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C. BALL

The London Hospital Medical College
Turner Street
London E1 2AD

The nature of dysthymia

SIR: While reconceptualisation of dysthymia as a 'chronic' and 'subaffective' form of major depressive disorder for which pharmacotherapy is the mainstay of treatment (*BJP*, February 1995, **166**, 174–183) may promote the treatment of a subgroup of subjects, this proposition should be tempered as follows.

Dysthymia has been termed 'a new plastic box for some rather old wine' (Goldberg & Bridges, 1990). It represents a highly heterogeneous group of conditions that are frequently associated with intractable interpersonal difficulties and social misfortunes – hence its considerably higher prevalence rates in poorer people. When the term is used outside of the more affluent West, it has been criticised for medicalising social problems related to severe economic, political and health constraints which create endemic feelings of hopelessness and helplessness. In these contexts, despair is a response to real conditions of chronic deprivation and persistent losses, while negative cognition is an accurate mapping of one's place in an oppressive social system (Kleinman, 1987). The evidence for considering dysthymia as an affective disorder is largely non-specific. The positive response of a subgroup of patients to thymoleptics is no exception. Although tricyclic drugs are labelled as antidepressants, they ameliorate not only depression, but also a diversity of other disease categories.

The official recommendation of 'recognising' dysthymia as a pharmaco-responsive variant of major depressive disorder runs the risk of encouraging practitioners to replace non-pharmacological ways of relieving chronic social distress with an excessive reliance on drug therapy. There is no perfect shorthand for the complex illness reality of chronically dysphoric subjects. The question of whether dysthymia is a 'sub-affective' or 'supra-neurotic' disorder is epistemological and political in nature.

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SING LEE

Prince of Wales Hospital
Shatin, N.T.
Hong Kong

Where shall we put lithium *et al*?

SIR: It is now 32 years since the notion of a new and special psychiatric role for lithium was first aired in this Journal (Schou, 1963). Nevertheless there is still uncertainty about the category in which this drug and its alternatives, carbamazepine, valproate, etc., should be placed and what they should be called. In textbooks and lists of 'current reading' they have appeared under tranquillisers, or antimanic drugs, or antidepressant drugs, or they have been catalogued as 'mood stabilisers'.

This confuses and misleads. Lithium and its alternatives act therapeutically on manias and depressions and prophylactically on manic and depressive recurrences. They are neither neuroleptics nor tranquillizers, and they have a wider range than drugs with exclusively anti-manic or exclusively antidepressant action. Clearly they belong in a class of their own.

But what should that class be called? The term 'mood stabilisers' is hardly a happy choice, for the drugs do not stabilise abnormal moods; they do not perpetuate a mania or 'freeze' a depression. What they do is to counteract episodes of abnormal mood and maintain normal mood in patients with recurrent manic-depressive illness.

In 1963 the terms 'normothymotics' and 'mood normalisers' were proposed. They never caught on, and the former has been spurned for being ugly and of bastard linguistic origin (Johnson, 1984). Well, the beauty or ugliness of a name is a matter of personal taste, and there is precedence for words of mixed Latin-Greek parentage. However, that particular term is not important; the crucial point is that lithium and its alternatives are placed in a special class, and I still feel that 'mood normalisers' is an apt name. But there may be other possibilities. The adjective 'euthymic' has been used for the condition of manic-depressive patients during the intervals between episodes; can it possibly function as a noun and a class name, 'euthymics'? Linguistically minded readers of the *BJP* may have further proposals, and a discussion could perhaps lead to agreement about a fitting term.

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M. SCHOU

The Psychiatric Hospital
2 Skovagervej
DK-8240 Risskov
Denmark

Lithium augmentation

SIR: Katona *et al* (*BJP*, January 1995, **166**, 80–86) described a well-designed study of lithium augmentation. I would, nevertheless, like to highlight some weaknesses that the authors did not discuss.

It was admitted that the 'self-generated' group of anti-depressant non-responders were less refractory than patients included in previous studies. I think it is questionable whether many of them were refractory to monotherapy at all. Given that nearly half the patients in Phase II were non-compliant with lithium it seems probable that many apparent non-responders to lofepramine or fluoxetine in Phase I were actually non-compliant. Preskorn (1989) has appealed for the use of antidepressant plasma concentrations as a criterion of 'true' refractory depression. Such blood tests, although complicating a multi-centre trial, are essential if claims of treatment resistance are to be sustained. Assessing compliance by means of a blood test is far superior to tablet counts.

The assertion that the study sample had 'controlled and documented prior antidepressant treatment' is only true concerning the six week Phase I period. No summary of the lifetime treatment histories of these patients was offered. Thus patients with extensive histories of failed treatment, those most clinicians would perceive as 'refractory', were mixed up with those suffering first episodes of depression.

Finally, it was baldly stated that lithium augmentation is the most important pharmacological strategy in the management of refractory depression. I would argue, following Bridges (1983) and Quitkin (1985), that reviewing compliance with monotherapy by means of an antidepressant plasma concentration, then increasing the dose of that compound where possible, is of equal importance. A multi-centre trial comparing these two strategies is due.

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A.D. HODGKISS

Wellcome Institute for the
History of Medicine
183 Euston Road
London NW1 2BE

Hypomania induced by gabapentin

SIR: We report a case of a 49-year-old man with epilepsy and mild learning disabilities who developed symptoms of hypomania on starting gabapentin. The patient had a long-standing history of tonic clonic and partial complex seizures, the latter of which were difficult to control. We added gabapentin 300 mg into his existing regime of carbamazepine and lamotrigine, increasing daily by 300 mg to a maintenance dose of 300 mg t.d.s. Within the next 48 hours his behaviour began to change and he became markedly disinhibited and over familiar towards female staff on the ward, making inappropriate sexual remarks and becoming physically demonstrative. Although he had no flight of ideas, there was evidence of pressure of speech and his sleep pattern was mildly disrupted.

We reduced his gabapentin and his mental state improved without the need for psychotropic medication while maintaining good control of his partial complex seizures. Unfortunately his behaviour became aggressive and unpredictable, culminating in a violent attack on a fellow resident two weeks later. We stopped the gabapentin, and he reverted to his usual self.

The patient did have a past psychiatric history although he had not received a formal diagnosis. In 1988 he had an episode of disinhibited behaviour which in retrospect may have been hypomania although he did not receive any psychotropic treatment at the time.

Gabapentin is a relatively new anti-epileptic drug which is recommended as an adjunctive treatment for patients with refractory partial epilepsy. It is rapidly absorbed, does not bind to protein, is not metabolised, and does not affect serum concentrations of other anticonvulsants. It has been marketed as an ideal anticonvulsant pharmacokinetically. In clinical trials it showed a low relative toxicity. There have been a total of 32 reports received by the Committee of Safety of Medicine of