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Review Article

Epilepsy and Schizophrenia

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Slater's work on the schizophrenia-like psychoses of epilepsy is re-examined in the light of subsequent developments in psychiatry and neurology. Simple causal links of the sort he postulated between epilepsies and psychoses appear increasingly tenuous, despite indications that some psychotic symptoms and some localised structural changes are linked. A resumption of the study of electrophysiological similarities between schizophrenia and limbic epilepsy may offer a useful alternative programme for research.

It is 30 years since Slater, Glithero and Beard presented a series of reviews that were landmarks in the study of psychoses among epileptic patients (Slater & Beard, 1963*a*,*b*; Glithero & Slater, 1963; Beard, 1963). Their studies of chronic 'schizophrenia-like' psychoses used historical, phenomenological, neurological and genetic observations to argue for an aetiological link between epilepsy, especially temporal lobe epilepsy, and these psychoses. Delusions, hallucinations, paranoid and mystical states were common; patients did not show a typically schizophrenic deterioration; and familial schizophrenia or a schizoid pre-morbid personality were relatively rare. In considering possible pathogenetic mechanisms, Slater & Beard (1963*b*) thought "the physiogenic causation of the psychosis the more important factor predisposed to by a stage in . . . a dementing process". Their discussion ended with an explanation of why an apparently rare syndrome should receive such elaborate attention: "It is as a mock-up of the genuine schizophrenic that the schizophrenic-like epileptic is worth special study".

Since then, schizophreniform psychoses among epileptic patients have continued to excite an interest out of all proportion to their frequency. However, the resulting literature contains few uncontested observations. It is a labyrinth, where two surveys currently provide useful orientations (Schmitz & Wolf, 1991; Trimble, 1991). This assessment of developments since 1963 will focus on one question: has understanding of schizophreniform psychoses among epileptic patients changed significantly since Slater's reports?

A first response might justifiably be 'No'. Toone (1991) summarised some generally accepted characteristics of what he termed the 'schizophrenia-like psychosis of epilepsy' (SLPE) (Slater preferred to refer to several 'psychoses', which broadly corresponded to paranoid and hebephrenic subtypes of schizophrenia and a chronic psychosis with intermittent confusion). Toone reports that psychoses that are not clearly distinguishable from schizophrenia occur more commonly than by chance among epileptic patients; that temporal lobe epilepsy is over-represented; and that a family history of schizophrenia is generally absent, while the onset of seizures usually precedes the development of psychotic symptoms by an interval of several years – all reiterations of Slater's 1963 conclusions.

One more controversial finding is added to these – that schizophrenia-like psychoses in epilepsy are preferentially associated with a dominant temporal lobe focus. This reflects later research (e.g. Flor-Henry, 1969; Perez *et al*, 1985), but has been challenged by arguments that the finding is not universal, but an artefact of the Anglo-Saxon emphasis on Schneiderian symptoms in diagnosis of schizophrenia (Stevens, 1991). Stevens argues that because most first-rank symptoms deal with verbal phenomena, an association with dominant rather than non-dominant temporal pathology would be expected, which does not appear to hold when different diagnostic criteria apply, as discussed in the Scandinavian literature. The reliability of judgements about lateralisation has also been questioned, in view of a tendency for apparently unilateral foci to disperse over a series of recordings (Toone, 1991).

Whether or not the association with laterality is upheld, this effort to establish more specific links between psychopathology and changes in neural functioning is in the spirit of Slater's work, even if his own findings here are contradicted. However, it does not represent the only direction in which Slater's research might have been consolidated and developed, and the failure to build on other components of this work, such as family and follow-up studies, may have important negative consequences. Moreover, independent developments in both psychiatry and epileptology suggest that Slater's goal of isolating a chronic psychotic syndrome that is associated with a particular form of epilepsy is more problematic

than it appeared to be. An assessment of how Slater's work stands now certainly needs to ask whether additional conclusions should be added to his own. But it must also ask how far his apparently solid generalisations still hold. To this end, Slater's work will be considered in the light of subsequent developments in psychiatry and epileptology, and alongside later attempts to model the relationship between epilepsy and psychosis.

Developments in psychiatry

In Slater's view, the psychiatric symptoms of his psychotic epileptic patients would have to be diagnosed as schizophrenia, although "The combination of symptoms shown by individuals differs slightly from the most usual schizophrenic patterns" (Slater & Beard, 1963*a*). In fact cases were only admitted for study after an experienced psychiatrist had diagnosed schizophrenia, and psychotic symptoms had persisted for several weeks in clear consciousness. He noted that delusions and religious mystical experiences seemed particularly common among these patients, and some schizophrenic features, notably catatonia and blunting of affect, were unusually rare. However, as catatonia in particular seems to have become even less common through the following decades among patients warranting a diagnosis of schizophrenia (Magrinat *et al*, 1983), these phenomenological differences may no longer hold.

With the advent of standardised symptom inventories, important attempts have been made to improve on retrospective clinical assessments using the Present State Examination (PSE) to record symptomatic profiles of psychotic patients with epilepsy in a standardised way, and to compare these with process schizophrenia (Perez & Trimble, 1982; Toone *et al*, 1982). In these studies, epileptic subjects have not been restricted to cases that Slater would have selected. Nevertheless, Toone *et al* (1982) found weak support for the theory that persecutory delusions are more prevalent among those psychotic patients who have epilepsy, but neither PSE study could validate the claimed absence of personality deterioration. The failure of phenomenological studies to provide a strong discriminator between psychotic patients who have epilepsy and those who do not casts doubt on Slater's case, and adds little to a debate he had to leave unresolved. If a schizophrenia-like syndrome that was restricted to epileptic patients could be identified by reliable methods, it would be possible to investigate how far it reflected the presence of underlying structural pathology (as Slater surmised), or whether it reflected seizure activity independently. Instead, the main contribution of PSE studies has been to link a syndrome of 'nuclear schizophrenia', not exclusive to patients with epilepsy, with left temporal lesions, irrespective of whether these are also associated with EEG evidence of local discharge activity (Perez *et al*, 1985).

Apart from phenomenology, Slater also used evidence from a follow-up study ranging from two to 25 years to argue for the distinctness of the schizophrenia-like psychoses of epilepsy (Glithero & Slater, 1963). He found psychotic symptoms, although chronic, tended to remit, paralleling an improvement in seizures. Again, it is arguable whether a contemporary nosographer might not place the 'psycho-organic' defect state that took their place within the ambit of schizophrenic deterioration. Significantly, there has been little enthusiasm to replicate these follow-up observations – despite their importance for the argument that SLPE was distinct from schizophrenia, and the fact that follow-up of many of Slater's own patients had been complicated by intervening surgery. (The exceptions here, Kenwood & Betts (1988) and Oyebode & Davison (1989), remain incompletely reported and potentially contradictory.) This is disappointing as the significance of Slater's follow-up data has probably increased. For instance, they appear to confound the promising hypothesis that the development of schizophrenic psychoses reflects a progressive facilitation of sub-ictal electrical discharges ('kindling'). Were this the case, the psychotic episodes would be expected to worsen with time rather than remit (Trimble, 1989).

A third and fundamental component of Slater's argument for the distinctness of the schizophrenia-like psychoses of epilepsy was an association with a characteristic history of epilepsy. However, his observation that a gestation period averaging 14 years separated onset of epilepsy and onset of psychosis was immediately challenged. It was dismissed as artefactual on the grounds that anybody whose psychosis did not succeed his epilepsy was excluded, and because of the tendency in the general population for presentations of epilepsy to peak at an earlier age than those of schizophrenia (Stevens, 1966; Davidson & Bagley, 1969). While Slater paid careful attention to the history of his patients' psychoses, their relationship to individual seizure episodes was unclear.

Subsequently, SLPE has tended to be regarded as typically inter-ictal as well as chronic, as opposed to the brief, confusional episodes of ictal and post-ictal psychoses. As such it has often been confused with so-called 'alternate' epileptic psychoses, a much rarer phenomenon often associated with generalised

epilepsy, and in which symptoms need not be schizophrenia-like (Wolf, 1991). (These also differ in being associated with certain drugs, notably ethosuximide and vigabatrin.) However, it is now evident that schizophreniform episodes are an important presentation of post-ictal psychosis, occurring after a delay of one to two days, often in a setting of clear consciousness (Logsdail & Toone, 1988). Clear consciousness has also been noted among the much rarer descriptions of schizophreniform psychoses as an ictal manifestation of partial complex status epilepticus (Wieser *et al*, 1985). No simple temporal relationship between schizophreniform episodes and the electrical activity associated with epilepsy has emerged, perhaps vindicating Slater's caution on the matter, but making argument for the specificity of the schizophrenia-like psychoses of epilepsy even more problematic.

Developments in neurology

The over-representation of temporal lobe epilepsy among Slater's psychotic patients was probably the most crucial finding of his study. It has found a good deal of subsequent support, if not unanimity (Trimble, 1991). Critics of this apparent association have stressed biases of sampling. Slater's own sample of people with active epilepsy was relatively elderly, ensuring that complex partial seizures would be over-represented. Furthermore, the specialised clinics where studies have been performed attract disproportionate numbers of patients having either temporal lobe epilepsy or psychopathology, making combinations of the two untypically common.

It is often said that the lack of proper epidemiological studies is regrettable. Yet the largest studies of an unselected community sample date from Slater's time (Krohn, 1961; Gudmunsson, 1966). Although their psychiatric data lack detail, what detail was supplied has been largely ignored. (In both studies, the occurrence of psychoses among people with epilepsy did not greatly differ from the occurrence among other groups if they had been assessed by comparable criteria.) Of course, the problems that new epidemiological projects would pose are vast: the conjunction of schizophrenia and epilepsy is rare (no cases were detected among general practices in Pond & Bidwell's (1960) survey), while the increased likelihood of sufferers being confined to institutions greatly complicates estimates of prevalence.

Instead, attempts to substantiate the association have to depend on the aggregation of existing studies, whatever their individual defects. However, it is easy for these rare patients to be multiply represented across different reports (Taylor, 1975), while the designation of temporal lobe epilepsy (like that of 'psychosis' or 'schizophrenia') has rarely been comparable from one study to another. One reason for this has been a progressive recognition that many psychomotor seizures do not represent 'temporal lobe epilepsy'. In Slater & Beard's discussions, with a single exception, these had effectively been equated. More recently, one large study using depth electrodes discovered 30% of complex partial seizures to have been extra-temporal in origin (Williamson & Spencer, 1986). (In retrospect, it is interesting that no fewer than seven of Slater's patients diagnosed as temporal lobe epilepsy on the basis of their seizures lacked any electrical evidence for this on repeated investigation, one having a known frontal focus (Beard, 1963).) At the same time, an increasing proportion of generalised seizures have been attributed to rapid spread beyond a focal origin, with the appropriateness of the distinction between generalised and partial seizures being questioned (Travers, 1991). (Again, one can find in Slater's sample ten patients who had an active temporal focus in the absence of clinical partial seizures, who for the purposes of comparative analysis were taken to have 'grand mal' rather than temporal lobe epilepsy.)

Sufficient indication has been given of how, in any study of epileptic psychosis, the classification of a patient's seizures will depend heavily upon the study's age and the sophistication of the investigator(s). The implications of recognising the non-temporal origin of a substantial proportion of psychomotor seizures are illustrated in a recent field study of risk factors for psychosis among outpatients attending a university neurology clinic. Schmitz & Wolf (1993) concluded that occurrence of psychomotor seizures was a significant common factor among psychotic patients, whereas a temporal focus was not.

There have been revisions in models of seizure initiation and spread that also affect the way psychological symptoms and neural events are linked in these illnesses. Seizure spread seems to be determined by factors additional to the presence of a source of abnormal discharges, notably the receptivity of areas surrounding the source (Fenwick, 1986). While seizures are no longer thought to be initiated at a centrencephalic source, their cortical spread is probably subject to remote regulation by the reticular activating system of the excitability of areas adjacent to their origin. Such a compound model of seizure spread is compatible with recent observations of psychological influence on seizure precipitation (Antebi & Bird, 1992). While its relevance for psychosis in epilepsy is uncertain,

it may bear out Wolf's speculation that some epileptic psychoses result directly from deviant seizure spread (Wolf, 1991).

A model of this kind might also justify new interest in Slater's description of the structural changes found among patients he studied, in which diffuse changes at sites beyond the temporal lobes were common. Slater was content to take the high incidence of organic change of any kind as evidence that "the cause of the epilepsy tends to cause the psychosis". When Flor-Henry (1969) found control subjects with uncomplicated epilepsy to be just as likely to show structural changes on air encephalography, Slater's deduction came under fire. Evidence from subsequent radiological studies tended to confirm Flor-Henry's observations (Kristensen & Sindrup, 1978; Toone *et al*, 1982) encouraging the assumption that pathological changes would need to be highly localised in order to support an association between structural change and psychosis. As a result, interest has focused on structural changes within the temporal lobes and their lateralisation, and these reports lack comparative information about other structural changes.

However, when more detailed comparative information about structural changes was given in the study of Perez *et al* (1985), it provided unexpected evidence of bilateral sub-cortical changes among the psychotic patients that were compatible with periventricular and basal ganglia abnormalities. Observations of this kind have been the basis of Stevens' opinion (1991) that "When psychoses and epilepsy do occur together, it appears that brain damage or dysfunction in critical areas beyond the temporal lobe is a common denominator". If these observations are confirmed, the apparent pathogenic effects of dispersed pathology would require explanation. The recognition that structural damage may modify as well as initiate seizures would provide one basis for this. The psychoses of epilepsy could be promoted by non-temporal lesions that were causing some seizures not to generalise, in addition to lesions that acted as an origin for seizures.

Linking schizophreniform psychoses and epilepsy

Slater had contrasted two aetiological models in his own attempts to explain an association between 'temporal lobe' epilepsy and schizophreniform-like psychoses. One emphasised the cumulative disruption to normal psychic functioning that resulted from seizures characterised by partial or total disruption to consciousness and volition. Following Pond (1962), he termed this the 'psychodynamic' hypothesis. Although Slater did not favour this explanation, it is echoed in recent work of Schmitz & Wolf (1993) that emphasises psychomotor seizures as an important risk factor, irrespective of localisation. (Because they found no association with the length and severity of seizure history, Schmitz & Wolf chose to interpret this in non-psychological terms, postulating that a physiological abnormality maintained an altered state of excitability in individuals prone to psychosis.)

Slater's preferred line of explanation viewed psychopathology and epilepsy alike as consequences of underlying structural pathology, terming this the 'physiogenic' hypothesis. Some of the ways this has been elaborated were discussed in the previous section. However, an important innovation among theories of how psychosis and epilepsy might reflect a common pathology has stressed the ontogenetic rather than the anatomical impact of epileptogenic lesions among people who also become psychotic (Taylor, 1971).

In a reassessment of Slater's results, Taylor found support for the idea that it was not the timing of seizures that was critical for the later development of psychosis, but the juncture in an individual's development at which the underlying neuropathology appeared. A detailed assessment of 100 patients on whom histological information also became available after surgery allowed him to attribute a strong correlation between later emergence of schizophreniform psychoses and the presence of one of a group of 'alien tissue' lesions (Taylor, 1975; Bruton, 1988) to their common, embryonic inception. It was an appealing idea, not only giving form to an independent factor that could underlie both the epilepsy and the psychosis, but also, through its developmental focus, offering some prospect of reconciliation between psychodynamic and neuropathological accounts of the genesis of these psychoses.

Subsequently, Taylor's views have been partially challenged in a reanalysis of the neuropathology of the same series of patients by Roberts (1991), who thought the involvement of neural tissue decisive in explaining the selective association of psychoses with lesions such as gangliogliomas. Too much should not be inferred from this heavily worked series, especially as the factors predisposing to psychosis among candidates for surgery, which these patients were, may not be the same as those applying to other groups with epilepsy (Mace & Trimble, 1991). The findings remain compatible with a revised ontogenetic argument being forwarded by Stevens (1992), in which she proposes that the critical factor predisposing to schizophrenic psychoses among some people with epilepsy (as well as other varieties of

of organically induced psychosis) is the reactive occurrence of abnormal neuronal regeneration, especially when this coincides with a vulnerable period in adolescence.

The schizophrenia-like psychoses in retrospect

So how does Slater's pioneering work bear up 30 years later? His arguments that temporal lobe epilepsy was critical to the appearance of a distinct schizophreniform syndrome were founded on an elaborate body of observations. However, comparisons made then between 'temporal' and 'generalised' epilepsies, or between Slater's patients and 'typical' schizophrenics, seem less valid now, and attempts to draw a simple correspondence between temporal lobe epilepsy and schizophrenic psychoses have been frustrated by continuing shifts in the boundaries of either – a state of affairs that does much to explain why no authoritative classification of psychopathology in epilepsy could emerge throughout this time. At the same time, the additional observations that Slater used to support his argument have not been corroborated because successors have largely abstained from the further prognostic, epidemiological and genetic comparisons that were indicated, given the idiosyncracies of his sample and subsequent changes in diagnostic fashion.

Instead, one plank of Slater's edifice received selective emphasis. His interest in neurological lesions that might be common to epilepsy and psychosis has been advanced to a point where, independently of epilepsy, Schneiderian ('nuclear') schizophrenia has been linked to medial temporal pathology, preferentially affecting the dominant lobe (Trimble, 1990; Roberts, 1991). This link rests heavily on structural studies, in which epilepsy has indicated the presence of local pathology, but with the tendency that this, rather than seizure activity, is viewed as the critical psychotogenic factor.

If Slater's research strategies were pursued in an unduly selective way, some inherent limitations in his methods have been copied too readily also. For example, Slater had emphasised the biological precipitants of psychosis among epileptic patients to the almost total exclusion of the role of other important factors such as the emotional, behavioural and social handicaps they share (Wolf *et al*, 1986), and other workers followed suit. Another instance is how Slater's failure to complement his observations with a study of appropriate controls has not always been corrected. Indeed, when appropriate comparisons have been attempted, the results have challenged some of Slater's basic assumptions. Flor-Henry's (1969) discovery of comparable radiological findings among non-psychotic patients with epilepsy has already been cited. In some other studies, familial schizophrenia was unexpectedly rare when sought, albeit with less than exhaustive methods, among the control subjects of Toone *et al* (1982) and Oyebode & Davison (1989).

The influence of Slater's work on the schizophrenia-like psychoses of epilepsy has been profound, if not positive in every respect. Now, as his conclusions risk appearing more and more tenuous in the light of subsequent developments, work to seek new regularities continues. As Slater's view that structural change would be primary has continued to dominate this work, the corollary that epileptic seizures themselves have no independent role in the pathogenesis of schizophreniform psychoses receives less dissent. However, this is by no means proven, and begs the question whether there are other avenues that research into the relationship between epilepsy and schizophrenia should pursue.

A different 'mock-up' for schizophrenia?

The idea that seizures were a direct cause of relatively chronic psychoses was widely prevalent prior to Slater, and has probably been responsible for ensuring that, within the field of schizophrenia research, the potential contribution of electrophysiology, and the study of spontaneous electrical activity in particular, has not been overlooked. In some ways, the rewards of this labour have been disappointing. The apparent independence of the EEG discharges that typically accompany seizures from findings in psychosis does not encourage the idea that seizures lead to psychosis. Among patients subject to inter-ictal psychoses, the scalp EEG generally shows no significant change during psychosis (Ramani & Gumnit, 1982). Furthermore, in a few cases even background abnormalities will disappear to leave a trace that is unusually stable during the psychotic episode – the so-called 'forced normalisation' of Landholt (1958).

Slater's work had assumed there was no physiological association between psychosis and epilepsy. He took them to be discrete entities that would need to be linked in other ways. Slater makes relatively little reference to the considerable electrophysiological data that were at his team's disposal. However, there is an important oversight in his review of prior and contemporary work, which could have challenged this separatist stance: his neglect of the surgical literature.

In the 1950s, neurosurgery was not only a recognised treatment for schizophrenia in many countries, but patients would also sometimes undergo

relatively invasive electrophysiological investigations comparable to those preceding surgery for epilepsy. Kendrich & Gibbs (1957) had already reported a remarkable series in which the use of implanted electrodes allowed comparisons between patients with a chronic schizophrenic psychosis and those with a similar psychosis and psychomotor epilepsy. The schizophrenics were as likely as those with recognised epilepsy to show spike discharges in medial, temporal and frontal structures. Heath (1986), in a set of precise comparative studies carried out at this time, had noted similar types of abnormal discharge which did not spread beyond the amygdala, hippocampus and septal region, finding these in schizophrenics as well as in patients with a psychosis associated with epilepsy. At the same time, they could be distinguished, spiking being most marked in the septal areas in schizophrenia, a distribution which would be transiently mimicked when psychosis intervened in epilepsy, although the patterns of discharge in either psychosis remained distinguishable. Depth electrode studies therefore implied that the syndromes were closely related yet distinct (Heath, 1962). In addition to similar findings, Kendrich & Gibbs (1957) had reported that temporal lobe surgery that selected medial structures was nearly always beneficial for their schizophrenic patients. Nevertheless, deep electrode studies and operative treatment for schizophrenia were discontinued.

Slater's study was to set a very different direction for research from this point. Later studies of psychotic patients that focused once more on deep electrode findings tended to do so in a piecemeal way, using small numbers. However, the possibility that activity recorded by superficial and deep electrodes can be relatively autonomous has gained more recognition, and is consistent with the prospect that, in cases of psychosis, epileptiform discharges are pathogenic in their own right.

In retrospect, a study by Kristensen & Sindrup (1978) may prove pivotal. They monitored a large group of patients with psychosis and epilepsy using sphenoidal as well as scalp electrodes, and compared them with patients having complex partial seizures in the absence of psychosis. The patients susceptible to psychosis were far more likely to have seizures with a medio-basal focus than those who did not become psychotic, and could therefore be differentiated physiologically. No investigation on this sort of scale has been able, for obvious ethical reasons, to take patients with 'functional' psychoses as controls, so that no replication of Heath's work has been attempted. (It must be admitted that Heath's psychiatric descriptions seem, in the light of nosographic developments, far more questionable than Slater's.) However, the prospect of doing so seems rather closer now, owing to progress in developing non-invasive methods of recording spontaneous as well as induced electrical potentials through magnetoencephalography. Although its lack of accuracy, sensitivity and patient-friendliness limit its usefulness at present (Reeve *et al*, 1989), magnetoencephalography promises to permit the study of spontaneous electrical activity in subcortical structures by non-invasive means (Fenwick, 1990).

Several objections to the idea that a 'schizophrenia-like psychosis of epilepsy' is a distinct psychiatric syndrome were described in earlier sections. These must entail that any attempt to cast it as a 'mock-up' of schizophrenia in the way Slater had proposed is also dubious. However, the prospect that a new generation of depth electrical studies could explore electrophysiological similarities and contrasts between schizophreniform illnesses occurring in epileptic and non-epileptic subjects means it may be time to seek a different sort of 'mock-up'. An identifiable pattern of limbic discharge prevalent among patients with joint diagnoses of epilepsy and psychosis could be sought, and this might well provide a more fertile kind of 'mock-up' for other psychiatric disorders. The exercise would represent a return to questions from which Slater's studies have largely wrested attention for decades. However, if the investigation were successful, the epidemiological, genetic, historical, pathological and phenomenological referrents of a physiological 'mock-up' would remain to be traced. Slater's work seems certain to remain a formidable example for anyone attempting to do so.

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References

ANTEBI, D. & BIRD, J. (1992) The facilitation and evocation of seizures. *British Journal of Psychiatry*, **160**, 154–164.

BEARD, A. W. (1963) The schizophrenia-like psychoses of epilepsy: (ii) Physical aspects. *British Journal of Psychiatry*, **109**, 95–112.

BRUTON, C. J. (1988) *The Neuropathology of Temporal Lobe Epilepsy*. Oxford: Oxford University Press.

DAVIDSON, K. & BAGLEY, C. R. (1969) Schizophrenia-like psychoses associated with organic disorders of the central nervous system: a review of the literature. *British Journal of Psychiatry: Special Publication No. 4*, 113–183.

FENWICK, P. B. C. (1986) What is an EEG epileptic discharge? In *What is Epilepsy?* (eds M. R. Trimble & E. H. Reynolds). London and Edinburgh: Churchill Livingstone.

—— (1990) The use of magnetoencephalography in neurology. *Advances in Neurology*, **54**, 271–282.

FLOR-HENRY, P. (1969) Psychosis and temporal lobe epilepsy. A controlled investigation. *Epilepsia*, **10**, 363–395.

GLITHERO, E. & SLATER, E. (1963) The schizophrenia-like psychoses of epilepsy: (IV) Follow-up record and outcome. *British Journal of Psychiatry*, **109**, 134–142.

GUDMUNSSON, G. (1966) Epilepsy in Iceland. *Acta Neurologica Scandinavica* (suppl.), **25**, 1–124.

HEATH, R. G. (1962) Common clinical characteristics of epilepsy and schizophrenia. *American Journal of Psychiatry*, **11**, 1013–1026.

——— (1986) Studies with deep electrodes in patients intractably ill with epilepsy and other disorders. In *What is Epilepsy?* (eds M. R. Trimble & E. H. Reynolds), pp. 126–138. Edinburgh: Churchill Livingstone.

KENDRICH, J. R. & GIBBS, F. A. (1957) Origin, spread and neurological treatment of the psychomotor type of seizure discharge. *Journal of Neurosurgery*, **14**, 270–284.

KENWOOD, C. & BETTS, T. (1988) 20-year follow-up of epileptic psychosis. *British, Danish, Dutch Epilepsy Congress Abstracts*, p. 53.

KRISTENSEN, O. & SINDRUP, E. H. (1978) Psychomotor epilepsy and psychosis I & II. *Acta Neurologica Scandinavica*, **57**, 361–379.

KROHN, W. (1961) A study of epilepsy in northern Norway, its frequency and character. *Acta Psychiatrica Neurologica Scandinavica* (suppl.), **150**, 215–225.

LANDHOLT, H. (1958) Serial EEG investigations during psychotic episodes in epileptic patients and during schizophrenic attacks. In *Lectures on Epilepsy* (ed. A. M. Lorentz De Haas), pp. 11–430. Amsterdam: Elsevier.

LOGSDAIL, S. J. & TOONE, B. K. (1988) Post-ictal psychoses. *British Journal of Psychiatry*, **152**, 246–252.

MACE, C. J. & TRIMBLE, M. R. (1991) Psychosis following temporal lobe surgery: a report of 6 cases. *Journal of Neurology, Neurosurgery and Psychiatry*, **54**, 639–644.

MAGRINAT, G., DANZIGER, J. A., LORENZO, I. C., *et al* (1983) A reassessment of catatonia. *Comprehensive Psychiatry*, **24**, 218–228.

MCKENNA, P. J., KANE, J. M. & PARRISH, K. (1985) Psychotic syndromes in epilepsy. *American Journal of Psychiatry*, **142**, 895–904.

OYEBODE, F. & DAVISON, K. (1989) Epileptic schizophrenia: clinical features and outcome. *Acta Psychiatrica Scandinavica*, **79**, 327–331.

PEREZ, M. & TRIMBLE, M. R. (1982) The phenomenology of the chronic psychoses of epilepsy. In *Advances in Biological Psychiatry, Vol. 8* (eds W. R. Koella & M. R. Trimble), pp. 95–105. Basel: Karger.

———, ———, MURRAY, N. M. F., *et al* (1985) Epileptic psychosis: an evaluation of Present State Examination profiles. *British Journal of Psychiatry*, **146**, 155–163.

POND, D. A. (1962) Discussion remark. *Proceedings of the Royal Society of Medicine*, **55**, 316.

——— & BIDWELL, B. H. (1960) A survey of epilepsy in fourteen general practices. II. Social and psychological aspects. *Epilepsia*, **1**, 285–299.

RAMANI, V. & GUMNIT, R. J. (1982) Intensive monitoring of interictal psychosis in epilepsy. *Annals of Neurology*, **11**, 613–622.

REEVE, A., ROSE, D. F., WEINBERGER, D. R. (1989) Magnetoencephalography: applications in psychiatry. *Archives of General Psychiatry*, **46**, 573–576.

ROBERTS, G. W. (1991) Schizophrenia: a neuropathological perspective. *British Journal of Psychiatry*, **158**, 8–17.

———, DONE, D. J., BRUTON, C., *et al* (1990) A 'mock-up' of schizophrenia: Temporal lobe epilepsy and schizophrenia-like psychosis. *Biological Psychiatry*, **28**, 127–143.

SCHMITZ, B & & WOLF, P. (1991) Psychoses in epilepsy. In *Epilepsy and Behaviour* (eds O. Devinsky & W. H. Theodore), pp. 97–128. New York: Wiley-Liss.

——— & ——— (1993) Psychosis in epilepsy: frequency and risk-factors. *Journal of Epilepsy* (under submission).

SLATER, E. & BEARD, A. W. (1963*a*) The schizophrenia-like psychoses of epilepsy: (i) Psychiatric aspects. *British Journal of Psychiatry*, **109**, 95–112.

——— & ——— (1963*b*) The schizophrenia-like psychoses of epilepsy: (v) Discussion and conclusions. *British Journal of Psychiatry*, **109**, 143–150.

——— & GLITHEROE, E. (1963) The schizophrenia-like psychoses of epilepsy: (iii) Genetical aspects. *British Journal of Psychiatry*, **109**, 113–133.

STEVENS, J. R. (1966) Psychiatric implications of psychomotor epilepsy. *Archives of General Psychiatry*, **14**, 461–471.

——— (1991) Psychosis and the temporal lobe. In *Advances in Neurology Vol. 55* (eds D. Smith, D. Treiman & M. Trimble), pp. 79–95. New York: Raven Press.

——— (1992) Abnormal reinnervation as a basis for schizophrenia: a hypothesis. *Archives of General Psychiatry*, **49**, 238–243.

TAYLOR, D. (1971) Ontogenesis of chronic epileptic psychoses: a reanalysis. *Psychological Medicine*, **1**, 247–253.

——— (1975) Factors influencing the occurrence of schizophrenia-like psychoses in patients with temporal lobe epilepsy. *Psychological Medicine*, **5**, 245–254.

TOONE, B. K., DAWSON, J. & DRIVER, M. V. (1982) Psychoses of epilepsy: a radiological evaluation. *British Journal of Psychiatry*, **140**, 244–248.

———, GARRALDA, M. E. & RON, M. A. (1982) The psychoses of epilepsy and the functional psychoses: a clinical and phenomenological comparison. *British Journal of Psychiatry*, **141**, 256–261.

——— (1991) The psychoses of epilepsy. *Journal of the Royal Society of Medicine*, **84**, 457–459.

TRAVERS, R. F. (1991) Limbic epilepsy. *Journal of the Royal Society of Medicine*, **84**, 454–456.

TRIMBLE, M. R. (1989) Kindling, epilepsy and behaviour. In *The Clinical Relevance of Kindling* (eds T. G. Bolwig & M. R. Trimble), pp. 177–190. Chichester: Wiley.

——— (1990) First-rank symptoms of Schneider: a new perspective: *British Journal of Psychiatry*, **156**, 195–200.

——— (1991) *The Psychoses of Epilepsy*. New York: Raven Press.

WIESER, H. G., HAILEMARIAM, S., REGARD, M., *et al* (1985) Unilateral limbic epileptic status activity: stereo EEG, behavioural, and cognitive data. *Epilepsia*, **26**, 19–29.

WILLIAMSON, P. D. & SPENCER, S. S. (1986) Clinical and EEG features of complex partial seizures of extratemporal origin. *Epilepsia*, **27** (suppl. 2), S46–S63.

WOLF, P. (1991) Acute behavioral symptomatology at disappearance of epileptiform EEG abnormality. In *Advances in Neurology Vol. 55* (eds D. Smith, D. Treiman & M. Trimble), pp. 127–142. New York: Raven Press.

———, THORBECKE, R. & EVEN, W. (1986) Social aspects of psychosis in patients with epilepsy. In *Psychopathology in Epilepsy: Social Dimensions* (eds S. Whitman & B. P. Hermann). New York: Oxford University Press

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