WANN, L. S., GROVES, J. R., HESS, T. R. *et al* (1983) Prevalence of mitral valve prolapse by 2D echocardiography in healthy young women. *British Heart Journal*, **49**, 334–40.

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.

W. D. FEWTRELL
Rotherham District General Hospital,
Moorgate Road,
Rotherham S60 2UD

References

HEIDE, F. J. & BORKOVEC, T. D. (1983) Relaxation-induced anxiety: paradoxical anxiety enhancement due to relaxation training. *Journal of Consulting and Clinical Psychology*, **51**, 171–82.

JACOBSEN, E. (1983) *Progressive Relaxation*. Chicago: University Press.

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).

R. SANDYK
M. A. GILLMAN
South African Brain Research Institute,
Johannesburg 2000,
South Africa

References

HORNYKIEWICZ, O. (1982) Brain catecholamines in schizophrenia a good case for nor-adrenalin. *Nature*, **299**, 484–6.

KORSGAARD, S. (1976) Baclofen (Liorasal) in the treatment of neuroleptic-induced tardive dyskenesia. *Acta Psychiatrica Scandinavica*, **54**, 17–22.

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.