

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.

HYPOALGESIA IN DEPRESSION

DEAR SIR,

I would like to comment on the interesting but slightly confusing paper by Ben-Tovim and Schwartz (*Journal*, January 1981, 138, 37-39). I say confusing as the reference to Table I in the text should surely read Table II and we are left guessing as to the meaning of the enigmatic 'NA*' in that table. (See Editor's correction below). However, leaving aside these minor difficulties there are some points relevant to their findings which require more consideration.

It seems odd that depressed patients should have raised pain thresholds considering the frequency with which such patients complain of increased pain from arthritis and other chronic conditions, only to disregard their symptoms when their depression lifts. Ward *et al* (1979) and others have commented on the frequency of the association between pain and depression. Consequently, it is important to realize that tricyclic and other types of antidepressant medication have pronounced analgesic properties which have been reported now by a number of authors (Ward *et al*, 1979; Turkington, 1980; Massey and Riley, 1980; Gade *et al*, 1980) although their mode of action is not understood. Such an effect might explain the slightly raised pain thresholds of the depressed controls compared with the normal controls who, presumably, were not receiving antidepressant drugs.

With respect to the two cases of severe impairment of emotional responsiveness, it is noted that their thresholds of appreciation of stimuli are no different from those of the other depressed patients. On the other hand, their lack of response to pain caused by more intense stimulation is striking. Can it be assumed that pain thresholds were raised to such a degree as to render these patients almost analgesic? Or would it be more correct to argue that whereas pain as a sensation is appreciated the normal emotional reaction was missing, so giving an impression of indifference interpreted as evidence of high pain thresholds in these two patients?

The possible role of endorphins in this situation is far from clear as there is evidence that CSF endorphins are raised in depression and mania (Lindström *et al*, 1978) but there is no evidence that naloxone will improve depression which one might expect it to do if the mood change was the result of alteration in

brain levels of enkephalins and endorphins (Terenius *et al*, 1977). The study by Almay *et al* (1978) claimed that patients with psychogenic pain had higher CSF levels of endorphins than patients with organic pain which they stated were correlated with the degree of 'depression' in their patients. However, there is no reason for thinking that these authors were describing patients with severe endogenous depression such as afflicted the two cases described by Ben-Tovim and Schwartz. Biegon and Samuel (1980) have reported that tricyclic antidepressants bind to brain receptors and can be displaced by naloxone. The fact that naloxone given to patient 1 made no difference to her pain thresholds seems to indicate that neither endorphins nor antidepressants were playing any part in the aetiology of her pain insensitivity.

An alternative explanation is based on the possibility that these two patients were severely depersonalized. Such patients do complain of a profound loss of normal emotional response as well as a loss of physical sensation. Consequently, these two patients bear some resemblance to the cases of episodic depersonalization described by Davison (1964). All his patients had marked obsessional personality features and Davison proposed a neurophysiological explanation for the phenomenon. Possibly the two patients under discussion were similarly affected at the time of their being investigated.

F. A. WHITLOCK

Neuropsychiatric Unit,
The Prince Charles Hospital,
Rode Road, Cherside, Queensland 4032,
Australia

References

- ALMAY, B. G. L., JOHANNSSON, F., KNORRING, L. VON, TERENIUS, L. & WAHLSTRÖM, A. (1978) Endorphins in chronic pain I: Differences in CSF endorphin levels between organic and psychogenic pain syndromes. *Pain*, 5, 153-62.
- BIEGON, A. & SAMUEL, D. (1980) Interaction of antidepressants with opiate receptors. *Biochemical Pharmacology*, 29, 460-2.
- DAVISON, K. (1964) Episodic depersonalization. *British Journal of Psychiatry*, 110, 505-13.
- GADE, T. N., HOFELDT, F. D. & TREECE, G. L. (1980) Diabetic neuropathic cachexia. *Journal of the American Medical Association*, 243, 1160-1.

- LINDSTRÖM, L. H., WIDERLÖV, E., WAHLSTRÖM, A. & TERENIUS, L. (1978) Endorphins in human cerebrospinal fluid. *Acta Psychiatrica Scandinavica*, **57**, 153–64.
- MASSEY, E. W. & RILEY, T. L. (1980) Tricyclic antidepressants for peripheral neuropathy. *Journal of the American Medical Association*, **243**, 1133.
- TERENIUS, L., WAHLSTRÖM, A. & DGREN, H. (1977) Naloxone (Narcan) treatment in depression: Clinical observations and effects on CSF endorphins and monoamine metabolites. *Psychopharmacology*, **54**, 31–3.
- TURKINGTON, R. W. (1980) Depression masquerading as diabetic neuropathy. *Journal of the American Medical Association*, **243**, 1147–50.
- WARD, N. J., BLOOM, V. L. & FRIEDEL, R. O. (1979) The effectiveness of tricyclic antidepressants in the treatment of co-existing pain and depression. *Pain*, **7**, 331–41.

Correction

Table I in the text should read Table II. This, like the entry, NA*, is quite clear from the text; the latter might read 'not achieved'.—*Editor*.

LITHIUM THERAPY IN AGGRESSIVE MENTALLY SUBNORMAL PATIENTS

DEAR SIR,

I read with interest Dr Dales' article (*Journal*, November 1980, **137**, 469–74).

These are a notoriously difficult group of subjects to treat. In the study, one of the patients was withdrawn from lithium because of the onset of tardive dyskinesia. From the discussion it would appear that lithium was involved in the production of this somewhat serious side-effect. This would certainly be a unique finding. I wonder if it is not possible that the patient in question had been receiving neuroleptics prior to entering the lithium treatment (no information is given in the tables with regard to prior medication). If this were indeed the case then this would be an example of a withdrawal dyskinesia which usually takes place some one to three weeks after withdrawal of medication, but in some cases longer.

GEORGE M. SIMPSON

*Department of Psychiatry and the Behavioral Sciences,
University of Southern California,
11400 Norwalk Boulevard, Norwalk,
California 90650*

DEAR SIR,

The patient in question was receiving chlorpromazine (50–100 mg t.d.s.), orphenadrine (50 mg

t.d.s.) and haloperidol (3 mg t.d.s.) at the time of institution of treatment with lithium carbonate (250 mg t.d.s.). Haloperidol was discontinued three weeks later but treatment with chlorpromazine and orphenadrine continued for a further four weeks, by which time the patient's behaviour had so improved that both drugs were stopped. Tardive dyskinesia in the form of tongue movements and sucking was first noticed two months later.

P. G. DALE

*Coldeast Hospital,
Sarisbury Green,
Southampton SO3 6ZD*

PERSONALITY CHANGE FOLLOWING ACCIDENTS

DEAR SIR,

Dr Parker's well documented case (*Journal*, November 1980, **137**, 401–409) was of considerable interest; yet the contribution might have been more profitable had the major emphasis been placed on the developmental predisposition rather than on the apparent precipitant. The title might indeed have been "Personality Vulnerability Following Severe Emotional Inhibition in Childhood—The Report of a Double Murder".

BASIL JAMES

*Department of Psychological Medicine,
University of Otago,
Dunedin, New Zealand*

DEAR SIR,

Basil James' criticism of my article (*Journal*, November 1980, **137**, 401–409) expresses a point of view, popular in some circles, which my experience does not support. This has been discussed in detail in a chapter on "Accident Neurosis" (Parker, 1976) and will not be repeated here. In essence, he is saying that "severe emotional inhibition in childhood" is of such overwhelming importance, that any subsequent life events pale into etiological insignificance when explaining disturbed behaviour occurring in a man in his forties.

In my case report the co-twin was subjected to the same disturbing childhood yet did not murder his wife and daughter. One must therefore look for something additional to explain the discordance for homicide, and all professional people involved with the monozygotic twins whom I described were satisfied that two terrifying accidents emerged as the obvious difference in their histories.

The aim of my paper was to highlight the significance of accidents, even of an apparently minor nature, as a precipitating cause in the development of