regime used in our trial. However, it is worth noting that similarly high drop-out rates have been recorded in other well-regarded trials of antidepressant treatment. For example, in the National Institute of Mental Health trial, the drop-out rate from imipramine treatment was 33% over 18 months (Jacobson & Hollon, 1996).

Dr Ogundipe's view about intention-to-treat analysis is contrary to current statistical opinion and the recommendations of the *British Medical Journal* for the reporting of clinical trials (Altman, 1996; Schulz, 1996). The comment about baselines is not relevant here, since subjects who comply may fare differently and in an unpredictable way from those who do not comply. Thus, any observed differences between groups constructed in this manner may be due not to treatment but to factors associated with compliance. In this study, patients who dropped out were younger and had higher depression scores than those who completed the trial. The method of analysis should be consistent with the experimental design of a study. For randomised trials, such consistency requires the preservation of the random treatment assignment. Because methods that violate the principles of randomisation are susceptible to bias, they should not be used.

An analysis of the number needed to treat may be a sensible suggestion in general, although for a number of technical reasons it is not popular among statisticians (see Hutton, 2000). In any case, in this trial the high drop-out rate from the medication group would make the results of such an analysis suspect.

Marital discord was assessed using the Dyadic Adjustment Scale. As shown in Table 1 in the paper, the two treatment groups did not differ on this score, making it unlikely that this variable confounded the results.

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Citalopram-induced bruxism

There have been several reported incidents of iatrogenic bruxism (involuntary clenching or grinding of the teeth). These have involved diurnal bruxism (Micheli *et al*, 1993), felt to be associated with dopaminergic blockade, and nocturnal bruxism. Nocturnal bruxism has been reported with venlafaxine, a serotonin/noradrenaline reuptake inhibitor, which responded to gabapentin (Brown & Hong, 1999), as well as three selective serotonin reuptake inhibitors (SSRIs), paroxetine (Romanelli *et al*, 1996), fluoxetine and setraline (Ellison & Stanziani, 1993). In both reports the SSRI-associated bruxism was treated with buspirone.

I report two cases of nocturnal bruxism secondary to the SSRI citalopram, a previously unreported adverse effect. One patient was started on citalopram 20 mg/day. After 6 weeks the dose was increased to 40 mg. Ten days later nocturnal bruxism developed to such an extent that extraction of a molar was required. Buspirone was started and the bruxism ceased.

Another patient with panic disorder and moderate depression with somatic symptoms was referred to the clinic. The existing medication was a tricyclic and buspirone. Subsequent to non-response, medication was changed to citalopram, eventually reaching 40 mg/day. After an improvement in mood a behavioural programme was used to treat his anxiety symptoms. Four months into the programme the buspirone was reduced from 10 mg twice daily to none. Three weeks later he reported nocturnal bruxism. This ceased after reducing the citalopram to 20 mg/day. Thus, in this case, occult nocturnal bruxism was revealed by the reduction of a treatment agent.

These cases highlight that nocturnal bruxism can occur in response to any of the SSRIs, and that induction may be dose-dependent. They add to the literature suggesting that nocturnal bruxism can be treated with buspirone.

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Psychological debriefing – does it never work?

Mayou *et al* (2000) conclude in their 3-year follow-up study of road traffic accident victims that psychological debriefing is ineffective and has, in fact, adverse long-term effects. The intervention group reported significantly worse outcome at 3 years in terms of more severe psychiatric symptoms, impact of event symptoms, anxiety, depression, obsessive–compulsive problems and hostility, pain, major chronic health problems and financial problems. The findings support the suggestion that routine use of psychological debriefing among trauma victims should be discontinued (Bisson *et al*, 1997).

However, this conclusion is premature. A most serious problem in previous research is that the term psychological debriefing has been used for different types of interventions, for example, in terms of number of sessions and individual or group debriefing. Mayou *et al* offered individual one-session intervention, without any follow-up. This kind of intervention is contrary to most clinical thinking: first, assess the trauma; second, offer treatment accordingly. Nobody would recommend that all victims of traffic accidents should be given a standard surgical procedure of 15 minutes in the operating room. For patients with major traumas, the results may be worse than having no operation. The conclusion based on such an approach might easily be that surgery after traffic accidents should not be performed.

A flexible and individual approach is a much more reasonable and appropriate strategy (Rose *et al*, 1999). Future studies of psychological debriefing should use an individualised design including screening of psychopathology before intervention, if any, is offered. To assess the effect of one session of debriefing, only subjects who are likely to benefit from such a limited intervention should be included (i.e. those who are at greatest risk for post-traumatic stress disorder should be excluded).

The Impact of Event Scale scores for patients with high initial scores was 25.9 *v.*

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