

# The Diagnosis of Anorexia Nervosa

D. MATTINGLY, MB, FRCP

Director and Professor of Postgraduate Medical Studies, *Postgraduate Medical School, University of Exeter* and Consultant Physician, Royal Devon & Exeter Hospital

S. BHANJI, MD, MRCPsych, DPM

Senior Lecturer in Adult Mental Illness, *Postgraduate Medical School, University of Exeter* and Consultant Psychiatrist to the Exeter Health Authority

More than a century has passed since Sir William Gull[1] described anorexia nervosa and gave the condition its name. He recognised that it affected mainly adolescent girls and young women and that eventual recovery was the rule despite the emaciated appearance and lack of any specific treatment. Since the cause was unknown, he believed that the diagnosis could be made only by the exclusion of other wasting diseases.

We are still ignorant of the true nature of this disease which has been variously described as a psychiatric disorder, an endocrine or metabolic disturbance, or a combination of the two[2]. Patients may therefore be referred to physicians, psychiatrists or gynaecologists. A recent survey suggests that physicians and psychiatrists are asked to treat comparable numbers of anorexics[3]. Nevertheless, most published reports concern those patients referred for psychiatric assessment and management. This article describes the clinical and laboratory findings in a consecutive series of 91 cases of anorexia nervosa seen by a physician (D.M.) in a provincial university city since 1966. In keeping with reports of an increase in the incidence of this disorder[4], three-quarters of these patients were seen during the past seven years.

A diagnosis of anorexia nervosa was made if the following criteria were present:

1. Weight loss greater than 10 per cent of the calculated optimum weight for the patient's age, sex and height, as determined from standard tables or growth charts.
2. Avoidance of carbohydrate foods. The term avoidance includes consumption and then riddance by vomiting or purgation as well as exclusion from the diet.
3. Amenorrhoea of at least three months' duration in females, provided the patient was not taking an oral contraceptive.
4. The absence of any obvious psychiatric illness.
5. The absence of any physical illness which could account for the weight loss.
6. The characteristic physical appearance described hereafter.

## Patients

Eighty-eight females and three males fitting the aforementioned description were seen between 1966 and 1981. Their ages ranged from 13 to 45 years, the majority being under twenty-five; six of the females had been ill for more than ten years. Their ages at onset are shown in Fig. 1. Nine patients were married and four divorced. Fifty were at school or receiving higher education. Of those working, 17 were engaged in clerical employment and six were nurses. The social class according to the chief wage-earner's occupation was ascertained in only the last 50 cases. Two-thirds of these patients came from families in social classes I and II.

All patients were referred because of weight loss, amenorrhoea or both. In one-third, the diagnosis of anorexia nervosa was not considered by their general practitioners. Three were suspected of having hyperthyroidism, one was thought to have Addison's disease and in another the diagnosis of an insulinoma had been made. Other diagnoses included malignancy and malabsorption.

Following referral, 72 patients were admitted to a general medical ward. Nineteen were seen only in the out-patient clinic, either because their condition was mild or they refused treatment.

All but one patient acknowledged their weight loss. Its duration varied from five months to 20 years. The three male patients complained of losing weight and obstinate constipation. Seventy-six females complained of or reported secondary amenorrhoea lasting from three months to 19 years; in nearly half these patients the amenorrhoea coincided with or ante-dated the onset of weight loss. Six women taking an oral contraceptive reported regular 'periods'. The mean age of menarche was thirteen years. Five girls under the age of fifteen and one young woman with Turner's syndrome had primary amenorrhoea.

All the patients avoided carbohydrates and most admitted to loss of appetite. Forty (44 per cent) confessed to self-induced vomiting, purgative abuse or both. In 37 (41 per cent) the illness appeared to have been precipitated by

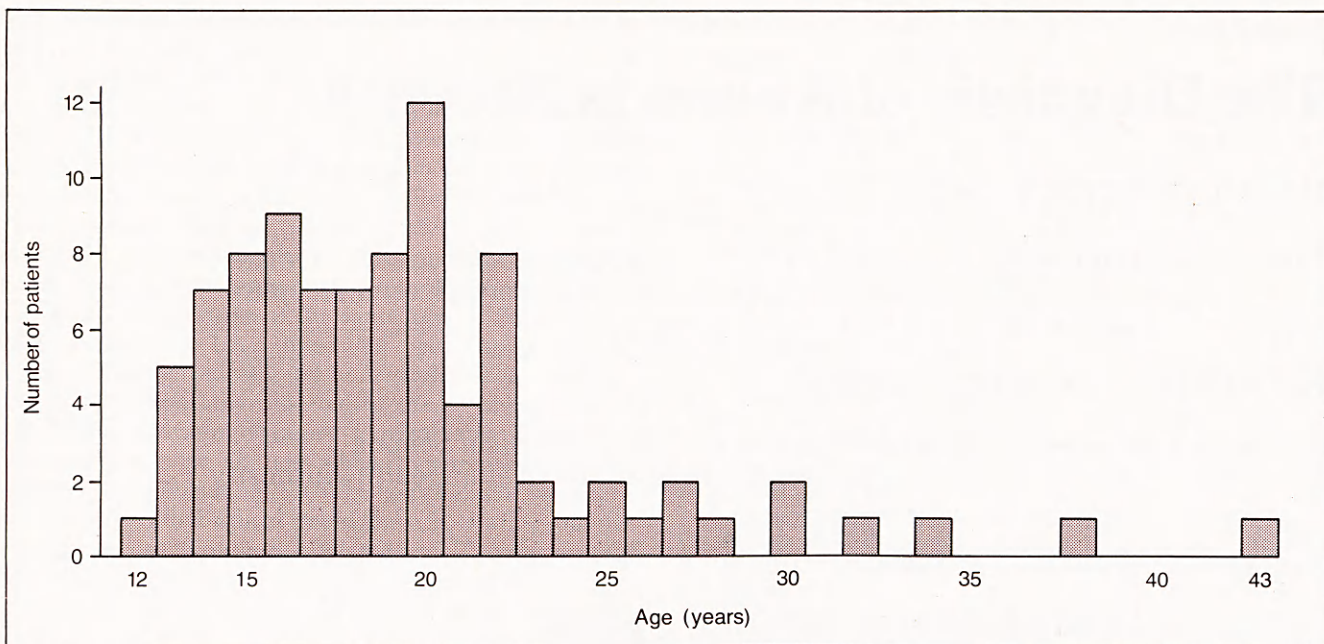


Fig. 1. Age at onset in 91 cases of anorexia nervosa.

dieting because they believed themselves to be overweight. Constipation, fatigue and insomnia were very common (Table 1). No detailed enquiry was made into psychological problems, but over one-third of the patients

Table 1. Symptoms in 91 cases of anorexia nervosa in descending order of frequency.

	%
Perverse appetite	100
Weight loss	99
Amenorrhoea*	93
Constipation	66
Fatigue	54
Insomnia	49
Depression	42
Feeling the cold	39
Nausea after food	32
Abdominal discomfort	30
Dry skin	28
Vomiting after food	26
Headache	23
Nocturia	23
Giddiness	23
Dyspnoea on exertion	18
Falling hair	17
Bulimia	14

\*Six women taking oral contraceptives reported regular 'periods'.

were mildly depressed and in many weight loss followed closely upon psychological stress. Thirty-four had been worried about forthcoming examinations and some of those studying for Advanced Level GCE had already experienced transient amenorrhoea and weight loss before their Ordinary Level examinations. Thirty-three had difficulties with family or sexual relationships, eight had

been upset by the recent death of a close relative and four were unhappy at boarding school. Twelve of the adolescent females came from families where the parents had been divorced or were separated.

Twelve patients had other disorders that were not thought to be responsible for their weight loss, including epilepsy, optic atrophy following encephalitis in childhood, chronic otitis media, benign parotid tumour, fibroma of rib, minor congenital abnormalities of one hand, hirsutism with polycystic ovaries and Turner's syndrome. One 25-year-old nurse died shortly after admission to hospital. She went into a coma following an episode of spontaneous hypoglycaemia at night and eventually succumbed to a salmonella septicaemia. At autopsy a hypothalamic astrocytoma was found. The details of this case have been published[5].

### Physical Findings

On examination, all the patients had clearly lost weight and about half were emaciated. The most striking feature was a marked loss of subcutaneous fat; the muscles were often well preserved. Their weights ranged from 54-89 per cent of the optimum for their age, sex and height. Lanugo hair, acrocyanosis, bradycardia and hypotension were commonly found (Table 2). The lowest recorded heart rate was 36 beats per minute and the lowest systolic blood pressure 70 mm Hg. Pulse rates tended to be slowest in the out-patient clinic, the bradycardia often disappearing after admission to the ward. Hypothermia was observed in one severely emaciated patient only; her rectal temperature on admission was 33°C. Secondary sexual characteristics were usually well-preserved, though the breasts tended to be small. Scanty sexual hair was noted in only nine patients and atrophic breasts in only the six who had been ill for more than ten years.

**Table 2.** Physical signs in 91 cases of anorexia nervosa in descending order of frequency.

	%
Loss of subcutaneous fat	100
Lanugo hair	70
Hypotension <sup>1</sup>	38
Bradycardia <sup>2</sup>	36
Acrocyanosis	36
Dry skin	29
Yellow hands	14
Scanty sexual hair	10
Ankle oedema	8
Atrophic breasts	7

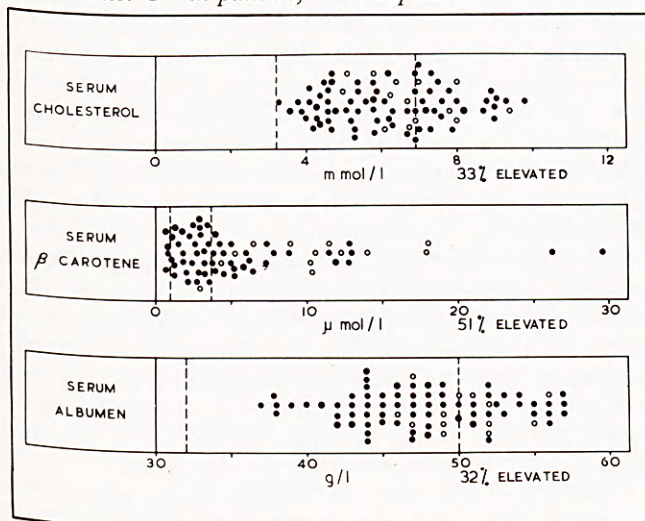
<sup>1</sup>Systolic blood pressure less than 100 mm Hg.  
<sup>2</sup>Pulse rate less than 60 beats per minute.

### Investigations

Haemoglobin concentrations, white cell counts and erythrocyte sedimentation rates were all normal, with the exception of three females with mild iron deficiency anaemia. The only definite abnormality found on chest X-ray was a fibroma of rib, confirmed at operation, but in several cases the heart shadow was noted to be very small. Serum urea concentrations were slightly raised in eight patients and grossly elevated in a purgative addict; none had proteinuria. Hypokalaemia was found in three females who were known to indulge in self-induced vomiting and purgative abuse. Ten untreated patients had slightly elevated serum aspartate aminotransferase levels and in four of them the serum bilirubin levels were also minimally raised. The serum concentrations of cholesterol, carotene and albumin were elevated in a significant proportion of those patients in whom these investigations were done (Fig. 2).

Serum protein-bound iodine or thyroxine concentrations were estimated in 60 patients and were abnormally low in eight; they returned to normal as weight was

**Fig. 2.** Serum cholesterol, carotene and albumin levels in anorexia nervosa. The normal ranges are shown by the vertical broken lines. ●—in-patients, ○—out-patients.



regained. Adrenocortical function was studied in 29 patients admitted to hospital and seven had elevated plasma and urinary 11-hydroxycorticoid levels; in two emaciated females they were comparable to those found in Cushing's syndrome.

### Discussion

Large-scale studies of anorexia nervosa are uncommon and tend to concern those patients referred to psychiatrists. Hence we feel that this report of an unselected series of patients seen by a physician will be of interest.

The diagnostic criteria we employed are consistent with those used by Dally and Gomez[6] and others, and our patients' histories contain a number of familiar features. In particular, the majority of our subjects were middle-class adolescent females. In many, the illness was precipitated by deliberate dieting, preceded by psychological stress and followed by depression. Secondary sexual characteristics were well preserved and lanugo hair, acrocyanosis, bradycardia and hypotension were commonplace. Nevertheless, the diagnosis of anorexia nervosa had not been previously considered in a third of our patients. This may have been because the three cardinal criteria of anorexia, weight loss and amenorrhoea were not always obvious. Some patients avoided carbohydrates but denied loss of appetite; weight loss was less marked in those who indulged in self-induced vomiting or purgative abuse, and regular 'periods' were reported by the six females taking oral contraceptives. The haematological and radiological findings strongly support the view that these are usually normal in anorexia nervosa. Hypercholesterolaemia was found in one-third of our cases; this finding has been well documented. None of our patients was clinically hypothyroid but a number had low serum thyroxine concentrations. This has been previously reported and is thought to be due to changes in peripheral thyroxine metabolism. The increased 11-hydroxycorticoid levels found in a small number of our patients could have been due to stress. Kanis *et al*[2] also found increased levels of these substances in the plasma and suggested this was due to increased protein binding. This seems an unlikely explanation in our cases, as the urinary excretion of 11-hydroxycorticoids was also raised.

Dally[7] first drew attention to the potential diagnostic value of a raised serum carotene level. This was present in about half our cases. Anorexia nervosa is probably the commonest cause of hypercarotenaemia in Britain today. The increased concentrations of serum aspartate aminotransferase did not appear to be due to medication as the patients had not received any drugs known to disturb liver function. The elevated serum albumin concentration found in a third of our patients was an unexpected discovery and could not be explained on the basis of dehydration.

The reason for all these biochemical abnormalities is unknown, but as they revert to normal on refeeding they are generally regarded as secondary to malnutrition. Nevertheless, the finding of raised serum cholesterol, carotene or albumin levels may help to distinguish anorexia nervosa from other wasting disorders. In malab-

sorption and malignancy, for example, the levels are either normal or low.

We agree with most of Gull's postulates but not that the diagnosis is arrived at only by exclusion of other causes of weight loss. Anorexia nervosa is usually readily recognisable from the characteristic history and physical findings. Extensive laboratory investigations are unnecessary and a burden to the patient and pathologist. In the majority, a normal blood picture, sedimentation rate and chest X-ray are all that are required to substantiate the clinical opinion.

Should the diagnosis remain in doubt, it may be helpful to estimate the serum concentrations of cholesterol, carotene and albumin.

#### Acknowledgements

We would like to thank Dr Andrew Marshall for his

assistance during the early stages of this study and Dr T. Hargreaves and Mr J. Barraclough of the Department of Chemical Pathology, the Royal Devon and Exeter Hospital, for carrying out the biochemical investigations.

#### References

1. Gull, W. W. (1874) *Transactions of the Clinical Society of London*, 7, 22.
2. Kanis, J. A., Brown, P., Fitzpatrick, K., Hibbert, D. J., Horn, D. B., Narin, I. M., Shirling, D., Strong J. A. and Walton, H. J. (1974) *Quarterly Journal of Medicine*, 43, 321.
3. Bhanji, S. (1979) *Journal of Psychosomatic Research*, 23, 7.
4. Crisp, A. H., Palmer, R. L. and Kalucy, R. S. (1976) *British Journal of Psychiatry*, 128, 549.
5. Lewin, K., Mattingly, D. and Millis, R. R. (1972) *British Medical Journal*, 2, 629.
6. Dally, P. and Gomez, J. (1979) *Anorexia Nervosa*. London: Heinemann.
7. Dally, P. J. (1959) *British Medical Journal*, 2, 1333.

---

## Book Review

*Scientific Foundations of Paediatrics*, 2nd ed, edited by J. A. Davis and J. Dobbins. Heinemann, London, 1981. Price £75.

Paediatricians like to claim that medicine is divided into the developmental medicine of childhood and the degenerative medicine of adults. Most readers of this Journal may not see medicine in quite such simple terms but there is no doubt that growth and development are the very stuff of paediatrics and this book, edited by John Davis and John Dobbins, is all about the anatomy and physiology of growth and development.

The book itself has grown and developed in the seven years since first published; it has put on some 250 pages and increased its price by £62! To recapitulate for those who are not familiar with the first edition; this is an eclectic work of a high academic standard whose editors must be congratulated on their scholarship and industry. There are 70 contributors drawn from many professional disciplines and from many parts of the world, more than half from the UK. The list of authors is impressive and includes such internationally known names as Cedric Carter, Lynne Reid, James Tanner, Henk Visser and Elsie Widdowson. The scientific basis of growth and development of all the systems of the body is described in great detail from fetal life to adolescence and there are additional sections on genetics, nutrition, epidemiology and oncology. Each section is a scientific monograph in itself and is accompanied by an extensive, sometimes formidable, list of references. The record probably goes to the authors of the section on the Psychology of Infants which lists 442 references.

Apart from its increase in size and cost there are no noticeable changes in this second edition but the editors assure us that the many advances of the last five years have been incorporated. The book remains a landmark in

paediatric literature and enhances the reputation of British academic paediatrics in international circles. There is something in it for every physician, developmental or degenerative. My personal recommendations as a clinical paediatrician would be the sections on The Effects of Emotional Disturbance on Somatic Growth by Dermot MacCarthy, on Physical Growth by David Davies, on Genetics by Cedric Carter and on Epidemiology by Ian Leck; but 'the child is father of the man' and I can recommend anyone practising adult medicine or its subspecialties to spend an hour or two with this book, brushing up on the scientific foundations of his subject in early life.

Although there is a wide range of authors, the editors have managed to achieve a certain degree of uniformity in the style and presentation. Nevertheless, many of the subjects are complex and are dealt with in a strictly scientific manner. This makes rather heavy going for the reader, and I found some of the sections, such as the one on Homeothermy and its Metabolic Cost (121 pages and 356 references), too difficult to get through. John Davis's admiration, shared by most older paediatricians, for the late Donald Winnicott led him to include an essay on this father of British child psychiatry, which I also found difficult to read because of its psychoanalytical language and concepts.

It is to be hoped that John Davis's translation from Redbrick to Oxbridge will not deter him from keeping in touch with John Dobbins and getting to work on the third edition of this book. It may be difficult to get your librarian to spend £75 on one book but it is a reference book of encyclopaedic content and every medical library should have one. One word of warning—it weighs as much as a newborn baby.

A. D. M. JACKSON