*Leading article***Keep on taking the weedkiller?**

The central theme of primary anorexia nervosa is the individual's relentless pursuit of excessive thinness.¹ She, for it is more commonly a woman, exhibits a phobic avoidance of normal weight and the terms 'weight phobia'² has been coined to describe this. The consequence of this behaviour is to reverse the pubertal process resulting in a regression in a biological, and in many ways in a social and psychological sense as well, to a prepubertal state. The pituitary gland becomes unresponsive to stimulation by hypothalamic releasing factors to LH/FSH production.³ This behaviour results in the resolution of earlier adolescent emotional conflict and turmoil which now becomes condensed within the anorectic stance. It is thus protective and defensive and for this reason the vigorous dieting behaviour may be denied and even concealed by the patient. This renders it a difficult diagnosis to make. Nevertheless, it is a diagnosis to be made on positive grounds rather than by the exclusion of other disease.⁴ The diagnosis needs a careful history, including information from those around the patient. The avoidance of weight gain to a normal level will also show itself during treatment aimed at accomplishing this. The term 'anorexia nervosa', meaning nervous loss of appetite, is poorly chosen, for loss of appetite is not a central or necessary feature. Indeed the anorectic, like any other starving person, may at times be very hungry.

The physical features of the disorder are the consequences of weight loss resulting in emaciation. They are not specific to primary anorexia nervosa. They include secondary amenorrhoea and if weight loss is severe, cold red or blue extremities, bradycardia, low body temperature, the presence of lanugo hair, lowered metabolic rate, and restlessness. It is predominantly a state of carbohydrate starvation and severe hypoproteinaemia is uncommon, except in longstanding cases. The patients may be subdivided into those who exhibit a pattern of abstention from eating and those who give in to their hunger and eat, but subsequently induce vomiting, often secretly. The latter group, who may also take large quantities of purgatives and/or diuretics, may exhibit complications of electrolyte disturbance: hypokalaemia in particular.

Although we do not fully understand the causes of primary anorexia nervosa a number of premorbid constitutional factors have been defined which may contribute towards a predisposition to the illness. There is a tendency for primary anorexia nervosa to occur in later born children in a family, and for them to weigh more at birth.⁵ Some, however, are under average weight at birth. There is also a tendency for patients who develop primary anorexia nervosa to have been premorbidly overweight⁶ and to have had an early menarchal age.⁵

Crisp *et al*⁷ studied the insulin response to an intravenous injection of dextrose 25 g in groups of patients with primary anorexia nervosa, with obesity and in normal subjects. The anorexia nervosa and the obese

patients had a sustained high concentration of insulin in the peripheral blood in contrast with the normal subjects in whom the insulin concentrations fell more rapidly. Within the anorexia nervosa group this response persisted after restoration of body weight to normal and although the response had returned towards normal in a few patients after full recovery from the illness, in two cases the delayed insulin response persisted. Thus it is difficult to be sure whether this reflects a constitutional tendency in those vulnerable to primary anorexia nervosa, or whether it is a response specific to the disturbed physiology of the disorder. In this issue of *Gut*⁸ Stacher and his colleagues describe delayed gastric emptying in 13 of 16 patients with a diagnosis of anorexia nervosa compared with 24 control subjects. Intravenous domperidone accelerated the rate of gastric emptying in those patients in whom it was markedly delayed, but not when the delay was less marked. It is not possible without a follow up study to determine whether the delayed gastric emptying is a consequence of the illness, or whether it has aetiological significance. The former possibility is likely, as delayed gastric emptying occurs in normal subjects during self-starvation.⁹ Acute dilatation of the stomach and paralytic ileus can occasionally be present in primary anorexia nervosa, usually following a binge after a period of starvation, but sometimes on refeeding in hospital. Dally *et al*¹⁰ have reported an incidence of acute dilatation of the stomach of one per cent and in this group of patients, the delay in gastric emptying is clearly aetiological important with respect to this complication.

Stacher *et al*⁸ also report disordered oesophageal motility in 15 of 30 patients studied, who had been classified as suffering from primary anorexia nervosa. Seven were found to have achalasia of the lower oesophageal sphincter: four of these were treated by oesophageal dilatation and three responded with weight gain and relief of symptoms. Six patients were found to have high amplitude, long duration contractions of the middle or distal third of the oesophagus, two of whom improved with nifedipine and four did not. One patient had diffuse oesophageal spasm and one with a hypertonic and on swallowing, inappropriately short, upper oesophageal sphincter, was found to have gastro-oesophageal reflux with severe oesophagitis and was treated surgically. Dysphagia, spontaneous non-acidic vomiting and chest pain were the characteristic features of the group with disordered, in contrast with the group with normal, oesophageal motility.

Dysphagia and chest pain are not common characteristics of primary anorexia nervosa, nor is spontaneous non-acidic vomiting, although self-induced acidic vomiting is often present, as it was in those with normal oesophageal function. The patients with disturbed oesophageal motility clearly were not suffering from primary anorexia nervosa in the sense of weight phobia, but from anorexia secondary to other disease. This has been called secondary anorexia nervosa, which can arise in relation to physical, or psychiatric illness. Secondary anorexia nervosa has been described as a consequence of gastrointestinal disease¹⁰ and also in infectious and metabolic disorders. Within psychiatric illness it can occur in severe depression, or as a result of a psychotic refusal to eat. The physical consequences of such a state of starvation are the same as in primary anorexia nervosa and in the secondary anorexia nervosa group the diagnosis is confirmed by examination and further investigation. The

history will not be typical of primary anorexia nervosa, in which the patient's brightness, activity and lack of concern about her state are unlikely to be confused with organic disease.

As a result of their findings Stacher *et al*⁸ have suggested that all patients with suspected primary anorexia nervosa should have disordered oesophageal motility excluded as the basis for their illness. I disagree with this view and suggest that there are three levels which need to be evaluated to make a positive diagnosis of primary anorexia nervosa. The first level consists of the physical and behavioural characteristics of starvation and these are shared by the secondary anorexia nervosa group. The second level is crucial, for it forms the nub of primary anorexia nervosa. It consists of the individual's need to maintain a low body weight and her terror of weight gain. Although the patient may seek to conceal this, it is more likely to be revealed within an encounter with those staff who are clinically experienced in this area. The initial interview will contain the seeds of possible effective treatment. The third level requires an understanding of the specific basis for the need of the individual to avoid a biologically mature stance, in experiential, behavioural, and social terms.

Primary anorexia nervosa provides an excellent paradigm of a psychosomatic disorder. One may show interplay of social and cultural factors influencing dieting behaviour in an individual who has a constitutional predisposition to respond by developing the disorder, if circumstances within her psychological development and her social matrix render her vulnerable. Once the disorder is established, one sees the impact of the anorectic state on the individual's physiological and psychological state and the way it affects the social matrix and the family in particular. Both the psychosomatic aetiology of the disorder and its somatopsychic consequences become transparently clear. I accept that it may sometimes be a difficult diagnosis to establish if the patient seeks to conceal her state of weight phobia, or if symptoms arising from the complications of behaviour such as frequent self-induced vomiting or massive purgation, are prominent, but nevertheless it should be a positive diagnosis, rather than one of exclusion and other disease only needs to be excluded in atypical cases. The diagnostic criteria established by Feighner *et al* are unsatisfactory.^{10 11}

Now let me contrast this with a totally different situation. The conclusion that in those attending the gastroenterology clinic, the symptoms have a major functional component, is usually formed after the exclusion of organic disease, or the view that such disease does not adequately explain the symptoms. This accounts for about half the patients attending gastroenterology outpatients.¹² Let me take the irritable bowel syndrome as an example of a psychosomatic disorder, which can only be diagnosed with certainty after the exclusion of organic disease. After clinical examination and sigmoidoscopy in a patient complaining of abdominal pain and an irregular bowel habit, the essential investigations which must be normal to exclude an organic cause are a blood count, bacteriological examination of the faeces and an air contrast barium enema. It is important to emphasise that evidence of psychogenic aetiology should be based on a positive process, rather than solely on the exclusion of organic disease. This requires a detailed psychiatric case history and mental state examination.¹² The approach to treatment should be holistic, embracing the physical symptoms and any psychological disturbance, with the

emphasis shifting from one to the other as dictated by clinical features. Psychological aspects of treatment may include psychotropic drugs, psychotherapy, behaviour therapy, or a combination of these. In common with other chronic relapsing conditions, the physician needs to pay particular attention to the doctor-patient relationship. A positive and continuing relationship with a doctor who can provide support, encouragement and discussion is often very important. The physician needs to guard against personal feelings of frustration and failure which may be expressed in aggression towards, or even rejection of, a patient who fails to improve. Unfortunately these aspects of treatment are often ignored.

It may be very difficult to show the clear display and interaction of multiple factors so evident in primary anorexia nervosa in patients diagnosed as suffering from psychosomatic disorder in the gastroenterology clinic, but the goal should be to do so. An attempt should be made to evaluate the significance of life events, personality attributes, coping resources and the extent and quality of social support on the development and course of the illness.¹³ Sometimes it is not possible to show such factors and sometimes it is not possible to influence them even if they can be demonstrated. Under these circumstances it only remains possible to treat the symptoms. It is all too tempting, however, to adopt the easy way out by taking a scant history and accepting the symptoms on face value. In good clinical practice there can be no short cut to a detailed and careful evaluation of the patient as a whole. Sometimes there may be no alternative to prescribing the weedkiller, but sometimes it is the lazy option.

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References

- 1 Bruch H. *The golden cage: the enigma of anorexia nervosa*. Shepton Mallet, England: Open Books Publishing Co, 1978: 150.
- 2 Crisp AH. *Anorexia nervosa: let me be*. London: Academic Press, 1980: 200.
- 3 Palmer RL, Crisp AH, Mackinnon PCB, Franklin M, Bonnar J, Wheeler M. Pituitary sensitivity to 50 µg LH/FSH-RH in subjects with anorexia nervosa in acute and recovery stages. *Br Med J* 1975; **1**: 179–82.
- 4 Stonehill E. Psychiatric illness and the gut. In: Misiewicz JJ, Pounder R, Venables C, eds. *Diseases of the gut and pancreas*. Oxford: Blackwell. (In press).
- 5 Crisp AH. Reported birth weights and growth weights in a group of patients with primary anorexia nervosa (weight phobia). *J Psychosom Res* 1970; **14**: 23–50.
- 6 Crisp AH, Stonehill E. Relation between aspects of nutritional disturbances and menstrual activity in primary anorexia nervosa. *Br Med J* 1971; **3**: 149–51.
- 7 Crisp AH, Ellis J, Lowy C. Insulin response to a rapid intravenous injection of dextrose in patients with anorexia nervosa and obesity. *Postgrad Med J* 1967; **43**: 97–102.
- 8 Stacher G, Kiss A, Wiesnagrotzki S, Bergmann H, Höbart J, Schneider C. Oesophageal and gastric motility disorders in patients characterized as having primary anorexia nervosa. *Gut* 1986; **27**: 1120–6.
- 9 Keyes A, Brozek J, Henschel A. *The biology of human starvation*. Minneapolis: University of Minnesota Press, 1950: 587–600.
- 10 Dally P, Gomez J, Isaacs AJ. *Anorexia nervosa*. London: Heinemann Medical Books Ltd, 1979: 216.
- 11 Feighner JP, Robins E, Guze SB, Woodruff RA, Winokur G, Munoz R. Diagnostic criteria for use in psychiatric research. *Arch Gen Psychiatry* 1972; **26**: 57–63.
- 12 Stonehill E, Misiewicz G. The irritable bowel syndrome – a psychiatric view. *Medicine* 1980; **36**: 1850–3.
- 13 Bass C. Life events and gastrointestinal symptoms. *Gut* 1986; **27**: 123–6.