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RELAXATION AND DEPERSONALISATION

DEAR SIR,

In a sample of forty anxious patients treated over the past few years with Jacobsen's progressive relaxation, there were seven who reported becoming distressed by the technique. A paradoxical outcome of this nature has been termed 'relaxation-induced anxiety' (Heide & Borkovec, 1983). Looking retrospectively at the clinical notes, it struck me that these seven could be singled out as reporting depersonalisation syndrome, prior to treatment. In a further retrospective investigation, the seven adverse responders were administered the 'Self Alienation Questionnaire' (Dixon, 1963) which purports to measure depersonalisation. As a group, they scored significantly higher self-ratings of 'Self-Alienation' than ten randomly selected control subjects who responded favourably to the relaxation procedure (adverse patients' mean = 32; controls' mean = 22; P = .05).

The questionnaires were administered post-treatment, which produces methodological problems in that treatment outcome may have flavoured response to the questionnaire items. Nonetheless, there is tentative evidence here that the presence of relaxation may even distress depersonalised patients, presumably by exacerbating feelings of unreality.

I wonder if any other reader has noticed adverse reactions to relaxation technique in depersonalised subjects? If so, the presence of depersonalisation may suggest that relaxation-orientated methods are contra-indicated.

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NORADRENALINE AND TARDIVE DYSKINESIA

DEAR SIR,

We read with interest the article by Jeste *et al* (*Journal*, February 1984, **144**, 177–80) in your Journal in which attention was drawn to findings supporting increased noradrenergic activity in tardive dyskinetic patients. The conclusions of the authors are in our opinion premature and even perhaps misleading. Most of the CSF samples for noradrenaline estimation were taken from schizophrenic in-patients, some of whom were receiving neuroleptic treatment and others who had been free of such treatment for at least 6 months.

Increased noradrenaline has been shown by other workers to occur in CSF samples of schizophrenic subjects (Hornykiewicz, 1982) as well as in certain cases in samples from some subcortical areas (Hornykiewicz, 1982).

Tardive dyskinesia is an abnormal movement disorder, reported in patients usually on long-term neuroleptic therapy. The dopamine theory implicating postsynaptic receptor hypersensitivity, has been the most widely accepted explanation of this condition. In view of the strong interrelationship between the dopamine and noradrenaline systems (for instance, damage to noradrenergic pathways in the prefrontal cortex prevents the development of denervation supersensitivity to D_1 dopamine receptors in the affected area (Hornykiewicz, 1982)), it is likely that chronic dopamine blockade leads to a compensatory noradrenergic hyperactivity.

The fact that beta-blockers have been shown to ameliorate tardive dyskinesia does not exclude the possibility that this condition involves other neurotransmitter systems, for instance Gaba-ergic drugs have also been useful in the therapy of this condition (Korsgaard, 1976).

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TREATMENT OF NEUROLEPTIC MALIGNANT SYNDROME

DEAR SIR,

In recent letters from Dr Jan Scott (*Journal*, January 1984, **143**, 98) and Dr P. D. White (*Journal*, April 1984, **144**, 437) in response to Dr Rosemarie V. Cope's letter (*Journal*, August 1983, **143**, 202–23) on neuroleptic malignant syndrome, various treatments are mentioned including dantrolene and bromocriptine.

A case of neuroleptic malignant syndrome successfully treated with amantadine has been reported (McCarron *et al*, 1982). Amantadine, which lacks significant anticholenergic action, is thought to increase synaptic dopamine availability, which may account for its effectiveness. Recently two cases were successfully treated with electro convulsive therapy (Jessee & Anderson, 1983). ECT was initiated because of the clinical deterioration that resulted from prolonged immobility and high fever. It resulted in dramatic reduction of fever and the beginning of overall clinical improvement. It seems that in cases where there is continued clinical deterioration or life threatening fever, in spite of the usual supportive measures, ECT should be considered in the treatment of neuroleptic malignant syndrome.

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DEXAMETHASONE SUPPRESSION TEST: DIFFERENT CATEGORIES OF RESPONSE

DEAR SIR,

The dexamethasone suppression test (DST) has been subjected to considerable research in the last twenty years. Carroll (1982) reviewed research in this area and concluded that the DST is a state-dependent biological marker which differentiates endogenous depression from other psychiatric disorders with a sensitivity of about 50% and a specificity of 96%.

On the basis of one DST result, patients are categorised as non-suppressors or suppressors, non-suppression being defined as a post-dexamethasone cortisol concentration of greater than 5 μg/dl (138 nmol/l), (Carroll *et al*, 1981). Despite the increasing use of serial DST's, (Albala *et al*, 1981; Holsboer *et al*, 1982, and Greden *et al*, 1983), this dichotomous categorisation has remained unquestioned. In a study involving serial DST's, I have found this categorisation to be inadequate, and have been able to define at least 3 distinct categories of serial DST response.

Thirty-six patients with a diagnosis of primary depressive illness were studied. A DST was performed before treatment and then weekly during a six week treatment period, with simultaneous ratings on the Montgomery and Asberg Depression Rating Scale (MADRS).

On the basis of the serial DST results, the following categories were identified:—

1. NORMALISING NON-SUPPRESSORS characterised by initial non-suppressor DST response which subsequently converted to and remained a suppressor response.
2. FLUCTUATING NON-SUPPRESSORS characterised by DST results fluctuating between suppression and non-suppression.
3. PERSISTENT SUPPRESSORS characterised by a persistent DST suppression response.

All three groups showed clinical improvement and the two non-suppressor groups both showed a significant reduction in post-dexamethasone cortisol concentrations during the treatment period, i.e. normalisation occurred. However, the two non-suppressor groups differed in two respects. The final post-dexamethasone cortisol concentrations of the fluctuating group were significantly higher when compared with the other groups, and when a Pearson's correlation was performed only the normalising non-suppressors had a significant correlation between post-dexamethasone cortisol concentrations and simultaneous MADRS scores—see table.

Correlations between weekly post-dexamethasone cortisol concentrations and simultaneous MADRS scores

Category	Pearson's r
Normalising non-suppressors	0.5350 ($P < 0.001$)
Fluctuating non-suppressors	0.1849 (NS)
Persistent suppressors	0.0034 (NS)

These findings appear to support the distinction, made in the above categorisation, between normalising non-suppressors and fluctuating non-suppressors. The longer term prognostic implications of these groups require further investigation.

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