

to their own particular units and the necessity of generating tertiary referrals to ensure their survival.

I am aware of the fact that there are outcome data available from these units (e.g. Rosser *et al*, 1987) but the authors go well beyond this evidence in their assumptions about efficacy. The article makes unjustified, cavalier and, at times, insulting assumptions about the therapeutic milieu on acute psychiatric wards. The acute ward is stereotyped as hierarchical, authoritarian, dominating and controlling. Ward staff are stereotyped as undemocratic, narrow-minded, given to bland rationalisations to protect themselves, and generally unable to make the empathic leap of understanding to their patients' internal world.

The staff of all disciplines on acute in-patient units are, I would humbly suggest, not lacking in flexibility, empathy and communication. In addition, unlike the isolated specialist in-patient unit, they are already integrated into the network of multidisciplinary and multi-agency services that connect directly with the real world into which the patient will be discharged, thus potentially mitigating the effects of this transition. Norton & Hinshelwood's description of general services is a caricature. By marketing the specialist unit as a place in which the problems they allude to do not exist, they promote the very 'splitting' (perhaps as evidenced by this letter!) that they are attempting to address.

NORTON, K. & HINSHELWOOD, R. D. (1996) Severe personality disorder, Treatment issues and selection for in-patient psychotherapy. *British Journal of Psychiatry*, **168**, 723-731.

ROSSER, R., BIRTH, S., BOND, H., *et al* (1987) Five year follow up of patients treated with inpatient psychotherapy at the Cassel Hospital for Nervous Diseases. *Journal of the Royal Society of Medicine*, **80**, 549-555.

P. MALLETT

North West London Mental Health Trust
Central Middlesex Hospital
Acton Lane
London NW10 7NS

Who responds to electroconvulsive therapy?

SIR: It was encouraging to read that ECT is a viable treatment option for depressed patients in New York, regardless of the presence or absence of psychosis, retardation and/or agitation (Sobin *et al*, 1996). These findings confirm those from the Nottingham ECT trial (O'Leary *et al*, 1995). In our analysis we highlighted the greater percentage response to simulated ECT at Northwick Park (Buchan *et al*, 1992) compared with those in

Leicester or Nottingham, and postulated this as the main discriminating factor between the centres. It was interesting to read, in view of the similarity of the overall conclusions from New York and Nottingham, how the average percentage response to 'ineffective ECT' in New York was very similar to those in Leicester and in Nottingham. Factors reducing the response to simulated ECT, we proposed, included the proportion of patients failing to respond to antidepressants (high in Nottingham and in Leicester) and mean length of hospital stay prior to ECT (longest in Nottingham). We suggested also that a prior history of ECT (lowest in Northwick Park) may have reduced the response to simulated ECT, as in the absence of a convulsion patients would guess that they were not receiving 'real' treatment during the trial and thus would not have remained blind to treatment type. An advantage of the New York study is that patients in the 'ineffective ECT' subgroup did experience a convulsion, thus minimising the confounding effect of previous history and underscoring the validity of their conclusions.

BUCHAN, H., JOHNSTONE, E., MCPHERSON, K., *et al* (1992) Who benefits from electroconvulsive therapy? Combined results of the Leicester and Northwick Park trials. *British Journal of Psychiatry*, **160**, 355-359.

O'LEARY, D., GILL, D., GREGORY, S., *et al* (1995) Which depressed patients respond to ECT? The Nottingham results. *Journal of Affective Disorders*, **33**, 245-250.

SOBIN, C., PRUDIC, J., DEVANAND, D. P., *et al* (1996) Who responds to electroconvulsive therapy? A comparison of effective and ineffective forms of treatment. *British Journal of Psychiatry*, **169**, 322-328.

D. O'LEARY

Littlemore Mental Health Centre
Oxford OX4 4XN

Artificial neural networks and psychiatric disorders

SIR: Zou *et al* (1996) report the use of a fully connected back-propagation artificial neural network (ANN) to classify interview data into three diagnostic groups. They describe a neural network with 396 input nodes and 40 hidden nodes. The number of output nodes is unspecified, but the ANN has a minimum of 15 880 connections. This is a very large feature space in which to distribute representations of only 60 training cases. The usual rule of thumb in ANN research is that the number of training cases should be at least twice the number of connections within the ANN for satisfactory learning to occur. In Zou *et al*'s study this would mean the ANN should have only 30 connections (e.g. nine input nodes, three hidden nodes and one

output node), or that the training set should include over 30 000 cases.

The aim in training a classification ANN is to extract features from the training data that characterise the classes of interest. Since Zou *et al*'s ANN has well over 15 000 path options for the training set of 60 cases, the feature extraction properties of the ANN were redundant. The ANN was simply required to represent each training case in learning space. Classification of the test data would have proceeded by finding the closest match among the training data representations and adopting this diagnosis. Zou *et al*'s approach has, therefore, probably created a pattern matching system rather than a true ANN classification system. As such, it achieved a satisfactory result, but would be expected to be less successful when more diagnostic groups are added and the classification task is more complex.

ANNs have considerable promise in the classification of psychiatric disorders (Galletly *et al*, 1996). However, an ANN is not merely a black box. Attention must be given to the choice of appropriate architecture, input data, training and test data sets, classification thresholds and initial weights, all of which appear to be either less than optimal or unspecified in Zou *et al*'s paper.

GALLETLY, C., CLARK, C. & MCFARLANE, A. (1996) Artificial neural networks: A prospective tool for the analysis of psychiatric disorders. *Journal of Psychiatry and Neuroscience*, **21**, 239–247.

ZOU, Y., SHEN, Y., SHU, L., *et al* (1996) Artificial neural network to assist psychiatric diagnosis. *British Journal of Psychiatry*, **169**, 64–67.

C. A. GALLETLY
C. R. CLARK

*Cognitive Neuroscience Laboratory and School of Psychology
Flinders University of South Australia
GPO Box 2100
Adelaide, SA 5001*

A. C. MCFARLANE

*Department of Rehabilitation and Community Psychiatry
University of Adelaide
South Australia*

Lithium and ECT in combination

SIR: We read with interest the article by Jha *et al* (1996). The clinical implication suggested by the authors, "The combination of ECT and lithium may be justified in selected cases" needs to be viewed with caution. There are some reports to

suggest increased risk of neurotoxicity associated with ECT and lithium in combination (Small *et al*, 1980; El-Mallakh, 1988). Potential risk of prolongation of depolarising muscle relaxant effects by lithium is possible (Hill *et al*, 1976). In any case, there are no confirmed trials showing therapeutic advantage of ECT and lithium in combination over ECT sans lithium.

It is therefore clinically prudent to withhold lithium during ECT. Lithium may be restarted, if indicated, after completion of a course of ECT. In our experience, such a strategy has not resulted in any symptom relapse during the period before therapeutic levels are achieved.

EL-MALLAKH, R. S. (1988) Complications of concurrent lithium and electroconvulsive therapy: A review of clinical material and theoretical considerations. *Biological Psychiatry*, **23**, 595–601.

HILL, G. E., WONG, K. G. & HODGES, M. R. (1976) Potentiation of succinylcholine neuromuscular blockade by lithium carbonate. *Anesthesiology*, **44**, 439–441.

JHA, A. K., STEIN, G. S. & FENWICK, P. (1996) Negative interaction between lithium and electroconvulsive therapy – A case-control study. *British Journal of Psychiatry*, **168**, 241–243.

SMALL, J. G., KELLAMS, J. J., MILSTEIN, V., *et al* (1980) Complications with electroconvulsive treatment combined with lithium. *Biological Psychiatry*, **15**, 103–112.

B. N. GANGADHAR
N. JANAKIRAMAIAH

*Department of Psychiatry
National Institute of Mental Health and Neurosciences
Bangalore 560 029
India*

False memory syndrome

SIR: I wish to clarify a comment about the False Memory Syndrome Foundation made by Brewin in his editorial (1996). He states:

"Recently, however, doubt has been cast on the process whereby forgotten memories of child sexual abuse appear to be recovered within therapy, and it has been suggested that many if not all of these memories are the product of inappropriate therapeutic suggestion. This suggestion has been promulgated in particular by the False Memory Syndrome Foundation in the US. . ."

The False Memory Syndrome Foundation has no way of knowing whether or not "many" memories recovered in therapy are the product of inappropriate suggestion. Therapy sessions are confidential. There does, however, seem to be ample evidence that some memories recovered in therapy may be false. There is also evidence that some therapists practice techniques that carry a high risk of suggestion. Research by Poole *et al* (1994) indicates that