Correspondence

EDITED BY MATTHEW HOTOPF

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Hospitalisation and adolescent anorexia nervosa

Gowers *et al*'s (2000) study of the impact of hospitalisation on the outcome of anorexia nervosa in adolescence is a useful and important contribution to a debate that is difficult to resolve meaningfully, because of lack of useful evidence. As the authors note, randomised controlled trials are both lacking and extremely difficult to perform, for both practical and ethical reasons. However, the significant mortality and morbidity associated with these disorders is such that this problem must not be ignored.

Although the paper raises some very important questions, we are concerned that the suggestion that in-patient treatment is associated with a poor outcome is premature, and may be taken by some to mean that in-patient treatment should not be considered. This view would be particularly worrying if adopted by cash-strapped health authorities that are already often reluctant to finance treatment of what is still sometimes seen as a trivial condition.

We believe that three questions need to be answered before making any general pronouncement on the appropriateness of in-patient treatment; (a) what factors lead to admission? (b) what is the relationship between these factors and outcome? and (c) what constitutes in-patient treatment, and is it a uniform concept?

Our experience of over 500 admissions of young people suffering from anorexia nervosa leads us to the view that many of the factors which lead to admission, but which are also predictive of poor outcome, are systemic. They will not therefore be measured by the Morgan–Russell Assessment Schedule (Morgan & Hayward, 1988) or other individual-based predictor variables. Such systemic variables include major psychosocial stresses within the family, and the health and strength of the professional network, but we have found it hard to find instruments that adequately measure these factors.

In other words, the measures used to assess severity in this study are all individual to the patient and do not sufficiently take account of the complex network of relationships within which anorexia nervosa takes root and either flourishes or dies. In our experience, the severity of symptoms such as weight loss does not bear a linear relationship to outcome because of highly complex intervening contextual variables, which need to be addressed by any outcome study.

We certainly share the view that in-patient treatment is not the only response, and that we need to be continually reflecting on the style and content of such treatment. However, we think it highly premature to conclude that it should be discouraged. It should be remembered that at present it is often a life-saver for many young people who are seriously ill.

Gowers, S. G., Wheetman, J., Shore, A., *et al* (2000) Impact of hospitalisation on the outcome of adolescent anorexia nervosa. *British Journal of Psychiatry*, **176**, 138–141.

Morgan, H. G. & Hayward, A. E. (1988) Clinical assessment of anorexia nervosa: the Morgan–Russell Outcome Assessment Schedule. *British Journal of Psychiatry*, **152**, 367–371.

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Author's reply: We are grateful to Drs Wood & Flower for contributing to the debate on treatment setting in adolescent anorexia nervosa. Our aim was indeed to open rather than close discussion.

We agree that it is of paramount importance that anorexia nervosa is seen for the serious condition with high morbidity and mortality that we know it to be, rather than the trivial disorder sometimes portrayed by the media. It is right, however, for commissioners to expect an evidence-based case for expensive treatments. Despite the questions raised in our paper, our service treats a large number of adolescents as in-patients and continues to make and support significant numbers of referrals to specialist eating disorder in-patient services.

Nevertheless, it is extraordinary that the following questions are so rarely addressed:

(a) Could it be that in-patient treatment has negative (side-)effects?

(b) Could there be some intrinsic features of anorexia nervosa, such as ineffectiveness, low self-esteem or past history of abuse, which might make those with anorexia nervosa particularly vulnerable to these negative effects?

(c) Might these negative effects sometimes outweigh the benefits?

We would take these questions for granted in evaluating a new drug therapy.

The point Drs Wood & Flower make about systemic factors as predictors of outcome is an important one that our group has previously researched (Gowers & North, 1999). Where there is family or social difficulty, however, does this mean that the adolescent is better treated within or outwith the family home? Does this difficulty add to the case for admission or the case against? In view of the high rates of relapse after weight restoration in hospital, we contest that one could form testable hypotheses either way.

The National Health Service Executive has rightly judged that further evidence of the effectiveness of treatment in different settings is required. We are pleased to report that our group was awarded a Health Technology Assessment grant to conduct a randomised controlled trial of treatment setting covering the north-west of England. We hope in the course of the 4-year pragmatic study to contribute to the debate on when specialist eating disorder in-patient units may be helpful and for whom. We are also examining family satisfaction and acceptability. Of course, this large study will not provide the last word on the issue, but we must avoid the negativism which suggests it is better not to carry out research in case the results are misinterpreted.

Almost certainly in-patient admission sometimes saves lives. Nevertheless, almost all series show high rates of relapse after discharge (Crisp *et al*, 1991; Eisler *et al*, 1997) and however loaded with poor

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prognostic features our series may have been, a good outcome for 3 out of 21 is very poor.

Crisp, A. H., Norton, K., Gowers, S., *et al* (1991) A controlled study of the effect of therapies aimed at adolescent and family psychopathology in anorexia nervosa. *British Journal of Psychiatry*, **159**, 325–333.

Eisler, I., Dare, C., Russell, G. F. M., *et al* (1997) Family and individual therapy in anorexia nervosa. A 5 year follow-up. *Archives of General Psychiatry*, **54**, 1025–1030.

Gowers, S. G. & North, C. (1999) Difficulties in family functioning and adolescent anorexia nervosa. *British Journal of Psychiatry*, **174**, 63–66.

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Possible causes of catatonia in autistic spectrum disorders

I read with interest the paper by Wing & Shah (2000) on catatonia in autistic spectrum disorders. The authors quite correctly make the point that catatonia, although a useful clinical concept, is a description of a number of behaviours. However, they have not attempted to investigate the aetiology of catatonia in their sample of 40 patients. Three other possible causes for their observations spring to mind.

First, the onset of catatonic symptoms in adolescence or early adulthood, in this largely male sample, could be related to the development of schizophrenia, although it may be difficult to diagnose. It has presumably been excluded as no patients had first-rank symptoms according to the accounts of relatives or carers, although in Table 3 (p. 359), the heading "bizarre/psychotic" catatonic manifestations were found in 40% of their patients. The fact that 'Others had occasional visual hallucinations or paranoid ideas' suggests that they may qualify for an additional diagnosis of schizophrenia according to the ICD–10 (World Health Organization, 1992). The authors have not specifically stated whether the patients had been assessed for a diagnosis of schizophrenia.

Although the patients may be difficult to interview on account of communication disorders or cognitive problems, nearly half did not have impaired language and the number of mute patients is not stated. Furthermore, 70% of the patients had a level of cognitive ability within the range from mild learning disability to average intellectual ability, not incompatible with a diagnosis of schizophrenia.

Second, the possible explanation for catatonic symptoms is the development of an affective disorder. In 13 of the 30 patients, precipitating factors included bereavement, pressure at school, lack of structure after leaving school and lack of occupation, which are more commonly associated with a depressive illness. Central to the diagnosis of catatonia are increased slowness, difficulty in initiating and completing actions and lack of motivation, among others, possibly symptoms of depression.

Third, and most importantly, catatonic symptoms may be difficult to distinguish from the extrapyramidal side-effects of antipsychotic drugs (American Psychiatric Association, 1992). In Wing & Shah's description of the criteria for catatonia, a secondary feature listed was "Parkinsonian features: tremor, eye-rolling, dystonia, odd stiff posture, freezing in postures, etc.". Although the patients are fairly young, they are also a tertiary referral group and it is likely that they would have received other, previous treatments. Recent estimates of prescriptions of psychotropic medication to adolescents and adults with developmental disabilities vary from 12 to 40% (Connor & Posever, 1998). There was no record of previous treatment and, more specifically, a history of current or prior exposure to antipsychotics is omitted.

It is helpful to know that catatonia can complicate autistic spectrum disorders and that individuals who present with catatonia may have an undiagnosed autistic spectrum disorder. However, although recognition is necessary to institute appropriate management, this paper offers only limited help in this direction. There would have been a greater clinical impact if it had addressed the possible causes of catatonia or the other associated psychopathology. The study also raises the question of whether catatonia represents the expression of other, more common mental disorders in those with limited communication skills.

American Psychiatric Association (1992) Differential diagnosis of tardive dyskinesia. In *Tardive Dyskinesia: A Task Force Report of the American Psychiatric Association*, pp. 9–34. Washington, DC: APA.

Connor, D. F. & Posever, T. A. (1998) A brief review of atypical antipsychotics in individuals with developmental disability. *Mental Health Aspects of Developmental Disabilities*, **1**, 93–102.

Wing, L. & Shah, A. (2000) Catatonia in autistic spectrum disorders. *British Journal of Psychiatry*, **176**, 357–362.

World Health Organization (1992) *The ICD–10 Classification of Mental and Behavioural Disorders*. Geneva: WHO.

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Authors' reply: Dr Chaplin notes that neither the possible causes nor the treatment of catatonia were discussed in our paper. As the *Journal* requires papers to be 3000–5000 words long, we decided to focus on the clinical picture of catatonia in autism and its prevalence. We have written and intend to publish a second paper dealing with causes and treatment and are grateful to Dr Chaplin for providing us with the opportunity to write a few more words on these subjects.

The individuals in the study had all been seen by one or more clinicians before the tertiary referral to Elliot House. During the course of the multiple assessments, possible underlying causes, including schizophrenia, depression, obsessive–compulsive disorder and identifiable brain pathology such as parkinsonism, would have been considered. These conditions, together with autistic spectrum disorders and catatonia, are defined and diagnosed only on history and clinical picture and there is overlap of clinical features among them all. In the individuals in our study, the developmental history and clinical picture, including the "bizarre/psychotic" behaviour in some people, fitted best with autistic spectrum disorders. We do not argue that psychiatric conditions, such as schizophrenia, cannot occur in association with autistic disorders. The point of our paper is that catatonia can occur as a complication of autistic spectrum disorders alone.

Twenty-one individuals in our study had received psychotropic medication for possible psychiatric conditions, and two people were treated with electroconvulsive therapy, all without useful effect on the catatonic features. The side-effects of neuroleptic medication were considered as possible causes of the catatonia. Of the 21 individuals who were medicated 10 were given drugs only after the onset of catatonia. The temporal relationships were

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