our own review. Just as importantly, quantitative analysis also allows summary conclusions to be reached on the basis of as many cases as possible. It is the number of cases entered in randomised studies that determine the confidence we can have in the findings. An overview of treatment studies in breast cancer illustrates the point ($n = 75\,000$), and previous controversy over the same treatments exemplifies what may be described as the qualitative fallacy (Early Breast Cancer Trialists' Collaborative Group, 1992). The latter publication shows what can be done when clinicians take treatment issues seriously. We will be delighted if our conclusions serve as a stimulus to further studies on patients defined more strictly for refractory illness. However, the existing data from randomised trials, together with a good deal of more anecdotal evidence which should not be discounted, support the view that lithium augmentation is an effective manoeuvre in patients who have not responded to a tricyclic antidepressant.

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Who benefits from ECT?

SIR: The casual or unsophisticated reader of the recent article by Buchan *et al* (*Journal*, March, 1992, **160**, 355–359), upon encountering the statements in the abstract that "patients who were neither retarded nor deluded did not benefit significantly from real as opposed to simulated ECT", and later in the summary that "real ECT does not appear to be effective in non-retarded, non-deluded patients", might not realise that the authors did not actually determine whether any non-retarded, non-delusional patients were ECT-responders. In fact, Buchan *et al* have simply demonstrated the truism that removing ECT-responders from a sample of depressives leaves a subsample of ECT non-responders.

Only randomised prospective comparisons of genuine v. sham ECT, with stratification of subjects by the clinical predictor variables of interest (e.g. presence of delusions or retardation), can definitely answer the question: "Who benefits from ECT?".

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Do benzodiazepines interfere with the action of ECT?

SIR: Cohen & Lawton (*Journal*, April 1992, **160**, 545–546) suggest that the presence of benzodiazepine drugs may interfere with the ability of the brain to respond to bilateral ECT. I would point out that, in experimental animals at least, there is indeed evidence for this.

When electroconvulsive shocks (ECS) are given to mice in a manner somewhat similar to the clinical administration of ECT (5 ECS given spread out over 10 days to anaesthetised animals) various changes occur in neurotransmitter function. These include enhanced behavioural responses to drugs stimulating dopamine and 5-HT_2 receptors and an attenuated response to the sedative effects of the α_2-adrenoceptor agonist clonidine (for review see Green & Nutt, 1987) and it has been proposed that some of these changes could be associated with the antidepressant action of ECT (Green & Nutt, 1987).

When diazepam was given before each ECS, the dopamine and 5-HT_2-receptor-mediated behavioural changes no longer occurred (Green & Mountford, 1985). This was clearly not due to any modification by the benzodiazepine of the convulsant effect of the ECS both because no obvious modification was seen to occur and, most critically, because the same effect was seen when the diazepam was given 5 minutes *after* the ECS administration. This effect of diazepam also appeared to be due to a specific action at the benzodiazepine receptor-binding site in the brain because the selective benzodiazepine antagonist flumazenil blocked the effect of diazepam on the ECS-induced changes (Green & Mountford, 1985).

It is always difficult to speculate on the relevance of animal experimentation to clinical practice.