and finally epileptic seizures. This has been observed in a variety of species, and subsequent neuronal changes which develop from kindling appear to be permanent and affect several limbic structures (Goddard *et al*, 1969). The higher the animal in the phylogenetic scale, the longer and more difficult kindling becomes, suggesting that in man, months or years may be required for kindling processes to develop. The possibility that arises from our data is that the psychoses in the NS group result from a mechanism similar to kindling, in which changes occur over a period of years in downstream limbic system structures as a result of persistent inter-ictal discharges from a temporal lobe focus.

We have looked at the relationship between prescription of anticonvulsant drugs and psychosis. As in other samples, many patients were receiving several drugs and unfortunately an accurate assessment of the total intake of medication over the years was not available; from the data obtained, little relevant information is gleaned. However, Slater & Beard thought that drugs were irrelevant to the development of psychosis and there are several other studies in which the relationship of anticonvulsant drugs to mental changes has been evaluated (Trimble, 1981). These show that such drugs influence both mental state and behaviour, leading to deterioration of cognitive function (Thompson & Trimble, 1983) and possibly to an increase in neurotic and depressive symptoms. In addition, it has been shown that improvements in such symptoms can occur following reduction of combinations of drugs (Thompson & Trimble, 1982). However, apart from idiosyncratic reactions

and the development of toxic psychoses, there is little evidence in the literature for any links between the persistent taking of anticonvulsant drugs and the development of psychosis.

Our data do not fully support the view that in the schizophreniform psychosis of epilepsy, especially in that form presenting with nuclear symptoms, we are dealing with an organic psychosis as suggested by Slater & Beard. Neither Kristensen & Sindrup nor Flor-Henry, in their studies using non-psychotic epileptic controls, were able to show significantly more lesions on pneumoencephalography in their psychotic epileptic patients; similarly Toone *et al* had the same proportion of lesions in both their index and control samples. Gross anatomical lesions are not clearly associated with the development of the psychopathology while the relevance of possible damage to periventricular structures reflected in the larger values for the septum-caudate distance and the size of the third and fourth ventricle are subject to speculation. Whether they represent the outcome of areas of gliosis in limbic pathways, or reflect some neuroanatomical or neurophysiological change that occurs as a result of a mechanism such as kindling, requires further investigation. There has recently been a growing literature on two types of schizophrenia. Thus, Crow (1980) in particular has advocated that a group which presents with positive symptoms may be separated from one in which negative symptoms predominate. It is of interest therefore that patients in this study who present with 'positive' symptoms come mainly from the temporal lobe group. This further underlines the potential importance of recognising different sub-groups of schizophrenic presentation and also gives weight to the possibility that the temporal lobes in particular are somehow involved in the genesis of positive symptoms in psychiatric patients. Recent evidence from *post mortem* studies presented by Ferrier *et al* (1984) has included biochemical separations which distinguish the groups; further work on neuropathological and neurochemical changes in the temporal lobes in patients with different clinical presentations of schizophrenia would clearly be of great interest. These results should not be interpreted, however, as a return to an old fashioned localisation hypothesis regarding the links between disturbance of brain function and behaviour disorders. We would wish to emphasise that certain clinical symptoms and signs, seen in psychiatric practice and often associated with the disease schizophrenia, signify for the observer disturbances within the central nervous system. Thus, the presence of Schneider's first-rank symptoms which typically emphasise thought disorder and specific forms of auditory hallucinations may suggest a disturbance in the temporal lobe/limbic system of the brain, in particular on the left side. The known association of the left temporal cortex with speech and auditory mechanisms and the well established neuroanatomical links between temporal neocortical sensory areas and anterior temporal/limbic cortex with their associated nuclei, emphasise that there are important neurological mechanisms which underlie these experimental and clinical findings.

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