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Post-Ictal Psychoses
A Clinical and Phenomenological Description

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Post-ictal psychoses have so far received little attention. The clinical details of 14 cases, diagnosed according to newly formulated criteria, were examined. Psychoses were usually precipitated by a run of seizures and occurred after a lucid interval. The seizures were partial complex with secondary generalisation in 11 cases. Catego analysis of the Present State Examination confirmed pleomorphic phenomenology. Follow-up details were available in all cases, for up to eight years. Psychoses tended to recur. Similarities with chronic epileptic psychosis are discussed, and a possible organic aetiology for post-ictal psychosis is proposed.

The association between epilepsy and psychosis has generated considerable debate and interest since the nineteenth century. Acute states of excitation related to seizures were first described by Esquirol in 1838 and later taken up by Jackson (1875). Modern research using scientific measurement (Slater *et al*, 1963) and methods of observation of known reliability (Perez & Trimble, 1980; Toone *et al*, 1982) has concentrated on the chronic, or inter-ictal psychoses; observations on post-ictal psychotic states have been largely ignored. Some studies include a proportion of such patients in a larger epileptic psychiatric cohort (Clark & Lesko, 1939; Levin, 1952; Dongier, 1959; Betts, 1974), but the salient features that would permit precise diagnostic classification are not always available or data is arranged in such a way that it is not possible to draw conclusions about this particular group. Basic information remains unknown (e.g. type of epilepsy, phenomenology, time course and outcome), and it is not possible to examine the possible links between post-ictal and inter-ictal epileptic psychoses. The present study aims to clarify the position by describing 14 cases.

Method

Admissions to the Bethlem Royal and Maudsley Hospitals were screened by computer search for the years 1967–1985; eight cases were thus identified. The department of Psychological Medicine at King's College Hospital was screened for 1980–1985; four cases were identified. One patient attended the National Hospitals for Nervous Diseases and one the David Lewis Centre for Epilepsy. Each patient had been in the care of a consultant psychiatrist with a special interest in epilepsy and had been studied as an in-patient at the time of psychosis. An EEG was available in all cases and most patients had undergone multiple recordings. Case-notes and EEG recordings were carefully scrutinised; further information from other hospitals was obtained wherever possible. The inclusion criteria for epilepsy were those of Gunn & Fenton (1969), and this diagnosis was unequivocal in each case.

A diagnosis of post-ictal psychosis was accepted if the following four criteria were fulfilled.

The first was that the episode of confusion or psychosis manifested immediately upon a seizure or emerged within a week of the return of apparently normal mental function.

The second criterion was that the psychosis had a minimum length of 24 hours and a maximum length of three months.

The third criterion was that the mental state was characterised by one of the following:

(a) clouding of consciousness, disorientation, or delirium
(b) delusions, hallucinations, in clear consciousness
(c) a mixture of (a) and (b).

The fourth criterion was that there was no evidence of the following extraneous factors which might have contributed to the abnormal mental state:

(a) anticonvulsant toxicity – based on anticonvulsant levels where possible and also physical examination for evidence of cerebellar dysfunction in each case
(b) a previous history of inter-ictal psychosis
(c) EEG evidence of minor status
(d) recent history of head injury, or alcohol or drug intoxication.

Clinical evaluation

Information included the following: onset age of epilepsy; type of seizure; anticonvulsant drug treatment; onset age of psychosis; lucid interval; mode of presentation; family history of psychiatric illness; premorbid personality and social situation; physical examination; psychiatric phenomenology; laboratory examination, including anticonvulsant levels; CT-scan; serial EEG examinations; psychometry and long-term follow-up.

When possible the Present State Examination (PSE) (Wing *et al*, 1974), a structured psychiatric interview of established high reliability, was employed. Both authors had been trained in the use of the PSE. The Syndrome Check List, a schedule derived from the PSE and adapted for retrospective evaluation of case records, was employed when PSE information gained at the time of the psychosis was not available. Symptoms thus recorded were fed into the Catego computer program, which classified them in a hierarchical manner.

Results

The results are summarised in Table I.

General characteristics

There were nine males and five females, average age 35 years at the time of the study. Five had one psychotic episode, six had 2–4 episodes, and three had multiple post-ictal psychoses.

Family histories were unremarkable except for one patient who had a father who was said to drink alcohol to excess, and another patient who had a cousin who was reputed to suffer seizures.

Five patients were married, two lived with parents, one lived in a convent and the remaining six lived in hostels. Five patients were employed. Six were given an extra diagnosis of personality disorder by their doctors according to ICD-9 criteria (World Health Organization, 1978).

General characteristics of epilepsy

The age of onset of epilepsy ranged from a few months to 41 years (mean 16.7 years) and the onset of first post-ictal psychosis was 17–56 years (mean 32.2 years). The gap between onset of epilepsy and onset of psychosis was 3–33 years (mean 15.5 years). Epilepsy classification was undertaken according to the criteria of Gastaut (1970). Three of the 14 patients had primary generalised epilepsy (PGE) causing *grand mal* seizures, and the remaining 11 had focal epilepsy causing partial complex seizures: generalisation of the seizures occurred intermittently in all 11. Two patients were also thought to suffer hysterical seizures, although these seizures were not temporally related to psychosis.

Seizure control, despite careful treatment, was variable. Five patients had fits once monthly or less often; one patient had them more than monthly but less than weekly; seven had seizures at least weekly. One patient varied in seizure frequency between these extremes, probably because of intermittent non-compliance in taking medication.

Four patients were taking a single anticonvulsant drug; the remainder were on polytherapy. Phenytoin, carbamazepine, sodium valproate and phenobarbitone were the most commonly prescribed, in varying combinations and doses. Blood levels taken at the time of psychosis were within the therapeutic range in 12 assays, and below in eight.

Seizures prior to the onset of psychosis

In 12 of the patients there was a clear history of an increase in major seizure frequency prior to the onset of psychosis, usually as a cluster of two or three. In one case the patient suffered one unusually prolonged fit from which it took him longer than usual to recover, and in the last case no reliable history could be obtained.

Lucid interval

Eleven of the 14 patients made a recovery from the seizures and immediate post-ictal confusion to the point where they (and a witness where available) said they were back to normal. One further patient developed clear consciousness prior to psychosis, but had not made a full recovery from the seizures, as her Todd's paralysis was incompletely resolved. It was after this interval of apparent normality of mental state that the onset of psychosis occurred. This lucid interval was 1–2 days in eight of the 12 patients; the range for the 12 was 1–6 days, mean 2.5 days. The remaining two patients were difficult to categorise: one was confused for 24 hours after recovering from epileptic status before becoming psychotic, the second was having numerous fits daily for a month before psychosis occurred.

Mode of presentation

Three patients were brought to a casualty department by family members, three were admitted after GP referral, four more were admitted after a domiciliary visit by a psychiatrist (two requests were from hostel wardens) and four were seen as in-patient consultations to other hospital departments. In this latter group of four, one had been under the care of physicians because of epileptic status, another was admitted to a labour ward suffering the delusion that she was in labour, and the last two had been admitted to neurological wards primarily because of mental state disturbance, but in the care of consultants who had been managing their epilepsy previously. Each admission was considered urgent, as were the ward consultations, although only one patient caused a significant public disturbance prior to coming into hospital.

Features of psychosis

All patients were given a diagnosis of post-ictal psychosis by their medical attendants. Of the nine who were confused at the onset of the psychosis, seven had experienced the intervening lucid interval of clear consciousness after recovery from seizures and before the onset of psychotic phenomena. This confusion persisted throughout the psychosis in five. Three further patients had no evidence of confusion during their psychosis, and the remaining two could not co-operate with testing to help clarify their conscious level. Only one of the patients had primary delusions and thought disorder. Nine patients had a markedly abnormal mood: elevated in three, depressed in four, and both (at different times) in two more. Three

TABLE I
Summary of results

Sex	*Epilepsy onset age: years*	*Epilepsy type*	*Seizure incidence*[1]	*Psychosis onset age: years*	*Number of psychoses*	*PSE category*	*CT scan*	*Psychosis length: days*	*Confusion*	*EEG when non-psychotic*	*EEG change during psychosis*
M	11	Focal, 2° generalised	+	19	1	Other psychosis	Normal	90	Initial	Right-sided sharp and slow waves	Increased slow waves; sharp waves absent
M	12	Focal, 2° generalised	+	19	3	Paranoid psychosis	Normal	4	None	Bilateral spikes and slow waves	Increased spikes and slow waves
M[2]	12	Focal, 2° generalised	+ +	25	>10	Schizophrenic psychosis	Right occipital pole tumour	2	None	Bilateral sharp and slow waves	No change
M	23	Focal, 2° generalised	+	37	1	Other psychosis	Cerebral atrophy	21	Throughout	Bilateral sharp and slow waves	Increased slow waves
M	0	Focal, 2° generalised	+ +	27	>10	Paranoid psychosis	Normal	10	?	Left-sided focal spikes; bilateral slow waves	Increased bilateral spikes
M	10	Focal, 2° generalised	+ +	19	2	Other psychosis	Right parietal low attenuation lesion	2	?	Bilateral spikes and slow waves	Increased slow waves
F[2]	8	Focal, 2° generalised	+ +	37	>10	Manic psychosis	Normal	2	None	Right-sided spikes	Increased right-sided spikes
M	40	Primary generalised epilepsy	+	46	1	Manic psychosis	Widened fissures and sulci	4	Throughout	Minor non-specific abnormalities	Not done
F	34	Primary generalised epilepsy	+	56	2	Schizophrenic psychosis	Not done	15	Initial	Bilateral slow waves	Bilateral sharp waves; increased slow waves
F	14	Focal, 2° generalised	+ +	29	3	Paranoid psychosis	Not done	1	Throughout	Bilateral sharp and slow waves	Not done
F	13	Focal, 2° generalised	Variable	46	1	Schizophrenic psychosis	Dilated lateral ventricles	9	Throughout	Generalised slowing	Bilateral sharp waves
F	41	Focal, 2° generalised	+ +	52	1	Manic psychosis	Normal	28	Initial	Minor non-specific abnormalities	Bilateral spikes and sharp waves; increased slow waves
M	14	Primary generalised epilepsy	+ +	17	4	Paranoid psychosis	Normal	5	Initial	Generalised slowing	Not done
M	2	Focal, 2° generalised	+ +	22	2	Schizophrenic psychosis	Normal	7	Throughout	Bilateral spikes and slow waves	Not done

1. + = monthly or less, + + = monthly or more.
2. Case history given.

patients presented in stupor: one was catatonic; another turned out to be depressed; no definite diagnosis was made in the third case. Paranoid delusions were reported in six patients; these were poorly systematised. Six patients reported visual hallucinations, six had auditory hallucinations, and two somatic hallucinations. The orthodox description of post-ictal psychosis (Lishman, 1978) has four main elements – confusion, visual and auditory hallucinations, and paranoid ideation; all of these features were present in only 5 of the 14 cases.

PSE data

An analysis was undertaken of the phenomenology of one psychotic episode in each patient. The best documented episode was taken in those cases where there was more than one. In each of these cases the abnormalities of mental state did not differ markedly from one episode to another, and apart from the confusion, did not change notably during each episode. Eighteen Catego subclasses were found: five schizophrenia (including one catatonic schizophrenia), four paranoid psychosis, three affective psychosis, two mania, one hypomania, one psychotic depression, and two borderline psychosis. The final Catego classes were schizophrenic psychosis, four; manic and mixed affective psychosis, three; paranoid psychosis, four; and other psychosis, three. The presence of confusion did not lead to a markedly differing phenomenology: the four PSE classes were found within the group of five patients confused throughout psychosis as well as within the remaining group of seven patients who were known to be confused initially or not at all.

Neurological examination

One patient had a longstanding left hemiparesis. During the psychotic phase, one had an unsteady gait, but no other abnormalities on examination. Another developed a right-sided Todd's paralysis and dysphasia: the dysphasia resolved over four days, and the weakness had almost gone by day 8, when the psychosis started.

Investigations

CT scan abnormalities were detected by a neuroradiologist in 5 of 12 patients: one had a right occipital pole tumour, one had an area of low attenuation superficially in the right parietal lobe, two had dilated lateral ventricles, and one had widened fissures and sulci.

EEG examination of the patients when not psychotic showed five patients with slow wave abnormalities only. There were intermittent spikes and/or sharp waves in the other nine: right unilateral location in two, left unilateral in one and bilateral in six. Repeat EEG examination during psychosis was undertaken in ten patients. There was no change in one, reduced abnormalities in another, and increased spikes and/or sharp waves in six. Slow waves were increased in six. There was no particular association between EEG abnormalities and clinical picture, apart from an increase in slow waves in those who were confused at the time of assessment.

Psychometry was undertaken in 11 patients; in three, assessment was during the psychotic state. IQ ranged from 80 to 120 in the ten patients in which this was measured; the mean IQ of the group was 92. There were localising abnormalities (visual memory, visuospatial ability, arithmetic, and naming ability) in seven cases and these correlated with an EEG focus or localised CT scan abnormality in three.

B12 and folate assays were performed in eight patients. Serum B12 was in the normal range for each. One patient had a red cell folate estimation, which was in the normal range. Seven others had estimations of serum folate: this was marginally reduced in three – 2.0, 2.8, 2.8 μg/l (normal range 3–15 μg/l).

Course and outcome

Six patients had a serious behaviour disturbance while in-patients, including one who made a suicide attempt and two who were physically aggressive. Two patients were held under the Mental Health Act. The length of psychosis varied from one to 90 days, mean 14.3 days, although eight of the patients had recovered within one week.

Eight were treated with major tranquillisers: trifluoperazine, chlorpromazine, or haloperidol. One patient was given lithium. ECT was not prescribed.

Six patients had further generalised seizures while psychotic, and in five these were followed by a worsening of psychotic symptoms. Two of these patients also had partial complex seizures without secondary generalisation, and in one this led to an exacerbation of the psychosis.

Follow-up details were available in all of the patients, for a period of three months to eight years after the first post-ictal psychotic episode. Four patients died: one from an astrocytoma, two from complications of epileptic seizures, and one was found drowned in a river (the cause was unknown). Two patients developed a chronic psychosis, in each eight years after the first post-ictal episode, and a further patient started to experience fleeting inter-ictal auditory hallucinations six months following the only episode of post-ictal psychosis she had experienced.

Illustrative case histories

Case 1 (third case in Table 1)

MB, a 33-year-old farm worker, first developed complex partial seizures with intermittent secondary generalisation at the age of 12. At 25 the patient experienced the first post-ictal psychotic episode, the symptoms manifesting themselves 48 hours after a major seizure. At the same time, the seizure frequency increased and the aura became more complex. Investigations yielded evidence of a right occipital pole astrocytoma.

During the remaining ten years of the patient's life, post-ictal psychotic episodes occurred at ever-decreasing intervals, while the duration of the episodes progressively increased from 24 hours at the onset of the disorder to a point when, shortly before death, chronic psychosis ensued. During this period the lucid interval diminished from 24 hours to 6 hours. At the beginning, psychosis appeared

only after major seizures; towards the end it followed upon partial seizures.

A full mental state examination was carried out at a point where chronic psychosis was about to supervene. Primary delusions, auditory hallucinations, thought disorder and ideas of reference were noted. The patient's original delusional idea, that he had been in communication with the angels via a special pen they had given him, was present. He now believed that he had been responsible for World War III and had destroyed the world, and that the tooth fairies were using his brain to smuggle drugs.

He was treated with major tranquillisers; these reduced the intensity of the psychotic phenomena but did not completely dispel them. Radiotherapy was given a few months prior to his death, but this had no marked effect on his psychotic phenomena, which had become continuous.

Case 2 (seventh case in Table 1)

PB is a housewife who suffered blank spells since childhood. During her teenage years, these became associated with an unpleasant abdominal feeling and a sensation of fear. The attacks occurred approximately weekly and led to tonic–clonic seizures, 2–3/annum. Despite careful treatment these partial complex seizures with intermittent secondary generalisation increased in frequency, and her first psychosis occurred after a run of generalised seizures.

Over the ensuing six years, seizures have occurred with gradually increasing frequency: she experiences major attacks approximately monthly and minor episodes weekly. Psychoses have been correspondingly more frequent, now approximately monthly, and are occasionally precipitated by a run of partial complex seizures without secondary generalisation.

Post-ictal psychotic features conformed to a consistent pattern. One day after the seizure she would develop elevated mood, become overtalkative and suffer persecutory ideas if prevented from undertaking irrational, grandiose projects. Visual hallucinations were present from the beginning; auditory hallucinations developed as the episodes became more frequent. The content of the psychosis varies; on one occasion she wanted to fly to Scotland on a settee to visit a friend, despite acknowledging that she had died a year previously. On another occasion, when preoccupied with religious ideas, she said she could purify her sister, that the neighbours were evil and that her husband and brother were against her. The prescription of lithium (levels of 0.5–0.9 mmol/1) reduced the affective element of the psychosis. Major tranquillisers have been given in an effort to avoid admission to hospital. As a result of her intermittently disturbed mental state and despite the fact that she returns to normal between episodes, her employment has been terminated and her marriage is now under considerable strain.

She is currently undergoing evaluation for a neurosurgical procedure in an effort to help control her seizures and their sequelae.

Discussion

Post-ictal psychosis is a relatively rare condition: a computerised search of the medical records of the Maudsley Hospital showed a referral ratio of 12:1 chronic or inter-ictal psychosis to post-ictal psychosis. As the condition is transient and presents acutely, the number may be unrepresentative, as some are probably admitted to their local psychiatric hospital, or perhaps not admitted to hospital at all. In view of the small size of the group reported here, and the possibility that teaching hospital referrals may be unrepresentative, any conclusions made are tentative.

Three features of the methodology merit further amplification. The diagnostic operational criteria required that the onset of psychosis should occur within a week of return of normal function after a seizure, and should endure for at least 24 hours but for no longer than three months. Little is known of the time course of the post-ictal psychoses. The criterion suggested, therefore, is to a degree arbitrary, and may subsequently need to be modified. However, all of our cases fulfilled this criterion, and we did not encounter a further case that satisfied the other operational criteria but fell outside this time-scale. A second point concerns the exclusion of patients who had previously suffered an inter-ictal psychosis. This criterion was used to prevent possible diagnostic uncertainty in the post-ictal psychotic group, although no patients had to be excluded on this basis. The third point concerned the use of the PSE. This was not designed for use in organically determined abnormal mental states, and the Catego classification presented here must be viewed with some caution (Wing, 1983). Nevertheless, the PSE and its derivative the Syndrome Check List are useful instruments for systematic collection of clinical data, and for this reason alone seem appropriate for a study of this kind.

The principal findings are as follows. The post-ictal psychoses are defined by their temporal relationship to seizure activity. Psychosis usually occurred after an exacerbation in seizure frequency or intensity and appeared after a lucid interval. The episodes were characterised by pleomorphic psychotic phenomena occurring either in clear or in clouded consciousness and dominated by marked and varied mood changes. Spontaneous resolution was usual, but with a tendency to recur.

We were unable to confirm certain previously reported observations. Post-ictal psychotic states are said usually to occur against a background of clouded consciousness (Lishman, 1978), but only five of 14 cases reported here provided evidence of confusion sustained throughout the episode, while three more showed no confusional features at any time. Perhaps repeated testing of patients previously reported would have identified a proportion who resumed clear consciousness while still psychotic, as

occurred in four of this series. The presence or absence of confusion was confirmed during mental state examination by one of the authors in five of the cases. The medical notes confirmed that this symptom was repeatedly sought in each of the remaining nine, and verbatim responses to questions of orientation were recorded. Organically determined states may occur in the absence of confusion according to Cutting (1980), who identified 74 patients with psychosis and cerebral dysfunction: 38% were in clear consciousness at the time of assessment.

Previously reported work (Dongier, 1959) has observed an association between post-ictal psychosis and generalised epilepsy. Although the majority of patients in this series experienced generalised seizures, in most cases this was due to secondary generalisation from complex partial seizures. It has also been suggested that post-ictal psychotic episodes may remit spontaneously following the occurrence of further seizure activity, and therefore ECT could be beneficial in refractory states (Toone, 1981). In this study, further seizure activity was associated with an exacerbation of psychotic symptoms in five out of six subjects.

Other clinical observations seem worthy of comment. Stuporose states are said to occur post-ictally: three patients presented in such a way. One was mute and catatonic, with waxy flexibility; intensive nursing care, including tube feeding, was necessary. Another, also admitted mute and not eating or drinking, recovered in three weeks. He voiced depressive ideas when he started talking and had a depressed affect. The third case of stupor lasted only a matter of days. Diagnostic phenomenology was never elicited.

All commonly used anticonvulsant drugs had been given to the patient group: no particular drug could be inculpated. Three of the seven patients had reduced serum folate levels. Reynolds (1967) postulated that folate deficiency might contribute to the development of a psychotic state in epileptic patients. Unfortunately, red cell folate assay, a more stable estimate of folate availability, was not carried out. However, the patient with the lowest level, 2.0 μg/l, had previously recorded a level of 1.1 μg/l when not psychotic (normal range 3–15 μg/l). The role of folate deficiency in the generation of post-ictal psychoses therefore remains unclear.

The distinction between inter-ictal and post-ictal psychosis is not usually problematical. Brief, spontaneously remitting psychotic episodes occurring in epileptic patients have been described by Landolt (1958), but these are exceedingly rare, and the relationship of psychotic episodes to preceding seizure activity distinguishes such episodes from post-ictal psychoses. The course of the chronic, inter-ictal psychosis is probably not greatly influenced by seizure activity, and spontaneous remission is unusual once the disorder is well established. In only two patients was the onset of psychosis more than 48 hours after the return of normal cerebral functioning following a transient, marked increase in seizure frequency. One had a lucid interval of six days (during which time he was reported to have increasingly odd behaviour), but his usual fit frequency was one seizure every month. Moreover, he suffered a seizure while still psychotic, markedly worsening his mental state, which ultimately recovered after three months. The other patient had a lucid interval of three days. Her usual fit frequency was one seizure every three months.

Post-ictal psychosis should be borne in mind as an unusual clinical entity that may occasionally be misdiagnosed and treated inappropriately. It is also of theoretical importance as it provides the possibility of an organic model for psychoses in general and for the inter-ictal psychoses in particular. A closer familiarity with its mode of presentation, phenomenology and clinical course may thus provide clues to aetiology of a far commoner group of disorders. It is admittedly not an entirely adequate model. First-rank Schniederian experiences are relatively uncommon, and many of the episodes tend towards phenomenological polymorphism rather than present with discrete, easily recognisable schizophrenic or manic–depressive symptoms. Even so, mental state phenomena occur that are identical to those which are found in, and are diagnostic of, the functional psychoses, and they occur frequently in clear consciousness. The post-ictal and inter-ictal psychoses also have points in common: the lack of family history of psychosis, the over-representation of partial complex seizures, the similar age of onset of both epilepsy and psychosis, and the observation that, in a minority of cases, post-ictal psychoses will progress to chronic inter-ictal states.

The observation that psychosis may occur more commonly in patients with epilepsy, especially temporal lobe epilepsy, and that it usually develops 10–15 years after epilepsy onset (Slater *et al*, 1963), has led some authorities to propose that a disturbance in limbic function may play a crucial role in the evolution of the functional psychoses in general (Bogerts *et al*, 1985), and particularly in the inter-ictal psychoses of epilepsy (Torrey & Peterson, 1974). A similar explanatory hypothesis may be advanced to account for the post-ictal psychotic syndrome. The increase in temporal lobe spike/sharp wave activity is certainly consistent with an active process rather

than neuronal exhaustion. An increase in slow wave activity, noted in the same number of patients, was associated with confusion, and this pattern may have more in common with the usually more brief periods of confusion occurring commonly after major generalised seizures.

Such a hypothesis raises as many questions as it answers. It is not at all clear why some form of as yet unspecified limbic malfunction should in one instance manifest itself as a chronic inter-ictal psychosis and in another as a post-ictal psychosis, nor how the latter may in some cases evolve towards the chronic condition. A lucid interval preceding the onset of psychosis was observed in 12 of the 14 cases, but its significance remains uncertain. Little is known of the effect of spontaneously occurring seizure activity in man on cerebral monoamine systems, but experimental seizures induced in animals may result in increased post-synaptic dopamine sensitivity (Modigh, 1975). It has not yet been possible to replicate these results in man. Whatever the mechanism, the presence of a delay in the onset of post-ictal psychosis does suggest the possibility of an indirect effect involving one or more neurotransmitter systems.

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References

BETTS, T. A. (1974) A follow-up of a cohort of English patients with epilepsy admitted to psychiatric care in an English city. In *Epilepsy: Proceedings of the Hans Berger Centenary Symposium* (eds P. Harris & C. Mawdsley). Edinburgh: Churchill Livingstone.

BOGERTS, B., MEERTZ, E. & SCHONFELDT-BAUSCH, R. (1985) Basal ganglia and limbic system pathology in schizophrenia. *Archives of General Psychiatry*, **42**, 784–791.

CLARK, R. A. & LESKO, J. M. (1939) Psychoses associated with epilepsy. *American Journal of Psychiatry*, **96**, 595–607.

CUTTING, J. (1980) Physical illness and psychosis. *British Journal of Psychiatry*, **136**, 109–119.

DONGIER, S. (1959) Statistical study of clinical and electroencephalographic manifestations of 536 psychotic episodes occurring in 516 epileptics between clinical seizures. *Epilepsia*, **1**, 117–142.

ESQUIROL, E. (1838) *Maladies Mentales*. Paris: J.-B. Balliere.

GASTAUT, H. (1970) Clinical and electroencephalographical classification of epileptic seizures. *Epilepsia*, **11**, 102–113.

GUNN, J. & FENTON, G. W. (1969) Epilepsy in prisons: a diagnostic survey. *British Medical Journal*, *iv*, 326–328.

JACKSON, J. H. (1875) On temporary mental disorders after epileptic paroxysms. *West Riding Lunatic Asylum Medical Reports*, **5**, 105–129.

LANDOLT, H. (1958) Serial electroencephalographic investigations during psychotic episodes in epileptic patients and during schizophrenic attacks. In *Lectures in Epilepsy* (ed. A. M. Lorentz de Haas). Amsterdam: Elsevier.

LEVIN, S. (1952) Epileptic clouded states. *Journal of Nervous and Mental Disease*, **116**, 214–225.

LISHMAN, W. A. (1978) *Organic Psychiatry*. Oxford: Blackwell Scientific Publications.

MODIGH, K. (1975) Electroconvulsive shock and postsynaptic catacholamine effects; increased psychomotor stimulant action of apomorphine and clonidine in reserpine pretreated mice by ECS. *Journal of Neural Transmission*, **36**, 19–32.

PEREZ, M. M. & TRIMBLE, M. R. (1980) Epileptic psychosis: diagnostic comparison with process schizophrenia. *British Journal of Psychiatry*, **137**, 245–249.

REYNOLDS, E. H. (1967) Schizophrenia-like psychoses of epilepsy and disturbances of folate and B12 metabolism. *British Journal of Psychiatry*, **113**, 911–919.

SLATER, E., BEARD, A. W. & CLITHEROE, E. (1963) The schizophrenia-like psychoses of epilepsy. *British Journal of Psychiatry*, **109**, 95–105.

TOONE, B. K. (1981) *Epilepsy and Psychiatry* (eds E. H. Reynolds & M. R. Trimble). London: Churchill Livingstone.

——, GARRALDA, 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.

TORREY, E. F. & PETERSON, M. R. (1974) Schizophrenia and the limbic system. *The Lancet*, *ii*, 942–946.

WING, J. K. (1983) Use and misuse of the PSE. *British Journal of Psychiatry*, **143**, 111–117.

——, COOPER, J. E. & SARTORIUS, N. (1974) *The Measurement and Classification of Psychiatric Symptoms*. London: Cambridge University Press.

WORLD HEALTH ORGANIZATION (1978) *Mental Disorders: Glossary and Guide to their Classification in Accordance with the Ninth Revision of the International Classification of Diseases*. Geneva: WHO.

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