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Chronic Schizophrenia and Long-Term Hospitalisation

SIR: It is always useful for a psychiatrist interested in long-term patients to re-read *Institutionalism and Schizophrenia* (Wing & Brown, 1970), as Wing has reminded me (*Journal*, January 1987, **150**, 129–130). I had hitherto thought that to do so with an eye to the questions it raises was fully in tune with the authors' intentions.

I do not dispute the book's conclusion that the negative handicaps of patients with schizophrenia are influenced by their current social milieu. My letter (*Journal*, September 1986, **149**, 382) was prompted partly by concern that its evidence was given insufficient weight by Mathai & Gobinath (*Journal*, May 1986, **148**, 509–516). I raised the different question of whether or not the institutional environment has a *cumulative* effect on negative handicaps such that they worsen progressively in the long-term, contrary to the case outside hospital (Bleuler, 1978).

I find it difficult to accept Wing's assertation that no one pattern of relationship between negative handicaps and length of stay in hospital is described. A summary (p. 87) states that "patients who were living in the most under-stimulating social environment were likely to show the greatest clinical poverty, and this complex was likely to be more severe the longer the patient has been in hospital"; a graph is said to show (Fig. 4.4 and p. 82) "the striking relationship between length of stay and three other factors, one 'primary' (social withdrawal), one 'secondary' (attitudes to discharge), and one 'social' (contact with the outside)"; and most revealingly (p. 183), "if social improvements had not been taking place at the hospitals the tendency would have been for the patients to have become more withdrawn and reticent, simply with the passing of the years. That such deterioration was mainly prevented, and that so large a proportion as one-fifth of the patients actually improved, is a considerable achievement".

I pointed out that the longitudinal course of individual patients shows the same tendency for deterioration to be concentrated in the early years, with later consistency, or even improvement, described outside hospital. The statistical association between length of stay and negative handicaps appears to result from selection combined with changes in the initial pattern of schizophrenic symptoms (Abrahamson, 1983). The methodological issue is illustrated by Wing & Brown's treatment of the association between length of stay and unfavourable attitudes to discharge. The same graph is used to calculate the rate at which patients supposedly changed their attitudes year by year and leads on to the recently reiterated conclusion (Wing & Furlong, 1986) that negative attitudes accumulate gradually as part of a process of 'institutionalism'. This assumes that patients' initial attitudes have always been similar and ignores the possibility that those changes that do accumulate may follow discrete events such as loss of family or a home or transfer to a particular ward.

As the institutional era winds down it is important to refine the information with which we are left. That negative schizophrenic symptoms may be sensitive to the immediate effects of an impoverished environment yet resistant to its assumed long-term, cumulative moulding is relevant to future care and to any socio-medical model.

Equally, evidence that attitudes to discharge reflect realities rather than being explained by the gradual development of a secondary handicap has pràctical and ethical implications (Abrahamson & Brenner, 1982).

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DST in Apathetic Hyperthyroidism

Lahey (1931) drew attention to a form of hyperthyroidism which he called 'apathetic hyperthyroidism', occurring mainly in the elderly and lacking many of the cardinal features of thyroid overactivity. The predominant features were depression and apathy, and subsequent reports have confirmed his initial description (Thomas *et al*, 1970; Brenner, 1978). We report a patient fitting the description of apathetic hyperthyroidism in whom the dexamethasone suppression test (DST) was carried out.

Case report: A 69-year old widow presented with a three and a half year history of social withdrawal and isolation to the

degree that in the six months prior to presentation she led the life of a recluse. In addition, she complained of persistent depression, hopelessness, loss of interest, loss of drive, anergia, and suicidal ideas. On examination she appeared thin and frail. Her pulse was 76 per minute and regular, blood pressure was 130/80 mm Hg. There were no tremors, eye signs, or goitre. A diagnosis of depressive illness was made and she was prescribed lofepramine. In addition to other routine investigations thyroid function tests were made, and the results showed a raised serum thyroxine (167 nmol/l, reference range 50–150 nmol/l) and a low TSH (0.1 mU/l, reference range 0.5–5.0 mU/l). DST showed normal suppression of cortisol levels. A diagnosis of apathetic hyperthyroidism was made and she was started on carbimazole (10 mg tds).

Apathetic thyrotoxicosis occurs most often in elderly patients, and the salient characteristics that are helpful in establishing the diagnosis include typical placid apathetic facies, a small goitre, the presence of depression or lethargy, absence of ocular manifestations usually associated with hyperthyroidism, substantial muscular weakness and wasting, excessive weight loss, and cardiovascular dysfunction with atrial fibrillation (Thomas *et al*, 1970). Our patient showed many of these features. It is also of interest that the DST showed non-suppression. This is a confirmation of the reports by Kronfol *et al* (1982) and Martin & Waltz (1984), showing that DST is unaffected in depressions secondary to thyrotoxicosis.

The pathogenetic events leading to a state of apathy and depression in these patients is unknown. However, it has been postulated that there is a depletion of catecholamines or a lack of end-organ sensitivity following continual stimulation of the sympathetic nervous system.

A diagnosis of 'apathetic thyrotoxicosis' must be borne in mind when an elderly patient presents with symptoms of depression and apathy. Treatment of the endocrine dysfunction will often produce a marked improvement of the condition.

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Catatonic Signs in Schizophrenia

SIR: Catatonia is a not uncommon syndrome of varied aetiology (Gelenberg, 1976). We report a patient with catatonia in whom a curious combination of catatonic signs existed. Our use of terms is in accordance with the definitions of catatonic signs cited by Hamilton (1976).

Case report: Mrs L, a 60-year-old institutionalised patient with chronic schizophrenia, was prone to episodes of transient catatonic stupor, usually lasting a few hours at a time, at a frequency of about one per year. Despite extensive biochemical, radiological and electroencephalographical investigation no causative factor was demonstrated, and the catatonia was concluded to be a stress-related feature of her schizophrenic illness. The catatonia was invariably characterised by stupor and diffuse waxy flexibility.

During one episode of stupor, however, we noticed that different parts of her body responded differently to the examiner's touch. Active negativism was present from the neck up: there was strong resistance to the examiner's efforts to open her eyelids, lower her jaw or rotate her head. Waxy flexibility characterised her upper limbs: these could be moulded into positions which were maintained for several minutes despite potential discomfort. Mitgehen was demonstrated in her lower limbs: gentle pressure led to the limb moving readily in the direction of the applied pressure, despite the examiner's injunctions that she need not move. It thus appeared that there existed a cranio-caudal gradient of voluntariness to movement in obedience to the examiner's guidance, leading to what would seem to be a combination of logically antithetical catatonic signs. She recovered within three hours; her subsequent course was uneventful.

We are unable to ascribe any clinical significance to this combination of seemingly antithetical catatonic signs.

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