Some mental symptoms in neurology

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Summary
Cases are presented in which cerebral disease was associated with schizophrenic-like or manic-depressive-like psychoses, anxiety or obsessive states i.e. psychiatric disorders usually classed as 'functional'.

Evidence is presented to suggest that this association is not due to chance.

The cerebral structural changes are characterized either by the presence of 'focal' lesions, e.g. multiple sclerosis, or by neuronal loss e.g. Huntington's chorea. The probable mechanisms whereby these two types of structural changes effect psychiatric disturbances are discussed.

It is inferred that psychiatric disturbances will occur insofar as these cerebral structural changes exert an influence on the limbic systems.

The opening passage of Kraepelin's Clinical Psychiatry reads as follows: 'It is true that in the strictest terms, we cannot speak of the mind as becoming diseased... Indeed, from the medical point of view, it is disturbances in the physical foundations of mental life which should occupy most of our attention. But the incidents of such diseases are generally seen in the sphere of psychical events... Here we are not so much concerned with physical changes in size, shape, firmness and chemical composition, as with disturbances of comprehension, memory and judgment, illusions and hallucination, depression and morbid changes in the activity of the will' (Kraepelin, 1904). It is proposed to discuss these psychical events listed by Kraepelin, not in isolation but as appertaining to and manifestations of neurological diseases. Various 'psychical states' experienced in the course of what he called 'Uncinate Group of Fits' were first surveyed by Hughlings Jackson 90 years ago and this approach by way of epilepsy may still be the best point of departure for a discussion of mental symptoms in neurological diseases in general (Jackson, 1880). The Uncinate Group of Fits later became known as 'psychic-motor fits' because of the conjunction of behavioural and psychic disturbances. This term has now been superseded by 'temporal lobe epilepsy'. Jackson described a gamut of mental phenomena occurring in this form of epilepsy, phenomena which range from 'dreamy state' to 'emotion of terror'. Since Jackson's era these 'psychical states' have become so familiar as not to require detailed description here. They are of bewildering variety, differing from case to case, very complex—'voluminous' was the adjective used by Jackson—and so singular as to defy the patient's description. Dreamy state in Jackson's terminology comprised what we would call to-day depersonalization, de-realization and déja vu states. After stating that 'it is not uncommon for a patient to have the emotion of terror at the onset of his seizure', Jackson observed: 'I never remember hearing a patient mention the emotion of anger yet some patients say that at the onset of their seizures they feel they must attack someone, or have a hatred against some person present'. Jackson also described cases presenting disturbances of thought processes characterized by 'forced thinking' when thoughts which force themselves into consciousness cannot be described. Most if not all these 'psychical states' have been evoked by electrical stimulation of the cortex in conscious individuals (Penfield & Jasper, 1954).

Jackson also wrote at length concerning the post-epileptic disturbances of behaviour—known as automatism—following temporal lobe seizures and he frequently referred to his patient Dr Z. who left a detailed description of automatism as experienced by him. One such episode is of interest. 'Whilst the patient was undressing I felt the onset of a petit mal. I remember taking out my stethoscope and turning away a little to avoid conversation. Next thing I recollect is that I was sitting at the writing-table and speaking to another person. I gathered indirectly from a later conversation that I had made a physical examination, written “pneumonia of the left base”, and advised him to take to his bed at once. I re-examined him with some curiosity, and found my conscious diagnosis was the same as my unrememhered diagnosis had been' (Jackson, 1889). Recent observations on the physiological mechanism of automatism are of some interest. During the ele-
Electrical stimulation of the cortex in a conscious individual, contact with the immediate environment is well maintained and perceptual responses evoked by stimulation are remembered. In marked contrast are the cases in which the deeper structures in the anterior temporal region are electrically stimulated when depth electrodes are placed in the uncinate, amygdaloid or peri-amygdaloid regions. Stimulation of these structures may be followed by a widespread suppression of the electrical cortical activity. Judged by the failure to retain memory of events occurring during the period of cortical suppression, when the subject appears conscious, he must be deemed to have exhibited post-ictal automatism. Such cortical suppression has also been demonstrated during the period of automatism following a seizure provoked by overbreathing (Jasper, 1964).

As Dr Z.'s experience suggests, post-ictal automatism can be of long duration and mimic the syndrome of Transient Global Amnesia (Fisher & Adams, 1964) and the following case illustrates this.

Case 1

J.P., aged 41. Two weeks before—according to his wife, he brought her up a cup of tea before leaving the house as usual at about 6.30 a.m. Later at about 3.30 his daughter received a phone call. Patient was crying; he claimed that he had disgraced himself and expressed doubt whether he would be forgiven by his wife. He suggested that she should meet him near his home where he arrived about 1½ hr later. He appeared exhausted and confused. He said to his wife: ‘You don’t want me back’. He retired immediately after returning home. On the following day he had no recollection of the events of the previous day after leaving his house in the morning. Since then he has lost consciousness on about half a dozen occasions. Each time the period of unconsciousness did not exceed ‘a few seconds’. These episodes occurred either shortly after waking during the night to void urine, or on waking at the normal times in the mornings. Neurological examination and neurological investigations were negative. EEG—theta activity in both temporal regions. He became free from symptoms shortly after commencing anticonvulsant treatment.

Another aspect of temporal lobe epilepsy must now be considered. On the basis of his extensive EEG material, Frederic Gibbs (1951) concluded that non-ictal psychiatric disturbances are more than three times commoner in cases with focal seizure activity in the temporal lobe than in cases with a focus in any other cortical area. He found that though such non-ictal psychiatric disorder is more or less constant for a given patient, no single psychiatric syndrome was encountered. ‘The patient may be paranoid, depressed, catatonic or hysterical. The psychiatric symptoms are a real and important part of the clinical disorder and may overshadow the epileptic manifestations to such a degree that the patient is diagnosed on a symptomatic basis as schizophrenic or depressive’. Gibbs then goes on to say ‘when focal discharging lesions occur in parietal, occipital and frontal lobes the symptomatology is largely neurologic; if the disorder is in one or both temporal lobes the symptomatology is largely psychiatric’.

In 1963 Slater & Beard gave an account of their investigations of sixty-nine patients presenting schizophrenic-like psychoses and epilepsy of predominantly temporal lobe origin (Slater, Beard & Glithero, 1963). These authors attempted at first to subdivide the psychiatric syndromes presented by these patients into four groups but found later that they had to abandon this subdivision. ‘On further investigation the distinction between these groups became somewhat blurred . . . the difference between the groups being quantitative rather than qualitative’. On the other hand they concluded that ‘It would not be possible to diagnose these patients, on psychological symptomatology alone, as suffering from anything other than schizophrenic psychosis’. They added that by and large this group differed in many respects from ‘typical’ schizophrenic patients. Not the least was the relatively high proportion in this group of personality changes indicative of brain lesions. However, of particular interest is that the premorbid personality of their patients was of normal type and that in the great majority of them it was not possible to relate the onset of mental illness to any change in the quality or frequency of the epileptic seizures. In the main these conclusions are similar to those reached by Flor-Henry who carried out a controlled investigation comparing a population of fifty temporal lobe epileptics with psychotic episodes, with a control group of fifty randomly selected temporal lobe epileptics who had never experienced psychotic disturbances (Flor-Henry, 1969). Using traditional Kraepelinian diagnostic criteria, he divided his epileptic psychosis material into schizophrenic, schizo-affective, manic depressive and confusional groups. It is evident from this series, and to some extent the series of Slater et al., that there is much to support the position adopted earlier by Gibbs with respect to the variety of psychotic states encountered in temporal lobe epilepsy.

The following are a few typical recent personal cases which also indicate the variety of psychotic states encountered:

Case 2

F.B. aged 39. (a) Since early childhood subject to epileptic seizures, probably of grand and petit mal
type. According to patient long remissions—some as long as 2 years—have occurred frequently. Available recent history certainly confirms this statement.

(b) Clinical features: head slapping, ideas about electronic machines, influences involving neighbours, bizarre mannerisms, delusive thought of paranoid nature. (c) EEG—occasional slow waves (within delta band) in posterior temporal areas.

Case 3

D.M. aged 44. Pilot in R.A.F. 1943–1948—rejoined R.A.F. 1955. ‘Tensions’ and other psychiatric disturbances caused his discharge in 1957. 1960—‘On top of the world’—working as personal pilot until 1961 when he had the first of his seizures, which are always immediately preceded by ‘a spinning sensation and feeling as though something was going to happen in the head’. Generalized epileptic seizures were observed on a number of occasions. 1969—admitted to Hospital untidy in dress, clothes in rags, weary in manner. Says he ‘has come to a standstill’—very tense. Talks relevantly and coherently. Obsession for writing things down—‘virtually everything I see, name of a book or cigarette or anything; obsession about checking things e.g., switches, doors etc.’ EEG theta activity seen bilaterally in both temporal regions.

Case 4

F.G. aged 53. Broadly speaking the symptomatology is of twofold nature:

(a) Epileptic seizures. The first recorded seizure occurred in his sleep, 8 years ago, at age 45. Patient stated his father was also subject to seizures. Investigations (March 1961) included examination of CSF; X-rays of skull and chest and air encephalogram—all negative. Air encephalogram repeated in 1968—no significant degree of cerebral atrophy. EEG—Numerous bursts of high voltage 2–3 c/s activity, usually seen over both frontal areas. Bilateral carotid angiograms normal.

(b) Psychiatric disturbances. The chief disturbance being lability of emotions, with quick rages and spasms of inexplicable crying.

1969—Complained of being ‘very downhearted’. He attributed this mood in the main to two causes. (1) The presence of a black coffin in the dormitory—‘I am unhappy and broken-hearted because people are doing silly things to me—they should not do it to an innocent man etc.’ He also mentioned ‘two white patches on the ceiling’ which he also regarded as of abstruse symbolic significance. (2) The appropriation by his mother of his rightful inheritance which he alleges was left to him by his uncle because he was the first-born. He had been dispossessed of a vast sum of money. His mother would never allow him to touch her purse which presumably contained some of the plunder.

Despite long periods during which he was not receiving any anti-convulsants, he has had relatively few seizures since 1961.

Since the majority of individuals with epileptic activity in one or both temporal lobes are free from psychiatric manifestations it is important in the first place to consider whether the association of these two components might be due to chance. Simple comparisons of the incidence of psychiatric disorders in temporal and non-temporal lobe epilepsies have not revealed any statistically significant difference (Small, Small & Hayden, 1966). In a group of schizophrenics the frequency of antecedent epilepsy was also no greater than could be accounted for by chance, yet it must be considered significant that seven out of eight cases in this series had clinical or EEG evidence of temporal lobe lesions (Bartlet, 1957). Much firmer evidence that temporal epilepsy and inter-ictal psychiatric abnormalities are related is provided by the result of temporal lobectomies. A number of studies of the results of anterior temporal lobectomy reveal that patients, whose psycho-social functioning was adequate before the onset of epilepsy and have shewn seizure relief as a result of lobectomy, have also shewn a corresponding improvement in the concomitant psychiatric disorder (Bailey, 1954; Hill et al., 1957; Falconer & Serafetinides, 1963). Similar abolition of seizures and severe behaviour disorder was observed in a proportion of cerebral hemispherectomies for infantile hemiplegia (Wilson, 1970). A great variety of focal structural abnormalities have been found in the excised temporal lobes. These range from mesial temporal sclerosis to hamartoma and neoplasms.

If the inferred aetiological nexus between epilepsy and inter-ictal psychotic state is accepted, the possible nature of this relationship must now be considered. Pond has suggested that seizures themselves could be adequate in causing psychosis—‘Years of attacks of clouded consciousness might lead to a confusion of reality and autistic thinking and experience’ (Pond & Symonds, 1962). On this theory mental seizure phenomena having become incorporated in the psyche may then exert some psycho-dynamic influences which in turn engender psychotic disturbances. The absence of any significant difference in the epileptic variables between the ‘non-psychotic’ and ‘psychotic’ groups of temporal lobe epilepsy would argue against this.

In essence the rationale of temporal lobectomy is the removal of a focal structural abnormality (‘epileptogenic lesion’) within the temporal lobe. If such surgical procedure is followed by improvement in both epilepsy and the psychiatric state, it is difficult
to escape the conclusion that both these clinical components have a common anatomical substratum. An epileptogenic lesion is a structural lesion which, by virtue of its presence induces in the ganglion cells in its proximity, an abnormal chemico-physiological state which results in neuronal disturbances. Under certain neuro-physiological conditions, hypersynchrony may ensue, and epileptic seizure may result. Microelectrode studies have shewn that an epileptic cell may fire at excessively high rates of discharge for brief periods of time, becoming refractory to excitation between bursts, and that such convulsive activity may remain restricted to a single neurone (Baumgartner, 1954). Such firing may result in disorganization of the patterns of neuronal integrative activity. These electro-physiological abnormalities are, of course, features of 'epileptogenic' foci in general yet only when located within the temporal lobes do such foci become associated in any frequency with inter-ictal psychiatric disturbances.

Mention was already made of widespread cortical suppression which may occur during an after-discharge from the peri-amygdaloid region. This observation may be taken as evidence that a local discharge in the peri-amygdaloid region may fire into subcortical zones related to both hemispheres. For the same reason a unilateral temporal lobe lesion may give rise to bilateral EEG abnormalities (Falconer & Kennedy, 1961). The amygdaloid grey matter can thus be considered as part of a diffuse projection system, similar to the brain-stem reticular formation, and that it is thus capable of exerting widespread regulatory effects on many other cerebral regions. It can be supposed, therefore, that sub-ictal disturbances occurring in an epileptogenic lesion in relation to the structures of which the limbic system is composed, may so alter the normal vastly complex and highly integrated pattern of firing in other remote neuronal systems, as to cause distortions in sensory and perceptual processes and thus lead to disturbances of mood, behaviour and cognition. Some supporting evidence was provided by Ervin et al (1969). By means of stereotactically implanted electrodes and remote recording and stimulation apparatus, they studied two patients with temporal lobe lesions. They observed focal electrical changes in the limbic system in response to appropriate environmental stimuli. These electrical changes occurred as a prelude to an abrupt alteration in behaviour characterized by rage. When considering Slater & Beard's material Symonds reached a similar opinion to that proposed here. He summarized his view as follows... '... it is not loss of neurones in the temporal lobe that is responsible for the psychosis, but the disorderly activity of those that remain' (Symonds, 1962). The following case illustrates that the effects of this 'disorderly activity' of the neurones can be relieved not only by temporal lobectomy but also by anterior cingulotomy. Le Beau has claimed repeatedly that the removal of the anterior cingulate cortex is 'especially useful' in relieving psychic states when the latter occur in association with epilepsy (Le Beau, 1954). 'Good results' defined as 'complete or nearly complete cure' were obtained by him in seven out of nine cases falling into this category. The following case illustrates the dramatic abatement of psychiatric symptoms while the patient remained under constant environmental conditions, without, however, any appreciable curtailment of seizures.

**Case 5**

P.B. aged 30. Generalized seizures commenced at the age of 11 following an accident in which her leg was injured while wheeling a bicycle. She did reasonably well at school but at the age of 15—fits increased in frequency and she became more and more intractable. At the age of 16 she was admitted after a suicidal bid with aspirin. At first she was regarded as a rejected adolescent who was reacting aggressively but gradually she became more intransigent in her behaviour. 1959—'fights with patients'. In the same year she was transferred to an epileptic colony, but sent back shortly afterwards because of her uncontrollable behaviour. IQ 95. In 1963 state of agitated aggressiveness reached a climax. She was described as subject to berserk rages and outbursts of profane screams; five nurses were required to control her. At the age of 24 bilateral cingulotomy. Following operation too obstreperous to be nursed in a general ward. By degrees her behaviour has improved remarkably though she still continues to have generalized seizures. The Sister of the ward described her demeanour during the past few years as quiet, helpful and staid.

In several publications attention has been drawn to a group of patients showing severe psychiatric disturbances and evidence of focally abnormal EEG disturbances in the temporal lobes but without overt seizures (Gibbs, 1951; Hill, 1952; Ervin, Epstein & King, 1955). Some of these cases must be considered to fall into the group of temporal lobe epilepsy with psychiatric manifestations, and the absence of overt epilepsy must be ascribed to the operation of factors which suppress seizures. There is ample neuro-physiological evidence that there are many agencies which determine the threshold for convulsive reactivity of an epileptic focus. There is some clinical evidence that the operation of such agencies which reduce synchronization may actually potentiate the mechanism whereby psychiatric disturbances are engendered by an epileptogenic focus within the temporal lobe (Glaser, 1964). A report was recently published of a man who lost his violent tendencies
following temporal lobectomy performed on grounds of persistent violent criminality and relevant EEG abnormality (Gibson, 1970). The following brief case summary illustrates well this syndrome of psychosis and structural lesion within the temporal lobe, without the occurrence of seizures.

Case 6
Woman with 5 years' history of typical paranoid state with persecutory ideas, chiefly directed against her husband whom she repeatedly threatened with a knife. During this period she suffered from headaches. At the time of her admission into hospital she might have been described as a typical paranoid dementia excepting for physical signs of an intracranial tumour which was later found to involve the temporal lobe (Brander & Golla, 1931).

At the time of admission of this patient the diagnosis was not in doubt because of the expanding and relatively massive nature of the brain lesion. However, it is not the dimension of the structural lesion within the brain but the precise topography which determines whether psychiatric disturbances occur. There are a number of neurological pathologies which are characterized by the scattered, potentially epileptogenic lesions—among these syphilis and multiple sclerosis are pre-eminent. If the lesion characteristic of these diseases chances to be sited in the temporal lobe, it may in this location engender personality disorders with or without epileptic seizures. Bostroem (1930) found that 3% of cases of G.P.I. presented a psychiatric picture akin to that of schizophrenia. The incidence of psychotic disorders in multiple sclerosis is higher than is generally recognized but only two well-documented accounts, one with autopsy confirmation—have appeared in the literature (Schmalzbach, 1954; Parker, 1956). The following are a few similar cases encountered recently in psychiatric hospitals.

Case 7
G.K. aged 35. According to his mother, patient visited U.K. about 3 years ago, and stayed with her for about 18 months. During this period she noticed that he had a tremor which became manifest when holding a cup and saucer; she attributed this to 'tension' though he did not exhibit at the time any neurotic symptoms. After his return to Australia she continued to receive letters from him which were 'normal' in all respects. About 4 months ago she received news from her daughter in Australia that he 'suddenly cracked up'. When he returned to U.K. he was very agitated and mentally disturbed. 'He chewed anything he could lay his hands on'. On admission to hospital—restless and agitated. 'I have no life at all; I am in a trance. I think God made me a devil. I've ruined the world'. There was little change in his mental condition following electro-convulsion therapy.

CNS—all tendon jerks greatly exaggerated; no nystagmus; extensor plantar responses. Minimal action tremor when performing finger-nose-finger test, especially with left upper limb. Right leg swings out when walking. CSF—Lange curve of paretic type.

Case 8
Mrs. J.T. aged 44. According to information contained in her doctor's letter, patient has for some years been suffering from 'schizophrenia'. Patient stated that 10 years ago 'when working in London' she was for a time troubled with double vision which made her fearful of the traffic. More recently she complained of urgency of micturition. She showed 'spastic' paresis of her legs—right more than left, with extensor plantar responses. Her psychiatric symptoms were in the main characterized by severe mental depression. This remitted dramatically 4 months later. She has a confused and vague recollection of the events which occurred during the period when the psychiatric symptoms were prominent. Her neurological signs remain as before.

Case 9
H.B. aged 49. Following admission to a psychiatric hospital 8 years ago for severe mental depression, she developed persistent paraesthesiae in the lower half of the body and legs. These symptoms disappeared after some weeks. Her mental symptoms responded to ECT. A progressive spastic paresis of the lower limbs and intermittent cerebellar disturbances first appeared 18 months later. CSF—Lange reaction of paretic type.

Huntington's chorea
While the small ganglion cells of the striatum show the most severe involvement, generalized neuronal loss is the outstanding histopathological feature in Huntington's chorea (Biggart, 1961). Though eventually becoming widespread, the neuronal degeneration in the early stages of the disease is localized. The wide variations of the topography of maximal neuronal loss account for the variability of early clinical presentations, even among members of the same family. Depletion of ganglion cells with astrocytosis is one of the common findings in the cortex which may be affected in a patchy manner. If this depopulation of neurone affects the hippocampal region, the neurones of which are in relation to the limbic system, the pattern of afferent input into this system must be expected to show a significant change, both quantitatively and qualitatively. An analogy is provided by Parkinsonism. While this disease itself is due to loss of neurones which form
the ‘extrapyramidal’ motor system, the symptoms and signs are manifestations of an imbalance between the reduced activity of ‘extrapyramidal’ motor system and the normal operation of the ‘cerebellar’ motor system (Carman, 1968). Attenuation of the latter by operation may correct the Parkinsonism. It would be reasonable to conclude that loss of neurones in the relevant cortical areas may result in a variety of psychotic disturbances in consequence of a neuronal imbalance. The incidence of schizophrenic-like and other mental disturbances in the early stages of the disease is relatively common in Huntingdon’s chorea. Heathfield reported that in ‘twenty-six out of eighty patients, psychosis occurred in the early stages of the disease’ and that depression and schizophrenia accounted for nineteen of these cases (Heathfield, 1967). Oliver found similarly that whereas ninety-four patients exhibited psychiatric symptoms ‘in the prodromal stages of the disease’, only in fifteen were these symptoms characteristic of dementia (Oliver, 1970). In the following case of Huntington’s chorea serious mental disablement resulted from an obsessional state.

Case 10
R.B. aged 51. Patient’s wife attributes the onset of her husband’s present illness to the ‘shock of finding his mother dead as a result of a suicide—he felt ashamed and this played on his mind’. In the course of his work the patient spent most of his time working on quays and in ships and he was recently obliged to resign mainly on grounds of safety because of his liability to frequent falls. She mentioned that even at home he ‘seems to topple over when he turns sharply’. She had not observed any personality change until about 6 months ago when he first developed obsessional tendencies manifesting mainly as anxious preoccupation concerning locking up at night and turning off gas taps. In recent months attempts not to comply with this compulsion has led to increasing distress. Patient, who is well aware of these impulses, ascribed them to feeling of uneasiness, ‘uncertainty about the future’ and loss of sense of security. On a recent occasion he became depressed and tearful. ‘Look what I have done to the family’ he told his wife. For some time past she has been aware that the patient has become unduly ‘fidgety’ as a result of slight choreiform movements. Both his brothers, now dead, suffered from Huntington’s chorea.

Discussion
Three adjectives, ‘dull, confused and amnesic’ were used by Golla (1931) to describe the mental symptomatology of brain tumours. These adjectives, which could be applied with equal propriety to patients with many other structural diseases of the brain, have however little relevance to the patients presented in this clinical study. In these patients structural diseases of the brain—presumed or proved—were associated mainly with disorders of thought and affect. The mental symptomatology was therefore of a type commonly encountered in diseases in which evidence of structural brain pathology is as a rule lacking. These patients were therefore classified accordingly as suffering from affective disorders, from schizophrenia, obsessional states, irritability, etc.

The conclusion to be drawn from this clinical study is that the topography of the structural brain lesion is the determining factor in influencing the behavioural disturbances which will result. This observation does not, however, imply the affirmation that a given disturbance of behaviour necessarily presupposes the presence of a structural lesion in a specific situation. An analogy with this concept is provided by the syndrome of trigeminal neuralgia. This syndrome, which as a rule is not associated with any manifest structural lesion, may on rare occasions be engendered by multiple sclerosis (Harris, 1927; Rushton & Olafson, 1965). It may perhaps be opportune here to repeat the salutary caution expressed by Golla many years ago against ‘the hope which we all find so difficult to eradicate, however much we publicly deny it, that the inconceivable complexity of mental life may be resolved into a few static mechanisms (Golla, 1931).

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References
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GOLLA, F.L. (1931) See Brander supra.


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