initially to a psychiatric hospital in 1925 and transferred in 1937 to a mental handicap home, which was later converted to a mental handicap hospital. She is still in hospital and has not been treated for her mood disorder except for two unsuccessful attempts in 1984 and 1988.

Case report: Miss A was initially admitted to a local psychiatric hospital on 9 July 1925, at the age of 15 years, under Section 16 of the Lunacy Act 1890. According to the notes, on presentation she was "extremely voluble, runs from one subject to another in an excitable and incoherent manner, talks to herself incessantly".

There is no information of her early history. However, her mother stated that from the age of eight she had been over-talkative, highly excitable, anxious, and hysterical. According to her mother, she used to roam the country lanes and would not keep away from men, laughed hysterically and talked all day. Miss A attended school until the age of 14 years and was good at reading and writing. Following admission, she was diagnosed as suffering from dementia praecox. The diagnosis was made on the following observations: "Eccentric, irrational in habits and manner. Chatters incessantly and wanders from one subject to another in conversation. Hears whispering voices and answers them and becomes excited and quarrelsome at times." The notes refer to her intelligence and state that it is much below the average standard for her years. It is difficult to ascertain from the notes how this statement was concluded. Signs and symptoms of either organic brain damage or other metabolic, chromosomal, and genetic disorders were not present.

According to the notes, during her stay in the psychiatric hospital she was treated with 'Mint Menth Pip' occasionally. Over all these years she had not received specific treatment for her mood disorders. Recent attempts to treat her hypomanic episodes with haloperidol and thioridazine were unsuccessful, as she developed idiosyncratic reactions: joint pain, running nose, hyperthermia, anergy, malaise and raised ESR. These symptoms disappeared on the discontinuation of the antipsychotic medication. Psychological testing in July 1973 established a Full Scale IQ of 54 on the Wechsler Adult Intelligence Scale, with a reading age of approximately 13 years. The conclusion was that at her age, after years of institutionalisation, the formal IQ was irrelevant. Her memory is not impaired.

It is rather sad that this lady should have spent all her life in an institution due to a different philosophy of care at that period. In all these years there has been no deterioration in her very pleasant personality. She is still a very friendly and amiable person.

This case highlights the fact that there are still institutions with patients who could have been discharged to the community several years ago, and if proper diagnostic criteria were applied at any given time during these years she would not have been in a mental handicap institution. Therefore, it is important for the future management of such patients not only to use proper diagnostic criteria but also to take an holistic approach when assessing patients.

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Hypomania Following Increased Epileptic Activity

SIR: I report two cases of hypomanic behaviour which followed a rapid increase in the frequency of epileptic discharges in two known temporal lobe epileptics. These findings are in keeping with those of Barczac *et al* (*Journal*, January 1988, **152**, 137–139), who noted an increase in epileptic discharges to be associated with hypomanic activity.

Case reports: (i) Mr M.N. was admitted via the Emergency Room, following an episode of hypomania. He attempted to donate his body to the State in a police station at 04.00 h. Mental state examination revealed disorientation, flight of ideas, pressure of speech, and paranoid delusions regarding his wife's sexual behaviour. Collateral history from his wife revealed that a change in maintenance therapy had been made, and four generalised seizures had occurred over a period of 3 days. The hypomanic behaviour occurred some 12 hours following the last seizure. Mr M. responded to high dose benzodiazepines administered orally. Carbamazepine therapy has been instituted, and his epilepsy has since remained under control.

(ii) Mr M.H. was admitted via the Emergency Room, following an episode of unusual behaviour in which he proposed to his brother's wife and attempted to pluck hot coals from an open fire. He displayed lability of mood, flights of ideas and pressure of speech at interview, and admitted to being able to see religious images on the curtains of his cubicle, which he found most enthralling. A collateral history from his brother revealed that he had been drinking heavily, and had failed to take his medication for some days. He had developed three generalised seizures in rapid succession, and the episode of hypomania had occurred approximately 8 hours later. Mr H. responded well to sedation with oral benzodiazepines and returned to his previous anticonvulsant medication. He has remained well for the 5 months since discharge. His alcohol intake has remained high, however.

Both of these patients have been diagnosed as having complex partial seizures from early childhood, one from birth, and the other as a result of a road

accident at age 10. Both patients responded well to benzodiazepines. Compliance had been a problem in both cases. Previous investigation had revealed lesions in the right temporal lobes in both cases. These are two more cases of hypomania in association with uncontrolled epileptic activity in the right temporal lobe. In both cases, increased epileptic activity was followed by a post-ictal mood change.

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Spectrum Concept of Neuroleptic Malignant Syndrome

SIR: The report by Adityanjee *et al* (*Journal*, July 1988, **153**, 107–111) and their discussion of the concept and diagnosis of neuroleptic malignant syndrome (NMS) brought to mind my recent first encounter with this reaction, outlined in the following case report.

Case report: A 15-year-old girl was admitted to our unit in an excited, apparently psychotic state, diagnosed initially as hypomania. She was treated initially with chlorpromazine, which failed to control her. Haloperidol was substituted, and she made a rapid recovery and was discharged without medication. After a short while, the original symptoms re-emerged and she was immediately recommenced on haloperidol as an out-patient. About 5 days later she was referred for admission, with a history of dysphagia, oral thrush, and withdrawal. On this occasion she was drowsy and mute. There was marked 'lead pipe' muscular rigidity, but a tremor accompanied any attempted voluntary movement. She appeared flushed, but had only a mild pyrexia of 37.5°C. Blood pressure was normal, but the pulse rate was raised, both fluctuating significantly during initial observation. Non-response to intramuscular procyclidine raised our suspicion of NMS. All medication was discontinued, and she was transferred for in-patient medical care. The girl recovered without active treatment within ten days. Investigations revealed no leucocytosis or other abnormality, although serum creatine phosphotinase (CPK) was not assessed.

Days after this recovery, a relapse of the original illness occurred and was successfully treated with a brief course of ECT. So far the patient has remained well on lithium.

We made the diagnosis of NMS satisfying the suggested criteria of Kellam (1987): muscular rigidity, altered consciousness, and 'vegetative dysfunction', including pyrexia of $\geqslant 37.5$°C, changes in pulse, blood pressure, etc. However, this case, in common with two of the three cases described by Dr Adityanjee's group, fails to meet their suggested minimum requirements for the diagnosis, which include a pyrexia of $\geqslant 39$°C plus at least two of the following: tachycardia, rapid respirations, blood pressure fluctuations, excessive sweating, and urinary incontinence. Nevertheless, Dr Adityanjee *et al* refer to many other reports of idiosyncratic reactions to neuroleptics which comprise some, if not all, of the above criteria. Clearly, neuroleptic drugs are capable of producing a variety of unwanted effects, the 'pure' syndrome being by no means always the rule. In addition, NMS is clinically indistinguishable from lethal catatonia, described in psychosis, and from malignant hyperthermia, seen in response to some anaesthetic agents, and is not specific to the use of neuroleptic drugs (Kellam, 1987; Abbott & Loizou, 1986).

I accept Dr Adityanjee *et al's* argument for clinically separating NMS from the commonly encountered extrapyramidal side-effects because of the important implications for treatment, but I do not believe that this is a justification for adopting a narrow concept of NMS, as they suggest. I doubt the validity of regarding NMS as a distinct clinical entity, and suggest that all that is required is that clinicians are aware that the signs which comprise the syndrome can occur, so that early detection will lead to discontinuation of the drug and initiation of appropriate treatment with, hopefully, the avoidance of fatality.

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Psychotherapy of the Elderly

SIR: I am disappointed by the dearth of psychiatric literature written and researched in Britain on the subject of psychotherapy of the elderly. Most of the literature has originated from American psychodynamically oriented psychiatrists and psychologists. Freud (1905) wrote: "The age of patients has this much importance in determining their fitness for psychoanalytic treatment, that, on the one hand, near or above the age of fifty the elasticity of the mental processes, on which the treatment depends, is as a rule lacking – old people are no longer educable – and, on the other hand, the mass of material to be dealt with