Assessing quality of life in schizophrenia

Reading Wilkinson *et al* (2000), I felt that their new questionnaire tends to measure symptoms rather than quality of life. Obviously the symptoms and side-effects scale measures symptoms but a number of items in the other two scales measure symptoms as well, for example, "I lack the energy to do things". The relationship between symptoms and quality of life is complex but issues such as the availability of money or quality of accommodation must have something to do with it and neither is covered in their questionnaire.

The authors state that in measuring quality of life the measure has to be subjective, which makes sense, but whether it has to be self-reported is questionable. The authors suggested greater honesty might be outweighed by the disadvantage that no help is available if there is confusion regarding an item. I can see no advantages in using this new tool over existing tools, such as the Manchester Short Assessment of Quality of Life (Priebe *et al*, 1999), which is similarly short and useful in clinical practice.

Priebe, S., Huxley, P., Knight, S., *et al* (1999) Application and results of the Manchester Short Assessment of Quality of Life (MANSA). *International Journal of Social Psychiatry*, **45**, 7–12.

Wilkinson, G., Hesdon, B., Wild, D., *et al* (2000) Self-report quality of life measure for people with schizophrenia: the SQLS. *British Journal of Psychiatry*, **177**, 42–46.

M. P. Klijnsma Mid Essex Community & Mental Health Trust, The Linden Centre, Woodlands Way, Chelmsford CM1 7LF

Authors' reply: The Schizophrenia Quality of Life Scale (SQLS) is a measure of quality of life based on statements made by people with schizophrenia. We asked participants to state how their quality of life was affected by their mental health and the result was a mixture of symptoms, side-effects and psychosocial issues. Naturally, there is an overlap in the use of these terms, but our method elicits responses related to quality of life from a patient's perspective.

We believe that nobody can know their quality of life better than the person him- or herself. We have no reason to believe, on the basis of our findings, that people with schizophrenia are incapable of accurately representing their quality of life using the SQLS. It is of interest that we found that the people we interviewed voiced a variety of concerns, albeit not about possible financial and accommodation problems, which are, in any event, objective issues.

The measurement of quality of life is in the scientific domain and different methods, whether by questionnaire or interview, need to demonstrate reliability and validity. Thereafter, the choice of instrument requires a trade-off, representing a decision about the best instrument for a particular purpose. Factors such as ease of use, acceptability and cost must be considered.

In contrast to measures such as the Manchester Short Assessment of Quality of Life (MANSA), the SQLS has been specifically developed from interviews with people with schizophrenia, as opposed to being based on other measures. It was also developed as a measure for use in clinical trials and other research studies, as opposed to the assessment of community programmes, which is the case with the Lancashire Quality of Life Profile (Oliver *et al*, 1996), the measure from which the MANSA was derived. We would emphasise that the administration procedures contained in *The User Manual for the SQLS* (available from Oxford Outcomes) recommend, among other things, that the SQLS is completed in the presence of the researcher or is administered as an interview if the patient has difficulty with self-completion.

The SQLS has been very well received by colleagues both in the UK and internationally and the development of the instrument is continuing. It is clear to us from the response so far that the SQLS is recognised to fulfil a significant clinical and research need in relation to the assessment of quality of life in people with schizophrenia.

Oliver, J. P. J., Huxley, P. J., Bridges, K., *et al* (1996) *Quality of Life and Mental Health Services*. London: Routledge

D. Wild Oxford Outcomes, Bury Knowle Coach House, North Place, Old High Street, Headington OX3 9HY

G. Wilkinson University Department of Psychiatry, Royal Liverpool University Hospital, Liverpool

Is couple therapy better than antidepressant drugs?

The conclusion that couple therapy "is much more acceptable than antidepressant drugs" is not based on the findings of this study (Leff *et al*, 2000). To generalise results of a trial on desipramine, a tricyclic antidepressant (followed by trazodone and fluvoxamine), to antidepressant drugs as a whole is misleading. Other antidepressant drugs may be more acceptable than the trial drugs and the conclusions should have been limited to the drugs used.

Intention-to-treat analysis can sometimes be taken to the extreme. In this study, the majority of subjects in the antidepressant group (56%) did not receive the antidepressant as randomised and therefore it is not surprising that the antidepressant group did poorly. It would have been interesting to see, in addition, the results of an explanatory analysis that would have shown the outcomes for those who actually received antidepressant drugs compared with those who actually received couple therapy. Although the study would, technically, no longer be a randomised trial, this would not be a problem because the baseline comparison shows that the two groups are comparable, the essence of randomisation. It is not always the case that intention-to-treat analysis is better than explanatory analysis.

Although comparatively new, the number needed to treat is now widely understood as a simple and meaningful analysis of trials. What is the improvement rate attributable to couple therapy in this trial and how many patients will need to be treated for one more patient to gain improvement with couple therapy over desipramine?

Finally, the effect of an important confounding variable has not been discussed. The results may have been confounded by marital discord that will respond better to couple therapy than to antidepressants.

These must be considered before recommending couple therapy over antidepressant drugs in people with depression living with partners.

Leff, J., Vearnals, S., Brewin, C. R., *et al* (2000) The London Depression Intervention Trial. Randomised controlled trial of antidepressants *v.* couple therapy in the treatment and maintenance of people with depression living with a partner: clinical outcome and costs. *British Journal of Psychiatry*, **177**, 95–100.

L. O. Ogundipe Lyme Brook Mental Health Centre, North Staffordshire Combined NHS Trust, Bradwell Hospital, Talke Road, Newcastle-under-Lyme, Staffordshire ST5 7TL

Authors' reply: Dr Ogundipe is, of course, quite right in pointing out that our conclusion about the greater acceptability of couple therapy compared with antidepressant drugs can only apply to the particular

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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.

Altman, D. G. (1996) Better reporting of randomised controlled trials: the CONSORT statement. *British Medical Journal*, **313**, 570–571.

Hutton, J. L. (2000) Number needed to treat: properties and problems. *Journal of the Royal Statistical Society, Series A*, **163**, 403–419.

Jacobson, N. S. & Hollon, S. D. (1996) Cognitive–behavior therapy versus pharmacotherapy: now that the jury's returned its verdict, it's time to present the rest of the evidence. *Journal of Consulting and Clinical Psychology*, **64**, 74–80.

Schulz, K. F. (1996) Randomised trials, human nature, and reporting guidelines. *Lancet*, **348**, 596–598.

J. Leff, B. Everitt Institute of Psychiatry, King's College London, Denmark Hill, London SE5 8AF

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.

Brown, E. S., Hong, S. C. (1999) Antidepressant-induced bruxism successfully treated with gabapentin. *Journal of the American Dental Association*, **130**, 1467–1469.

Ellison, J. M. & Stanziani, P. (1993) SSRI-associated nocturnal bruxism in four patients. *Journal of Clinical Psychiatry*, **54**, 432–434.

Micheli, F., Fernandez Pardal, M., Gatto, M., *et al* (1993) Bruxism secondary to chronic antidopaminergic exposure. *Clinical Neuropharmacology*, **164**, 315–323.

Romanelli, F., Adler, D. A. & Bungay, K. M. (1996) Possible paroxetine-induced bruxism. *Annals of Pharmacotherapy*, **301**, 1246–1248.

M. E. J. Wise Paterson Centre for Mental Health, 20 South Wharf Road, London W2 IPD

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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