Leading Article

Gastrointestinal disturbance in anorexia nervosa

A.H. Crisp

Academic Department of Psychiatry, St George's Hospital Medical School, Cranmer Terrace, London SW17 0RE, UK.

Introduction

Anorexia nervosa would seem to be an attempt to cope with life. It is hard for the outsider to recognize that the physical and biological distortions which characterize it are, in fact, a sought-for and fiercely defended refuge. To this end the anorectic will conceal any of those aspects of her disorder the revelation of which would jeopardise her stance. It is small wonder that the condition is often missed altogether. It shares this distinction with other egosyntonic disorders such as alcoholism. With attention focussed on some inescapable, and sometimes rare, aspect of the condition, there is inevitably a fertile soil for the spawning of new and essentially falsely founded syndromes.

The correct diagnosis would seem to have several levels to it. Firstly, the behavioural expressive syndrome – often evident at a distance – of the emaciated young female with the stigmata of starvation and which will include endless preoccupation with food and her endless need to defend against ingesting it. Secondly, the presence of the underlying often denied but fundamental and apparently unnatural terror of weight gain and its attendant 'fatness', and thirdly, the nature of the adolescent maturational crisis which has precipitated out the disorder and which it seems is now, through it, being avoided (Crisp, 1980).

Severe anorexia nervosa is quite common, affecting about one in 200 of teenage females. The psychological mainsprings of the condition determine the various and profound disturbances of bodily function that arise and persist and nowhere is this more dramatic than within the gastrointestinal tract.

Ingestive patterns

The omnipresent task for the anorectic is to avoid calorie ingestion. Abstinence or restrained eating may predominate and from the anorectic standpoint this is the most effective technique if it can be achieved and

sustained. It may be facilitated by such means as eating relatively large quantities of protein and fibre, by drinking large quantities of acaloric fluid, e.g. water/black coffee (polydipsia can be a form of presentation), or by smoking – usually tobacco and cigarettes. If restraint gives way to endless bingeing, then vomiting will supervene if maintenance of the low body weight stance remains psychologically necessary. Such binge eating can become bizarre both in terms of volume and content. It can, for instance, include at one sitting a whole fresh cauliflower, a loaf of bread, half a pound of butter, several packets of biscuits, a whole gateau plus a pint of cream and sometimes such distressing objects as the patient's own faeces. The form of starvation now changes from being predominantly one of calorie depletion to being a more general protein/calorie/essential element depletion. The anorectic may well seek help actively at this stage for curbing of her appetite although this motive may not be explicit. Clinically, one is confronted by the problem of a restless person presenting with a voracious appetite coupled with weight loss and with a variety of metabolic disturbances, the latter depending on the (hidden) severity and chronicity of the anorexia nervosa and the particular ingestive style currently associated with it. The immediate bulimic episode may be denied. The differential diagnosis of such syndromes is, of course, complex. Meanwhile, the false assumption by the anorectic that any ingested food can be flushed through with purgatives, coupled with the inevitability of constipation as a feature can lead to massive purgative consumption, often up to 50 or more Senokot tablets or the equivalent per day. The reward is the dehydrating (body weight losing) effect and the anorectic is now trapped into the terror of experiencing major weight gain even when drinking acaloric fluids. Diuretic abuse may be the next step. It is these patterns that dictate much of the gastrointestinal tract pathology that can accompany anorexia nervosa, some of the more common of which will be referred to.

A.H. Crisp, M.D., D.Sc., F.R.C.P., F.R.C.Psych.

Received: 7 June 1984

Mouth

Dental decay – this can be profound in anorectics, especially those who binge and vomit (Hurst *et al.*, 1977). It would appear to be a function of the destructive effect on dental enamel of acidic vomit. Periodically high carbohydrate intake into the mouth may contribute to the severe dental caries sometimes seen, whilst the reduced and altered salivary secretions that accompany starvation will also render teeth more vulnerable to decay.

Swollen salivary glands – the parotids in particular may become tender and swollen and a source of great distress because of the fullness of the facies that this induces.

Stomach

Gastric motility is not greatly altered in many instances but stomach size can be profoundly affected. Abstaining anorectics develop small shrunken stomachs, evident radiologically and at autopsy and reflected in substantial reduction in functioning parietal cell mass (Crisp, 1970). Nevertheless, such an anorectic can readily enough begin to eat normally within treatment. The bingeing anorectic can develop a huge dilated stomach which becomes a chronic feature. The stomach may come to virtually occupy the abdomen when viewed radiologically or at autopsy. Over the years several such cases have been described since Russell (1966) importantly first drew our attention to the relationship. A case is described in this issue (Bckett, 1985) wherein the dilatation appeared to follow two weeks or so of refeeding and it is suggested that the attendant pancreatitis was related to reflux consequent on associated duodenal ileus. This is certainly an unusual complication of which the writer has not been aware before but which sounds as though it could plausibly be related to the anorexia nervosa.

Abdominal pain

Anorectics often complain of abdominal pain. They may complain of feeling distended, especially after eating. It should be borne in mind that such a complaint can be entirely psychologically defensive and indeed a very effective defence against those pestering the anorectic to eat more. On the other hand,

of course, the gastrointestinal tract can become so distorted by chronic anorexia nervosa that distension can arise at almost any level under certain circumstances, e.g. disuse atrophy followed by bingeing or the effects of constipation. Colic is often complained about and is then often purgative related or else a function of bizarre dietary intake, such as the drinking of a bottle of pure lemon juice. It is important to remember that abdominal pain can be ovarian in origin. Rapid changes in nutritional status can produce major changes in the ovaries and the writer has had a number of patients over the years who, following persistent abdominal pain, have undergone laparotomy and been found to have a cystic ovary that has been removed. The discomfort of constipation can predominate and become a focus of complaint by the anorectic deviously seeking help to maintain her anorexia nervosa. One must also always bear in mind the possibility of independent pathology. Over the years the writer has come across clear cut cases of anorexia nervosa where ulcerative colitis or Crohn's disease has also been active.

The large bowel

Symptoms referable to the colon have already been touched on. Another rare complication is described in another article in this issue by Kaye *et al.* (1984) who suggest that the fatal necrotising colitis was secondary to large bowel obstruction resulting from faecal impaction.

Rectal prolapse is relatively common in anorexia nervosa. It is a feature of the chronic syndrome and probably related to excessive straining at stool and massive purgative abuse. It must always be borne in mind that such abuse may be expertly hidden but will then usually be evident because of its clinical and metabolic consequences.

This brief review of gastrointestinal complications has focussed mainly on attendant clinical disabilities. Anorexia nervosa is a distressing syndrome and humbling for those who strive to help bring about change in the person concerned. Sometimes it is possible to help alter the course of the disorder. It is likely meanwhile that anorectics, through their grossly disordered gastrointestinal functions, can teach us a great deal about the gastrointestinal tract and its relationship to the rest of the body and the mind including the appetite.

References

- BACKETT, S.A. (1985). Acute pancreatitis and gastric dilatation in a patient with anorexia nervosa. *Postgraduate Medical Journal*, **61**, 39.
- CRISP, A.H. (1970). Anorexia nervosa 'feeding disorder', 'nervous malnutrition' or 'weight phobia' *World Review of Nutrition and Dietetics*, **12**, 452.
- CRISP, A.H. (1980). *Anorexia Nervosa: Let Me Be*. Academic Press: London.
- HURST, P.S., LACEY, J.H. & CRISP, A.H. (1977). Teeth, vomiting and diet: A study of the dental characteristics of 17 anorexia nervosa patients. *Postgraduate Medical Journal*, **53**, 298.
- KAYE, J.C., MADDEN, M.V. & LEAPER, D.J. (1985). Anorexia nervosa and necrotising colitis. *Postgraduate Medical Journal*, **61**, 41.
- RUSSELL, G.F.M. (1966). Acute dilatation of the stomach in a patient with anorexia nervosa. *British Journal of Psychiatry*, **112**, 203.