ECT at the unit were in keeping with national trends reported by the Department of Health (1999). Over this 3-year period, consultant groups in the unit remained largely unchanged.

Concluding from this study, I feel that ECT is more commonly used in treating older people with depression. Availability of newer antidepressants and other treatment modalities, as highlighted by Eranti & McLoughlin (2003), could be some of the reasons why there is a decline in the number of patients under 65 who receive ECT. Furthermore, the limited response to ECT in the subjects of our study could be due to the fact that these patients had been treatment-resistant. On the other hand, in the case of older people suffering from severe depression, there are other factors that tilt the treatment options towards ECT. Factors such as physical frailty, propensity to develop side-effects from antidepressants, and the serious effects of dehydration and weight loss (as a result of severe depression) make it imperative that depression is controlled rapidly.

I feel that in the future, it will be old age psychiatrists who will be using ECT more commonly as a treatment option for depression. Old age psychiatrists could take a leading role in ensuring that psychiatric trainees have the opportunity to obtain experience in ECT. The effective (albeit reduced) use of ECT resulting in good clinical outcomes will ensure that clinical interest in this treatment modality is maintained.

Department of Health (1999) *Electro Convulsive Therapy: Survey Covering the Period from January 1999 to March 1999, England* (Bulletin 1999/22). London: Department of Health.

Eranti, S. V. & McLoughlin, D. M. (2003) Electroconvulsive therapy – state of the art. *British Journal of Psychiatry*, **182**, 8–9.

Glen, T. & Scott, A. I. F. (1999) Rates of electroconvulsive therapy use in Edinburgh (1992–1997). *Journal of Affective Disorders*, **54**, 81–85.

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Lithium augmentation in treatment-refractory unipolar depression

Stimpson *et al* (2002) have taken an 'all or nothing' approach to evaluating randomised controlled trials (RCTs) for their systematic review. Their rigorous procedures eliminated over 98% of the 919 RCTs considered (although we note that the flow chart in Fig. 1 appears to 'lose' 166 of them without explanation). As a consequence, they have provided a matchless summary of the very best evidence about intervention for treatment-refractory unipolar depression but have left undescribed the very large quantity of remaining levels of evidence.

In 1999 Bauer and Dopfmer identified 11 placebo-controlled studies of lithium augmentation. As always, the trials were of varying quality; nevertheless, they concluded (using the three studies of highest quality, two of which were used by Stimpson *et al*) that there is 'firm evidence' in favour of lithium as an augmentation strategy for treatment-refractory unipolar depression, with a number needed to treat of 3.7. They supported their conclusion by performing a separate analysis adding a further six studies (that used either lower doses or shorter duration of lithium augmentation) and found a similar, indeed slightly stronger, effect size (Bauer & Dopfmer, 1999).

We note that there have been no studies of lithium augmentation against placebo for treatment-resistant unipolar depression that are of a suitable quality for a systematic review in the approximately 3-year period between the acceptance dates of the two papers cited above. We suggest that many clinicians now consider the weight of evidence (at many levels) supporting the use of lithium as an augmentation strategy for treatment-refractory unipolar depression sufficiently compelling. Thus, it is unusual for our service dedicated to treatment-resistant depression to receive referrals of patients not yet tried on lithium. Although further and better RCTs of lithium augmentation would be welcome (even Bauer & Dopfmer identified only 234 subjects studied), many would feel that other questions now have more clinical salience. Pressing examples might include whether psychological treatments are effective in these patients, how they compare with lithium augmentation, and how olanzapine augmentation (for which a large body of evidence is emerging; see Dube *et al*, 2002) compares with both.

Bauer, M. & Dopfmer, S. (1999) Lithium augmentation in treatment-resistant depression: meta-analysis of placebo-controlled studies. *Journal of Clinical Psychopharmacology*, **19**, 427–434.

Dube, S., Anderson, S. W., Paul, S., *et al* (2002) Metaanalysis of olanzapine–fluoxetine use in treatment resistant depression. *International Journal of Neuropsychopharmacology*, **5** (suppl. 1), 105–106.

Stimpson, N., Agrawal, N. & Lewis, G. (2002) Randomised controlled trials investigating pharmacological and psychological interventions for treatment-refractory depression. Systematic review. *British Journal of Psychiatry*, **181**, 284–294.

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Authors' reply: According to Drs Lee and Cleare 'many clinicians' regard the current evidence for lithium augmentation in treatment-refractory depression as 'compelling'. They are correct in repeating one of the principles of evidence-based medicine, that all levels of evidence need to be taken into account when making clinical decisions.

Previous systematic reviews of this area have included patients who have had $\leqslant 3$ weeks' treatment with an antidepressant or who have bipolar disorder. We do not think that many UK psychiatrists would consider lithium augmentation in unipolar depression that had not responded to an antidepressant for only 3 weeks. For patients with bipolar disorder, most UK psychiatrists, we think, would in any case be treating with lithium or another mood-stabiliser. Our inclusion criteria, which were set before the review started, were based therefore upon sensible and pragmatic clinical considerations.

We too were surprised and shocked by the lack of randomised evidence to support lithium augmentation; but it is also important to remember that lithium may well be effective, even though the evidence to support its use is extremely weak.

Lithium has a number of potentially serious side-effects, even at normal therapeutic doses (Bell *et al*, 1993). When we discuss the advantages and disadvantages of lithium with our patients we are unable to provide them with much more than clinical anecdote in its favour. We certainly have no idea from empirical research about the severity of depression for which lithium augmentation might be effective.

We have a collective responsibility to our patients to provide them with good-quality research evidence to justify the treatments we recommend. As a profession we need to address areas of uncertainty

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such as this using well-designed RCTs that will inform clinical practice.

Declaration of interest

G.L. has received payments for lectures from the pharmaceutical industry.

Bell, A. J., Cole, A., Eccleston, D., *et al* (1993) Lithium neurotoxicity at normal therapeutic levels. *British Journal of Psychiatry*, **162**, 689–692.

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Getting closer to suicide prevention

We would like to offer a slightly different perspective from De Leo (2002) on the progress of suicide prevention. There is no argument against suicide representing a complex set of variables. The general method of science, however, is to analyse phenomena in order to find the most simple explanation – the principle formulated by William of Occam in the early 14th century. In the medical paradigm, death results from a disease process. Studying people with heart attacks led to the identification of atherosclerosis as the underlying disease process for the vast majority. Treating myocardial infarctions is important. The development of various approaches to prevention and treatment of atherosclerosis has, however, prevented more premature deaths from heart attacks. Why must one conclude that suicide is a more complicated medical problem than myocardial infarction?

A fundamental discovery was made in the late 1950s (Robins *et al*, 1959): the majority of suicides were committed by people with clinical depression. This finding has been replicated over and over again and we believe that many, like us, have concluded that this connection has been replicated enough to be proven. We have also presented evidence that suicides occur infrequently in people with depression taking antidepressant medication (Isacsson *et al*, 1994).

Thus, in spite of the 'extreme complexity' of the phenomenon of suicide, a simple and testable hypothesis can be stated: depression is a necessary cause of most suicides. Based on this proposition, it has been suggested that effective suicide prevention must focus on improving identification and treatment of depression in the population (Isacsson, 2000). When we look at the declining suicide rates over the past decade or so, we see a great deal of support for that theory. Since the introduction of the new generation of antidepressants during the past 10–15 years, the use of antidepressants has increased up to 5-fold. Concurrently, suicide rates have decreased considerably in many Western countries (e.g. Joyce, 2001). It appears to us that we *are* getting closer to suicide prevention.

We believe that a lack of focus on depression as the basic disease leading to suicide is most likely the reason why the current decline in suicide rates 'seems reasonably unrelated to the existence of any national plan'.

Declaration of interest

Both authors have delivered lectures at scientific meetings sponsored by pharmaceutical companies.

De Leo, D. (2002) Why are we not getting any closer to preventing suicide? *British Journal of Psychiatry*, **181**, 372–374.

Isacsson, G. (2000) Suicide prevention – a medical breakthrough? *Acta Psychiatrica Scandinavica*, **102**, 113–117.

___, Bergman, U. & Rich, C. L. (1994) Antidepressants, depression, and suicide: an analysis of the San Diego Study. *Journal of Affective Disorders*, **32**, 277–286.

Joyce, P. R. (2001) Improvements in the recognition and treatment of depression and decreasing suicide rates. *New Zealand Medical Journal*, **114**, 535–536.

Robins, E., Murphy, G. E., Wilkinson, R. H., *et al* (1959) Some clinical considerations in the prevention of suicide based on a study of 134 successful suicides. *American Journal of Public Health*, **49**, 888–899.

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Author's reply: There is little doubt that depression has a major role in suicide, being identifiable in approximately 50% of cases (Andersen *et al*, 2001). For this reason, depression is a target in all the national plans that I am aware of.

The role of depression in suicide has been well known since antiquity (Van Hooff, 2000) and this understanding has been largely responsible for the decline in a punitive attitude towards those exhibiting suicidal behaviour since the Enlightenment. Consequently, the 'fundamental discovery' at the end of the 1950s of the role of affective disorders in suicide was far from revolutionary. It is worth remembering that in the vast majority of cases, fortunately, depression does not culminate in suicide. The relative risk for suicide across the lifespan has been recently revised downwards (see, for example, Bostwick & Pankratz, 2000). In addition, a significant percentage of patients who die by suicide appear to have been adequately treated (25% in the experience of Andersen *et al*, 2001). A World Health Organization (1998) technical report has pointed out that optimal treatment of clinical depression would have little impact on global suicide rates, leaving the field open to speculations around more powerful factors in suicide prevention. In any case, the 'medical paradigm' is, in my view, only one of many possible perspectives, and needs to be integrated with other disciplines. Clearly, it is not the different prevalence of depression among countries that helps to explain the enormous diversity in rates of suicide that I mentioned in my editorial. Religious, cultural and social factors play very relevant roles in suicidal behaviour. It is in this light that the World Health Organization has correctly endorsed an ecological model, to help both understand and prevent/intervene in suicidal behaviours.

I am aware that Isacsson and Rich, through their research, strongly support the role of the newer antidepressants in preventing suicide. But others are a bit more hesitant in accepting this hypothesis (see, for example, Van Praag, 2002), and maybe lithium has shown more consistent (and convincing) effects, so far, on suicidal behaviour (Tondo *et al*, 2001).

With regard to the comments about a possible overemphasis on the complexities of suicidal behaviour, I am afraid that the philosopher Albert Camus, if he came back to life, would die again on hearing that!

Andersen, U. A., Andersen, M., Rosholm, J. U., *et al* (2001) Psychopharmacological treatment and psychiatric morbidity in 390 cases of suicide with special focus on affective disorders. *Acta Psychiatrica Scandinavica*, **104**, 458–465.

Bostwick, J. M. & Pankratz, V. S. (2000) Affective disorders and suicide risk: a re-examination. *American Journal of Psychiatry*, **157**, 1925–1932.

Tondo, L., Ghiani, C. & Albert, M. (2001) Pharmacologic intervention in suicide prevention. *Journal of Clinical Psychiatry*, **62** (suppl.), 51–55.

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