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Correspondence

Correspondents should note that space is limited and shorter letters have a greater chance of publication. The Editors reserve the right to cut letters and also to eliminate multitudinous references. Please try to be concise, strictly relevant and interesting to the reader, and check the accuracy of all **references in Journal style.**

DANTROLENE FOR NEUROLEPTIC MALIGNANT SYNDROME

DEAR SIR,

With reference to a recent letter from Dr Rosemarie V. Cope (*Journal,* August 1983, **143,** 202–3) which drew attention to the Neuroleptic Malignant Syndrome (NMS), it was stated that there is no specific treatment for this condition apart from supportive measures. There are however a number of reports in the literature on the successful use of dantrolene sodium in such cases (Delacour *et al,* 1981; Goekopp and Carbaat, 1982). The drug is normally given by the intravenous route, and the recommended dose is 2–3 mg/kg (Hall, 1980). Delacour *et al* (1981) report rapid muscular relaxation and return to normal temperature with this treatment, and the recovery time may be as little as one hour (Boules *et al,* 1982).

The reported mortality rate of NMS is 20 per cent (Caroff, 1980) and the risk of death or irreversible brain damage is thought to be related to the duration of the hyperthermic syndrome (Caroff, 1980). With supportive treatment such as cooling with ice or routine intensive care, it may take forty-eight hours or longer to return the temperature to normal (Goekopp and Carbaat, 1982). So the use of intravenous dantrolene should improve the outlook in this condition.

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DEMENTIA AND THE ABNORMAL DEXAMETHASONE SUPPRESSION TEST (DST)

DEAR SIR,

Balldin *et al* (*Journal,* September 1983, **143,** 277), in showing abnormal DST results in "Alzheimer's disease" and multi-infarct dementia, have confirmed other recent reports (Spar and Gerner, 1982; Raskind *et al,* 1982) that a proportion at least of demented patients reveal this implied impairment of the hypothalamo-limbic system function. In doing so they have disputed the view, put about by earlier reports, that an abnormal DST helped distinguish between depressive illness and dementia.

However, the restricted nature of these recent conclusions needs to be pointed out. In trying to establish the presence of abnormal DST in dementia, they were naturally at some pains to see that the demented patients they were investigating were free of depression. Roth (1978) has shown that depression, when present, is an early feature in both Alzheimer's disease and multi-infarct dementia. In excluding depression, it is possible that demented patients at a later stage of their illness were studied in relation to DST. This can, indeed, be shown to be the case. In the 3 studies quoted above, the patients with "parenchymatous" dementia (the term Alzheimer's disease is avoided as in none of the studies was the diagnosis established by histology) had a mean duration of dementia, among those responding abnormally to DST, of 4.6 years, 4.75 years and 7.7 years respectively. This is impressive survival for a collection of cases of pre-senile and senile dementia where mean survival could have been expected to be around 5–6 years. Moreover, far from being in a terminal phase of their illness, the patients exhibited a wide range of severity. Balldin *et al* say, "(the) subjects showed a range of dementia from mild to severe, which suggests that some were at a rather early stage of the disease"; Spar and Gerner's (1982) data show that though the abnormal DST cases were at a later chronological stage of the illness (4.75 years) compared to the normals (3.75 years), their mental test scores were comparable and exhibited a wide range; Raskind *et al* (1982) cases were "severe" but the mean duration of illness was over 7 years.

It would seem that in cases of dementia with abnormal DST, the duration was long, the clinical state variable and a disproportionate number of cases actually displayed mild and moderate dementia. It is thus possible that an abnormal DST may well be a marker of a less malignant course, at least in some cases of dementia.

It has been claimed that the abnormal DST may also reflect abnormality in central monoaminergic neurotransmission in relation to hypothalamo-limbic system dysfunction, but Carroll *et al* (1978) have speculated that the abnormal DST in depression may well be the result of muscarinic cholinergic hyperfunction in the limbic system. As hypofunction in cholinergic activity is the widely accepted view of Alzheimer disease pathology, it is also possible, in the light of these recent studies, that there is a more complex disturbance in cholinergic transmission (with the suggestion that prognosis depends on the direction of cholinergic dysfunction), than we have been led to believe.

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USE AND MISUSE OF THE PSE

DEAR SIR,

Wing in his lecture "Use and Misuse of the PSE" (*Journal,* August 1983, **143,** 111–17) is responding to Berner and Küfferle's comments (*Journal,* 1982, **140,** 558–65) on the failings of what they call "British Psychiatry", a generalization which would hardly survive for long a closer acquaintance with the wide range of opinions in Britain. This indeed is what one would find in any other country where the subject is alive, and debate is free. But be that as it may, Berner *et al* made a number of interesting points, which they present as those of "The Viennese School", quoting as their main authority Janczarik from Heidelberg.

I wish here to concern myself only with one point which was also dealt with by Wing, namely the proper place in the PSE of 'hypochondriasis'. The PSE lists this as item 9, as a worrying preoccupation with possible disease or bodily malfunction. Berner *et al* for the sake of 'increased categorical thinking in psychopathology' suggest that it should be ranged among what they call "fact phobias" which are more or less what Fish called 'fears restricted (or linked) to an idea'. Wing accepts Berner's point and announces that "fears of illness" will be included in the next edition of the PSE in the section on phobias.

The literature on 'hypochondriasis' has often been flawed by a lack of appreciation that it refers to the *content* rather than the *form* of an experience. It is in that respect comparable to jealousy, persecution or any of the other great themes of human existence. A few examples may illustrate this:—

1. A patient after an heart attack is taken by a strong fear of a repetition brought on by any activity. This fear makes it impossible for him to cooperate with efforts at rehabilitation.—Form: anxiety state.
2. A patient becomes a health-food buff, talks of little else, and makes life for his family and others difficult with his insistence on excluding all kinds of food from the diet to prevent diseases.—Form: overvalued idea.
3. A patient is constantly afraid of picking up germs when touching doorknobs, and goes to great length in avoiding this. He says he often realizes the absurdity of his fears has doubts about them, but is unable to resist these thoughts.—Form: obsession.
4. A melancholic patient is convinced he has cancer and is doomed to die.—Form: delusionlike idea.
5. A patient is convinced that rays are being directed from aeroplanes onto his genitals in order to bring about impotency and sterility.—Form: delusion.
6. A patient hears voices repeating over and over that he is riddled with VD.—Form: hallucination.

In all these diverse experiences the content reveals a fearful preoccupation with health. The list of the forms this might take can of course be extended. But if one conceives of a phobia as an avoidance of an overwhelming fear which the patient knows is triggered by objects such as for instance insects or certain ideas, although this strikes the patient himself as senseless but nevertheless irresistible, then it is difficult to think of clinical examples of phobias where the content would be hypochondriacal. It is therefore surprising