Social anxiety in patients with facial disfigurement

Newell & Marks (2000) highlight an important, under-researched area. They suggest treatment using cognitive–behavioural therapy, concentrating on exposure to avoided situations. However, their conclusions and recommendations appear broader than the data support.

They recruited from dermatology clinics, ex-surgery lists and media adverts. Their sample might therefore be expected to include subjects with less severe disfigurement or even a primary diagnosis of dysmorphophobia. The preponderance of women requires explanation when many conditions causing facial disfigurement affect both genders equally. The nature and severity of disfigurement should be described. Facial disfigurement can result from bone deformity, scarring, muscular paralysis or abnormal movement. It may be congenital or acquired and onset can be sudden or gradual at any age. Aetiology includes trauma, surgery, neoplasms and infections and likelihood of recovery varies greatly. All these factors are likely to influence the psychological difficulties experienced, including those manifest in social settings. Exposure would only be expected to help the phobic components of these problems.

Impaired control of functions important in social situations – including eating, drinking, speaking and facial expression – and altered self-image and differences in the reactions of others are likely to require changes to the routine advice given to people with social phobia. Repeated exposure to distressing events such as dribbling may reinforce negative thoughts about the self rather than minimise anxiety, as in typical phobic states. Specialist advice regarding make-up has improved patients' confidence and mood (Kanzaki *et al*, 1998), which would be expected to aid social interaction despite helping patients 'avoid' their true appearance.

Newell & Marks' study, therefore, does not support the conclusion that all social anxiety in patients with all types of facial disfigurement has the same psychopathology as social phobia. Cognitive–behavioural interventions probably need to address more than the avoidance or beliefs typical of social phobia. The need for exposure, a range of cognitive techniques, grief work, specialist physiotherapy and speech therapy is likely to vary. Future research should describe the type, course and severity of disfigurement and associated difficulties and clarify specific concerns occurring in social settings.

Kanzaki, K., Ohshiro, K. & Abe, T. (1998) Effect of corrective make-up training on patients with facial nerve paralysis. *Ear, Nose, and Throat Journal*, **77**, 270–274.

Newell, R. & Marks, I. (2000) Phobic nature of social difficulty in facially disfigured people. *British Journal of Psychiatry*, **176**, 177–181.

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Authors' reply: We welcome Dr Butler's comments and endorse the call for further research in the area of psychological difficulties following disfigurement. While we accept the call for caution, given the heterogeneous nature of our sample, we believe our conclusions are appropriately modest. We do not suggest either that phobic avoidance is the sole element of psychological distress following disfigurement, or that "all social anxiety" in such people has the "same psychopathology as social phobia". Indeed, the role of multiple contributing factors to such distress has been emphasised by one of us (Newell, 1991), and we noted our awareness of, but inability to investigate, the role of stigma. Moreover, Newell (1991) stresses the importance of sensitivity when advising of exposure exercises, precisely to reduce the potential for reinforcing negative thoughts and increasing anxiety, as Dr Butler suggests. More generally, the need for individualised treatment has been repeatedly stressed in behaviour therapy and cognitive therapy, although self-help methods (which, of necessity, give general prescriptions of advice which clients modify themselves) show promise. In the context of disfigurement, a simple self-help leaflet (Newell & Clarke, 2000) produced modest benefits relative to untreated controls and of a level roughly similar to those found in a group social skills intervention (Robinson *et al*, 1996).

Although the nature of the sample is important, it is difficult to obtain participants from this group, as previous studies have found. However, findings regarding gender differences among disfigured people with respect to psychological disturbances have been equivocal, and findings tend to suggest that level of disfigurement is a poor predictor of psychological adjustment.

Exposure therapy is obviously not a panacea, but rather a promising approach to psychological disturbance following disfigurement where social anxiety is present. Dr Butler rightly draws attention to the need for flexible, individually tailored treatment, although this is questioned by some results (Schulte *et al*, 1992). There is likewise a need to avoid the inclusion of poorly supported interventions, and to build an appropriate evidence base. For example, we know of no studies that demonstrate the effectiveness of grief work among people with disfigurement, and there is likewise little evidence of the effectiveness of other interventions for psychological difficulties following disfigurement, despite the size of the problem.

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Schulte, D., Kunzel, R., Pepping, G., *et al* (1992) Tailor-made versus standardized therapy of phobic patients. *Advances in Behaviour Research and Therapy*, **14**, 67–92.

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Outcome of hospital-treated depression

> " 'Then you should say what you mean', the March Hare went on. 'I do,' Alice hastily replied; 'at least – I mean what I say – that's the same thing, you know.' " (Lewis Carroll, *Alice in Wonderland*).

Tuma (2000) gives a 'recovery' rate of 24% for depressed elderly patients but this figure actually refers to his category 'lasting recovery'. If the term recovery may be used to include those who have relapsed but then recovered for a (specified) period of time, the rate from Tuma's study is 44%, or 66% once natural deaths have been removed. Tuma appears to be following Murphy's (1987) view of recovery in depression as being a pint-pot only half-full.

This is not to disagree with the conclusion drawn from the study's data that elderly patients with depression have a poorer prognosis than younger adults, but there is a need to respond to the call for more clarity, if not unanimity, in what terms mean (Frank *et al*, 1991). Low detection and treatment rates for depression in

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older patients in the community are not likely to be improved by 'term'-inally induced therapeutic nihilism. If we are to avoid such confusion, we should heed the words of another Lewis Carroll character:

> " 'When I use a word,' Humpty Dumpty said in a rather scornful tone, 'it means just what I choose it to mean – neither more nor less.' "

Frank, E., Prien, R. F., Jarrett, R. B., *et al* (1991) Conceptualisation and rationale for consensus definitions of terms in major depressive disorder. Remission, recovery, relapse, and recurrence. *Archives of General Psychiatry*, **48**, 851–855.

Murphy, E. (1987) The prognosis of depression in old age (letter). *British Journal of Psychiatry*, **150**, 268.

Tuma, T. A. (2000) Outcome of hospital-treated depression at 4.5 years. An elderly and a younger adult cohort compared. *British Journal of Psychiatry*, **176**, 224–228.

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Anorexia nervosa: treatment with olanzapine

Anorexia nervosa is a multiply determined disorder of unknown aetiology. Restriction of food intake culminating in profound emaciation is considered to be pathognomic (Kaye *et al*, 1999). One of the diagnostic criteria for anorexia nervosa is body image disturbance that is characterised by feeling and judging oneself to be fat and by claiming to 'see' oneself as fat despite being underweight. The bizarre body self-image in anorexia nervosa can be regarded as a psychotic way of thinking. We tried to treat anorexia nervosa as a psychotic disorder with olanzapine. Hansen (1999) has previously reported a case of treatment of anorexia nervosa with olanzapine; here we report three further cases.

A 50-year-old woman with anorexia nervosa since she was 17 years old applied for treatment in our clinic with an initial weight of 34 kg and height of 157 cm. By that time she had already been treated with antidepressants, traditional neuroleptics and psychotherapy, without success. She was commenced on olanzapine, 5 mg daily. The first 2 months were difficult because she had to accept the side-effects of hunger and weight gain. After 2 months she stopped talking about being overweight; according to her drawings, her body self-image changed towards normality. Now she weighs 53 kg and feels herself completely healthy. She still receives 5 mg olanzapine daily.

A 30-year-old woman with anorexia nervosa since she was 18 years old additionally developed bulimia nervosa 5 years before presentation to our clinic. She was treated traditionally for many years without significant positive results. When she applied to our clinic her weight was 44 kg, height 167 cm. Mental state examination revealed severe body self-image disturbance. We commenced her on 5 mg olanzapine daily. In 9 months her weight was 53 kg, and she recovered from symptoms of both anorexia nervosa and bulimia nervosa. According to her drawings her body self-image changed from a fat little girl to an attractive grown-up woman. She still receives 5 mg olanzapine daily.

A 34-year-old woman had been suffering from anorexia nervosa and borderline personality disorder since puberty. When she came to our clinic her weight was 60 kg, height 180 cm. She complained of confusion, having too many thoughts in her head at one time. She had a seriously disturbed body image – in her drawings she looked like a little fat girl without hands and secondary sexual signs. She was started on olanzapine 5 mg, and after 2 months her body image has improved (she now sees herself as a grown-up woman) and she feels healthy.

In all three cases olanzapine was well tolerated. Our patients restored their body weight and appetite as well as their body self-image. They now think of themselves as normal, mature adults. The problem is to convince the patient to start and to continue with olanzapine therapy within the first 2 months, because it takes a few weeks before a full antipsychotic effect is achieved. We think that further investigations in this area should take place.

Hansen, L. (1999) Olanzapine in the treatment of anorexia nervosa (letter). *British Journal of Psychiatry*, **175**, 592.

Kaye, W., Strober, M., Stern, D., *et al* (1999) New directions in treatment research of anorexia and bulimia nervosa. *Biological Psychiatry*, **45**, 1285–1292.

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Nicotine reduction: effectiveness of bupropion

Hayford *et al* (1999) and others have found sustained-release bupropion useful as part of nicotine reduction treatment programmes. However, our clinical experience suggested the primary benefit of sustained-release bupropion (300 mg/day) occurred within the first month of treatment and the recommended second month of medication was probably not helpful.

We evaluated the treatment progress of 74 (30 one-month, 44 two-month) research volunteers (further details available from the first author upon request). Treatment condition assignment was random but one-month patients had the option of an additional two months of medication if they were not successful at quitting at immediate follow-up, thus decreasing the number of one-month participants for this study. Volunteers had a mean age of 56 and were primarily male (91%), cigarette smokers (96%) and White (92%). One- and two-month groups did not differ significantly on a number of treatment motivation measures, demographic variables and self-reported health variables.

All patients signed consent forms and received the same behavioural treatment information in the first session. All were also instructed to return to a second meeting at which time their progress was evaluated and they received more behavioural strategy information and their assigned sustained-release bupropion. Participants were followed (a) as soon as convenient after nicotine quit date, (b) three-months after immediate follow-up, and (c) six-months after initial group meeting. About 80% of potential subjects volunteered for this project and follow-up return rate averaged 81%.

Use of one or two months of bupropion did not significantly affect self-reported quit rates at immediate, three-month or six-month follow-up periods. Self-reports of decreased nicotine intake among patients who did not quit entirely also did not differ between one- and two-month groups at immediate, three-month, or six-month follow-up periods. Only weight gain was associated with six-month treatment success. Participants who were successful reported more weight gain than those who were not. However, one- or two-month dosing schedule was not significantly associated with reported weight changes. Quitting success at six months was not related to age, gender, tobacco type, income category, race, combat exposure, years' service, side-effect ratings, health problem ratings concerning breathing, heart, general medical, psychiatric, or substance dependence areas, religious behaviour, immediate weight gain, or initial self-ratings of treatment programme helpfulness/motivation/self-control/completion.

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