

enough to reach a diagnostic threshold. This is partly due to the reactive nature of CMHT care and partly because of concerns about treating ‘false positives’, benign and transient states that will not make a transition into a major mental disorder. An unfortunate consequence of this well-meaning caution is that young people are denied earlier and safer interventions, which are not only clinically appropriate at an early stage, but have the potential for altering the prognosis and preventing the emergence of more serious illness.

A staging approach also offers exciting possibilities for developing specific clinical and biological markers of mental illnesses and understanding the relationship between clinical states and neuropathological and neurophysiological changes that accompany illness progression.³

I also share Dr Agius’ concern about the short-term financial pressures that may encourage managers to amalgamate early intervention services into CMHTs. This will simply dilute the well-established effectiveness of early intervention services in caring for vulnerable young people, while offering no improvement in CMHT functioning.

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Dilemma over antipsychotic use in dementia

The editorial by Treloar *et al*¹ has raised a controversial but justified issue regarding antipsychotic prescription in patients with dementia. We agree with the editorial supporting the cautious use of these drugs based on the ethical premise of reducing patient distress and palliation. However, we felt that there was a relatively quick and unchallenged submission to another important premise of the observed harm, which is intricately related to the topic in question. Our strong concern is that such unequivocal acceptance of the observed risks is likely only to enhance the ethical dilemma in a reader’s mind. The decision to use these drugs, even for palliative purposes, is likely to be strongly governed by our safety and risk assessments. Are we not in a dilemma over the available safety evidence as well?

Is the observed harm specific to antipsychotic drugs, old age, dementia or behavioural and psychological symptoms of dementia (BPSD)? Is the observed association necessarily causation or are there certain limitations to a definite conclusion? For example, many a time the indication for which a drug is prescribed in dementia may be the cause of increased mortality rather than the drug *per se*. To quote the detailed Department of Health report,² ‘people with dementia and BPSD may be more likely to die (and to be prescribed antipsychotic drugs) than people with dementia and no BPSD’. Safety concerns regarding the use of antipsychotic drugs in elderly populations are a valid consideration, but are the risks also specifically higher for elderly people with dementia? The landmark meta-analysis of randomised controlled trials (RCTs),³ which concluded with a small increased risk for

death with antipsychotics compared with placebo, also mentions that these results should be considered as hypothesis-generating. None of the individual drugs included in the 17 RCTs was sufficient to conclude for an increased risk, but a combined statistical effect was found. Does this call for a verification or should it be taken as conclusive?

Regarding efficacy studies, antipsychotic drugs have mostly been tested for treating BPSD. Behavioural and psychological symptoms of dementia is quite a heterogeneous term, used for an array of challenging behaviours such as restlessness, agitation, wandering, vocalisations, resisting help with dressing and personal hygiene, and verbal and physical aggression. Although the use of the term BPSD is quite appropriate in social dementia research (e.g. caregiver burden), is such a heterogeneous amalgamation of behaviours, which may or may not be of psychotic origin, a justified end-point to study clinical efficacy of drugs, or do we need more specific symptom clusters as indications of antipsychotic use in dementia? Further, is the number needed to treat (5–11) for antipsychotic drugs for behavioural improvement in dementia² any different from numbers needed to treat for antipsychotic drugs in schizophrenia?⁴

Undoubtedly, from a clinical perspective, extreme care and caution should be exercised in prescribing antipsychotics in old age, especially for those with an underlying organic illness (e.g. dementia). Regarding the dilemma whether they should ‘ever or never’ be prescribed for patients with dementia, our point of contention is: (a) we cannot focus the debate only on the ethical angle to resolve this dilemma, there are several unanswered medical questions; (b) we cannot close our eyes to the caveats in existing safety and efficacy studies; and (c) we need to resolve the ambiguity surrounding the available evidence to empower us for an ethical as well as informed decision. More than ever, the dilemma is to arrive at certain indications for which we can use antipsychotics with relative safety.

- 1 Treloar A, Crugel M, Prasanna A, Solomons L, Fox C, Paton C, et al. Ethical dilemmas: should antipsychotic ever be prescribed for people with dementia? *Br J Psychiatry* 2010; **197**: 88–90.
- 2 Banerjee S. *The Use of Antipsychotic Medication for People with Dementia: Time for Action*. Department of Health, 2009.
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I agree with the views expressed by Treloar *et al*¹ regarding antipsychotic use in dementia. This is not only an ethical dilemma, but an issue of medical prescribing practice that has entered public and political domains. The present widespread use of antipsychotics seems to be unjustified but the emphasis should be on more rational use of these medications rather than an either/or debate. Our focus should be to develop policies and protocols which can lead to justified use of antipsychotics, with continuing reviews of the need for these medications. Their editorial is a step in right direction.

It seems that antipsychotic use in dementia is being demonised in the media.^{2–4} Policy makers are also pushing for a decrease in their use. I have two issues with the direction this debate is taking us. First, I hope the pendulum does not swing

so far in the other direction, that psychiatrists find it hard to prescribe the medication even to those who will benefit from its use.⁵ Second, although the main push seems to be towards reduction of antipsychotic use, less is said on how to develop the resources that can provide good non-pharmacological approaches. Audits and targets should not solely focus on the quantity of antipsychotic use in dementia but also on the quality of non-pharmacological approaches available to this population. Long strides are required in this direction to improve behavioural and psychological symptoms of dementia care in the community, hospitals and care homes.

- 1 Treloar A, Crugel M, Prasanna A, Solomons L, Fox C, Paton C, et al. Ethical dilemmas: should antipsychotics ever be prescribed for people with dementia? *Br J Psychiatry* 2010; **197**: 88–90.
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Authors' reply: We are pleased to see support for our views in the responses received, in particular for the focus on rational debate in this area, rather than an 'either/or' debate. However, we would in reply air the following cautions.

Pattanayak claims that antipsychotics may not be as harmful as we have been led to believe. We would urge caution here. The data produced so far do suggest a reliable and quantifiable degree of harm resulting from antipsychotic use. We do not think that it is truly reasonable, now, to suggest that these drugs may be harmless. But we would also argue that Pattanayak's desire to claim a lack of harm is unnecessary. If they are harmful, their use may be justified under the doctrine of double effect by balancing the likely benefits against harm. It is our clinical experience that the discussions of benefit versus harm with relatives and advocates, informed by the principle of double effect, are effective and well understood.

Finally, we note what is, perhaps, an important slip of the pen in Sekhri's letter. He uses the term non-pharmacological as shorthand for alternatives to antipsychotics. He is correct in saying that there are multiple causes of behavioural and psychological symptoms of dementia. But if we describe all alternatives to antipsychotics as non-pharmacological, we may forget the appropriate treatment of physical illness with analgesics or depression with antidepressants. Alternatives to antipsychotic use in distress include both pharmacological and non-pharmacological approaches. One of us remembers replacing haloperidol for behavioural and psychological symptoms of dementia with effective treatment for scabies. Aromatherapy would have done little here! The term 'non-pharmacological' may therefore distract clinicians and compound a paucity of response to a complex problem.

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Interventions for self-harm: are we measuring outcomes in the most appropriate way?

Kapur and colleagues provide a brief review of contact-based interventions for self-harm and note their continuing appeal, despite largely unconvincing trial results.¹ The question the authors should have posed is not 'How might these interventions work?' but 'Why, when participants report that the interventions are of benefit, are trial results so unconvincing?'

The importance of self-harm lies in its strong association with suicide. The ultimate aim of interventions in this area is to prevent suicide, but the rarity of suicide makes it difficult to use as an outcome measure. Of those studies reviewed by Kapur *et al*, only two used death as an outcome.^{2,3} The remainder used repetition of self-harm, which is the best available proxy measure.⁴

Measuring repetition of self-harm is problematic. Hospital-treated episodes represent the standard measure but fail to capture the true pattern of self-harming behaviour, most of which occurs in secret and does not result in hospital presentation. Those who repeatedly self-harm avoid accident and emergency (A&E) departments at all costs and, when forced by the severity of their injuries to present, are adept at concealing the self-inflicted nature of those injuries, resulting in possible miscoding of visits. There is a need for a reliable, user-designed self-report instrument and a better understanding of the relationship between acts of self-harm and hospital visits.

Hospital-treated episodes do not provide a measure of reduction in self-harm; only a measure of reduction in clinical encounters for self-harm. It is debatable whether reducing clinical encounters is a beneficial outcome for this highly vulnerable and hard-to-reach population (repeat self-harmers). Reducing the number of hospital presentations may cut service costs in the short term; it may not save lives.

In a recent pilot study of a text-messaging intervention for self-harm,⁵ we had an interesting case. One of the participants reported during the trial that the intervention (a text message) had saved their life by interrupting a suicide attempt and prompting them to call for help instead of taking an overdose. They were conveyed to A&E and treated for very severe lacerations. Partly as a result, their visits to A&E increased during the 6-month pilot trial compared with 6 months prior to entry: a negative result using hospital-treated episodes as a measure. Two further participants told us that a suicide attempt had been interrupted by the timely arrival of a text message and begged to be allowed to continue to use the intervention at the end of the trial, yet standard reporting of the results of the study would not provide convincing evidence of effectiveness.

So why are trial results unconvincing, despite qualitative evidence to the contrary? The low status of qualitative data is one possible reason. Another is that we are measuring outcomes in inappropriate ways. We do not yet understand what outcomes are important to those who engage in repeated self-harm, nor how best to measure them.

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