psychiatric disorder (CIS score = 7; HAD score = 4). She remained well one month later.

(ii) A 58-year-old shop assistant attended the dermatology clinic with a three-month history of delusional parasitosis. She claimed she had been infected by her sexual partner, and that she could now feel insects crawling under her skin, stinging her, and living in her anus. She also felt worms moving in her stomach. She felt dirty, and thought her body odour was offensive. She had attended her GP, a private dermatologist, and several alternative practitioners, but without benefit. She had had a genuine hook worm infestation as a teenager. She had also received antidepressants from her GP when she was 32 and 42, during times of marital stress.

When seen by the psychiatrist she had been ill for four months. Her beliefs were delusional in nature, and she recorded maximal conviction on visual analogue scales of intensity of belief. Other than fatigue there was little to suggest depression (CIS score = 21). She refused treatment. One month later she reported that she had passed a "worm" in her stools, and described a three inch long creature with horns. Since then the rest of the parasites had died, and she no longer felt infested. She still felt she had a dirty tongue and skin, and still felt self-conscious of her body odour (CIS score = 12).

Previous reports in the psychiatric literature have emphasised the intractable nature of untreated delusional parasitosis (Monro, 1980; Sheppard *et al*, 1986), unless as part of a depressive illness. In a questionnaire survey of dermatologists, Lyell (1983) identified 282 cases, and confirmed the generally poor prognosis, but claimed 13 had remitted. He also described one case history of a spontaneous remission after three months. Batchelor & Reilly (1986) used a similar method to obtain details of 55 patients. Although again confirming the poor prognosis, "a third had a duration less than a year" while one had "improved slowly on dermatological treatment only". The gloomy natural history reported by psychiatrists may partly reflect patterns of referral.

SIMON WESSELY

Department of Psychological Medicine
King's College Hospital
Denmark Hill
London SE5

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Lucid Intervals in Catatonia: A Neuropsychiatric Snare for the Unwary

SIR:

Case report: A 42-year-old woman with a recent history of treatment of a major depressive disorder with dothiepin (150 mg/day) suddenly became agitated, complaining of being unable to walk and that her mind was going blank. She was admitted in a state of retarded depression, but despite an increase in dothiepin (225 mg/day) continued to deteriorate. Her mental state fluctuated between mute immobility and terror-stricken agitation, for which she was given chlorpromazine (100 mg i.m.) on three occasions over a one-week period. She resisted all nursing care and had to be fed and toileted, at times exhibiting verbigeration, bizarre and stereotypical behaviour, and catalepsy. Diazepam (20 mg i.m.) prior to CT scanning produced a dramatic improvement, lasting several hours. She spoke coherently and started eating, before relapsing. Investigations, including CT scan and EEG, were normal. Full recovery followed after three bilateral ECTs, and she remained well at six-month follow-up.

Amylobarbitone sodium interview continues to be advocated as a diagnostic aid in distinguishing catatonia secondary to idiopathic psychiatric ('functional') disorders from those that are toxic-metabolic or neurological ('organic') in origin (Altshuler *et al*, 1986). Our patient's response would be a case in point were it not for the fact that the catatonic signs became apparent only after the introduction of neuroleptic treatment. Ainsworth (*Journal*, January 1987, **150**, 110–112) and Chick *et al* (*Journal*, July 1987, **151**, 130–131) describe similar results with benzodiazepine and barbiturate infusions in catatonia, due to viral encephalitis and structural brain damage respectively. Further doubt is thrown on the diagnostic value of these techniques by reports of temporary or permanent relief obtained in a case associated with pituitary adenoma (Sheline & Miller, 1986), neuroleptic-induced catatonia (Fricchione *et al*, 1983) and the neuroleptic malignant syndrome (NMS) (Lew & Tollefson, 1983), which clinically resembles an iatrogenic variety of Stauder's "lethal catatonia". In addition, Lim *et al* (1986) have reported dramatic responses to i.v. phenytoin in catatonia as a manifestation of non-convulsive status epilepticus.

Several lines of evidence suggest that catatonia is the product of disturbed central neuroregulatory-vegetative functioning. The similarity in symptomatology between the neuroleptic-induced and other

varieties of catatonia may reflect a common pathophysiology, involving dopamine and GABA neurons in the mesostriatal and mesolimbic systems and hypothalamus (Fricchione, 1985). Dopamine agonists, e.g. dantrolene and bromocriptine, appear to relieve NMS by direct alteration of dopaminergic transmission, while barbiturates, hydantoins, and benzodiazepines, which interact with receptors closely related to the GABA/chloride-ionophore complex, do so indirectly, mediated by GABA feedback loops in the mesostriatal and mesolimbic systems. The relatively weak GABA-ergic properties of barbiturates may also account for the shorter duration of lucid intervals following amylobarbitone sodium infusion compared with those produced by benzodiazepines.

In view of the wide variety of conditions associated with catatonic states it seems doubtful whether a response to barbiturate or benzodiazepine infusion has any *diagnostic* validity, although the latter may be of therapeutic benefit, depending on the extent and localisation of underlying cerebral pathology. A safeguard against the pitfalls of the traditional 'functional/organic' dichotomy would therefore be to conceptualise catatonia as a non-specific neuropsychiatric syndrome, a final common pathway of response to an overwhelming psychiatric, neurological, or medical insult.

KAREL W. DE PAUW

Psychiatric Department
Leicester General Hospital
Gwendolen Road
Leicester LE5 4PW

T. KRYSTYNA SZULECKA

Department of Psychiatry
Bassetlaw District General Hospital
Worksop
Notts S81 0BD

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Sub-Cortical Dementia and the EEG

SIR: The review of the concept of sub-cortical dementia by Cummings (*Journal*, December 1987, **149**, 682–697) was timely, comprehensive and persuasive. A further body of evidence that can be cited in favour of the nosological distinction between cortical and sub-cortical dementia comes from the electro-encephalogram (Fenton, 1974). The EEG in the cortical dementias of the Alzheimer-senile type is invariably abnormal, characterised by diffuse asynchronous delta and theta dominant records. By contrast, the EEG of the sub-cortical dementias of Huntington's chorea, Parkinson's disease, post-traumatic encephalopathy, and post-encephalitic states is either normal or 'flat' low voltage in type. The vascular encephalopathies occupy a variable and often intermediate position between the two.

These differing EEG patterns in the cortical and the sub-cortical dementias must reflect the progressive disintegration of different neural systems, and is further evidence in support of employing this nosological classification of the dementias. We are at present attempting to carry out a double-blind assessment of the EEG in these two categories of dementia.

JOHN JOHNSON

EEG Department
Department of Psychiatry
University Hospital of South Manchester

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'Barking Mad'

SIR: "She had something that girl. She's mad, that's the worst of it. Bonkers, barking, round the bend". 'Barking mad' is a term in colloquial use which has started to appear in English literature as the line above, from John Welcome's 1968 play *Hell Is Where You Find It*, illustrates. In David Hare's play, *Plenty*, Sir Leonard Darwin observes that "in the diplomatic service it isn't as if a mad wife is any kind of professional disadvantage... Some of our senior men, their wives are absolutely barking". Despite such current use, barking is not described in psychiatric texts as a sign of mental illness. I have recently seen a patient in whom barking was part of the clinical picture.

Case Report: A sixty-year-old Irish divorcee had lived alone in a council flat since separation from her husband four years previously. Two months prior to the onset of symptoms she had retired from her work as a caterer. At presentation