'PSEUDODEMENTIA' A MISLEADING AND ILLOGICAL CONCEPT

DEAR SIR,

A 62-year-old man was referred for assessment of cognitive impairment amounting to dementia. He also had moderate depression which was treated and he made a full recovery, only to become depressed again a year later with a clinical picture very similar to that seen previously. He recovered once more with treatment.

The conventional diagnosis of this state would be 'pseudodementia' i.e. a clinical picture of dementia existing without discernible organic brain disease. However, this diagnosis raises several points and it can be argued that 'pseudodementia' is a misleading and illogical term.

Modern definitions of dementia (e.g. Lishman, 1978) are in agreement about the basis for the diagnosis: it is an acquired global impairment of intellect, memory and personality, but without impairment of consciousness. Dementia exists when the criteria mentioned in the definition are satisfied. There is no presupposition of aetiology. Yet, a diagnosis of 'pseudodementia' would imply that confused aetiological and descriptive considerations existed in the clinician's mind during assessment. Logically, considerations of aetiology and pathology must follow the descriptive clinical diagnosis.

What 'functional' psychiatric illness really is must be considered moot at the best of times in the light of neurobiological data which continue to accumulate about the schizophrenias as well as depression. It need hardly be said that a satisfactory evaluation of the presence or absence of all brain pathology is unlikely to be made in the consulting room.

Depression may cause a clinical picture of dementia and also be a feature of the syndrome. But the investigation of a demented patient does not depend on ancillary symptomatology. Every demented patient deserves vigorous investigations and preoccupation with 'pseudodementia' or 'functional' dementia may only encourage delay in the investigation and management of other potentially treatable causes.

There is widespread feeling that dementia is malignant, terminal and incurable. A proper appreciation of the fact that a substantial minority (up to 20 per cent of cases in representative series) of those presenting with a picture of dementia in accordance with the modern definition can be treated with some expectation of success is a considerable morale booster.

For these reasons it is felt a case can be made for dropping the term 'pseudodementia'.

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Reference

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NEUROSIS AND PERSONALITY DISORDER

DEAR SIR,

Tyrer *et al's* article, focussing on the relatioships between neuroses and personality disorder is to be welcomed (*Journal,* April 1983, **142,** 404–8). The authors are highlighting a frequent clincial problem fraught with research problems. Their attempts to define and demarcate such an area with the Personality Assessment Schedule (PAS) deserves plaudit.

However, I take exception to their interpretation of the General System Theory (GST) notion that as "neurosis and personality disorders constitute different levels of disorder, they should be regarded as separate". Indeed, their quoted reference, Gray, Duhl and Rizzo, 1969, emphasize that 'personality' should NOT be compartmentalized. Further, that "the more one examines the mental reactions of neurotics, the more one finds that there are no sharp lines of demarcation between the various types. Even the psychopathic personality is not always sharply demarcated from the neurotic" (p. 306).

I feel it is important to refer to the GST literature in discussing mental illness as Tyrer *et al* do. But equally important is to quote what the authors have written.

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NEUROLEPTIC MALIGNANT SYNDROME

DEAR SIR,

With reference to a recent letter from Dr Cremona-Barbaro (*Journal,* January 1983, **142,** 98–9), reporting a case of neuroleptic-induced catatonia, I would like to bring to readers' attention a related but little-known complication of neuroleptic drugs. This is the Neuroleptic Malignant Syndrome (NMS) which has been reported in the French and American literature (Delay *et al,* 1960; Smego and Durack, 1982; Caroff, 1980), but there has been only a single British case report (Allan and White, 1972) up to now.

The NMS has been reported in connection with all the major tranquillizers but haloperidol and depot fluphenazines are most commonly implicated. Characteristic features of the NMS have been described in some case reports of patients receiving combined lithium and haloperidol (e.g. Cohen and Cohen,

1974). The syndrome is characterized by severe extrapyramidal symptoms of muscular rigidity and akinesia, with hyperpyrexia, fluctuation in the level of consciousness and autonomic dysfunction, which includes tachycardia, labile blood pressure, hyperventilation, profuse diaphoresis, sialorrhoea, dysarthria and dysphagia. The patient may be alert, but mute, with other catatonic symptoms, progressing to stupor and coma in severe cases.

The syndrome may develop within hours of initial drug exposure, or after months of drug use, and occurs at therapeutic rather than toxic dosage. Once started, the syndrome develops rapidly over the next 24–72 hours, and if neuroleptic medication is not stopped the outcome may be fatal. A mortality rate of 20 per cent has been reported (Cardoff, 1980). Cardio-respiratory collapse is the usual mode of death, and there is no specific treatment, apart from supportive measures.

There are no specific or diagnostic laboratory findings in the NMS, but there may be a polymorphonuclear leucocytosis, abnormal liver function tests, and elevated serum CPK, probably caused by myonecrosis after prolonged skeletal muscle contraction (Smego and Durack, 1982). The EEG is generally normal, but may show non-specific slow activity. Examination of CSF, isotope brain scans and CT scans where performed have been normal (Caroff, 1980), and post mortem findings have been negative.

The disorder may be mis-diagnosed as encephalitis or other infectious diseases of the CNS. The NMS has been compared to acute lethal catatonia described by Stauder (1934) many years before the advent of neuroleptics, and to the syndrome of malignant hyperthermia associated with general anaesthetics. Heat stroke which may occur in patients receiving phenothiazines and butyrophenones can be distinguished from the NMS by the lack of muscular rigidity.

Although the pathogenesis of the NMS is unknown, it has been suggested that features of the syndrome can be explained by dopamine-receptor blockade in the basal ganglia and hypothalamus. It is not known why some individuals are susceptible to the NMS, but there have been some case reports of pre-existing organic brain disease (Meltzer, 1973) or physical exhaustion and dehydration (Itoh *et al*, 1977). It is interesting that some patients have been safely re-exposed to the same neuroleptic without recurrence of the syndrome.

Having recently seen a case with fatal consequences (Cope and Gregg, 1983), I hope that this brief summary of the condition will lead to more widespread recognition.

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MACROCYTOSIS AND DOWN'S SYNDROME

DEAR SIR,

We reported macrocytosis in 92 adult Down's syndrome patients (MCV 102.5±7.87 fl), the MCV having been calculated from separate red cell counts (Model D Coulter counter) and microhaematocrit estimations (Eastham and Jancar, 1970). No significant sex difference was found. Using a Coulter-S-Plus electronic cell counter, we have now found the MCV in 16 male and 17 female adult Down's syndrome patients to be 96.3±3.79 fl (1 SD), again with no sex difference, and with an RDW of 10.68±1.67. (The normal RDW of about 10 indicates no abnormal anisocytosis). These patients were not being treated with anticonvulsants and were not anaemic.

This latest result is significantly smaller than our earlier result (t test, P 0.001), and almost certainly reflects the change in technological method, but the mean value for the MCV is still above the accepted upper limit of the normal range (95 fl). The earlier method included centrifugation of red cells with associated plasma trapping, while the current method measures the MCV directly and eliminates the effects of plasma trapping. The explanation for macrocytosis in Down's syndrome is still unknown, but it has been shown that the red cell envelope in trisomy Down's