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.

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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.

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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

she gave a four-month history of feeling depressed and anxious with loss of appetite and energy. Two months previously, while on a shopping trip with her sister, she had started to grimace and make growling noises in a supermarket. She continued to make barking and growling noises, frequently on buses and in shops, until, finally, she was admitted for assessment.

While on the ward it became evident that her barking was partly under voluntary control: she was able to reduce its frequency, but not abolish it altogether, upon being asked to do so. She was agitated, but showed no clear evidence that she was suffering from a depressive illness. She was treated with diazepam (2 mg t.i.d.), and over the course of a month her agitation progressively resolved. She stopped grimacing and making growling or barking sounds and was discharged.

I wonder if others have observed this symptom which, even in the diplomatic service, would seem likely to confer some disadvantage.

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Ofloxacin-induced Psychosis

SIR: The new quinolone derivatives, also known as gyrase inhibitors and widely used as potent chemotherapeutic agents, have frequently been implicated in causing central nervous system side-effects (Blomer *et al*, 1986; Cohen *et al*, 1984; Gleckman *et al*, 1979). Until now, only a few patients who have developed central nervous system side-effects have been seen by psychiatrists. We present two such cases, both induced by the latest gyrase inhibitor, ofloxacin (presently under clinical evaluation in the UK and US).

Case Reports: (i) A 28-year-old woman was given ofloxacin (200 mg po b.i.d.) over a course of 5 days for recurrent scalp infections. On the night of day one, she experienced a decreased need for sleep, and on the following day progressed to a hypomanic state with an overly cheerful mood, increased social and physical activity, restlessness, racing thoughts, and loss of appetite. Her condition did not change until day 4, when she appeared to enter a kind of intermediate stage with predominant depressive features, lasting until day 10 (ofloxacin was discontinued on day 5): she was dysphoric and irritable , with labile affect, complained of fatigue, loss of energy, indecisiveness, and lack of concentration. At the same time, she still experienced racing of thoughts, insomnia, loss of appetite, and a 10% loss of weight. During this time she also had four panic attacks. From day 10 to day 18 she progressed to a marked depressive syndrome with loss of energy, depressive mood, loss of interest in all usual activities, continued lack of appetite, and insomnia. After day 18, she improved rapidly; 25 days after the onset of symptoms she was back to normal. Our patient had no relevant psychiatric or family history and had not received any medication in the four weeks preceding her illness. Medical examination, EEG, CT-scan, and laboratory studies were normal, except for an elevated TSH (21.1 U/ml).

(ii) A 64-year-old woman had been treated with ofloxacin (200 mg po b.i.d.) for a complicated urinary tract infection. The drug was discontinued on day three, when she progressed acutely to a catatonic state with rapidly alternating episodes of stupor and excitement: short periods of mutism, catatonic posturing, and waxy flexibility changed within minutes to states of excitement with perplexity, screaming, verbigeration, and stereotypies. Her thinking became incoherent, with marked loosening of associations. At times she was perplexed, and partly disoriented in time and situation. She had religious delusions, auditory hallucinations, and recurrent optical illusions; all symptoms were present on days one and two after ofloxacin was discontinued. She was given chlormethiazol on days three and four, and improved within hours: she was oriented, and her thinking was coherent. She still complained of pressure of thoughts and distractibility. During the following 10 days she improved steadily and was almost back to normal on day 14, with partial amnesia for the duration of the psychosis.

This patient had had a psychiatric history with a poorly defined depressive episode in full remission, 15 years previously. Prior to admission, she had been given several spasmolytic and analgesic medications for urolithiasis, and had a history of compensated hyperthyroidism (TSH 0.98 U/ml, T3 75 g%, T4 8.37 g%). Medical examinations and other laboratory investigations were normal, and CT scan and EEG were negative.

The essential feature of case (i) was a bipolar mood disturbance, and our diagnosis according to DSM–III was organic affective syndrome. Using these criteria in case (ii), we diagnosed both an atypical organic brain syndrome and an organic delusional syndrome. According to ICD-9, our diagnosis in both cases was subacute organic psychosis.

Ofloxacin seems to be potent enough to produce different yet well-defined psychiatric syndromes. We have received an increasing number of reports on ofloxacin-induced psychoses. Further research should focus on ofloxacin's ability to generate model psychoses.

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