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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