Case Reports

A CASE OF ANOREXIA NERVOSA IN A MALE

A. H. CRISP, M.B., M.R.C.P.E., D.P.M.

Lecturer, Academic Psychiatric Unit, Middlesex Hospital, London, W.1

F. J. ROBERTS, M.B., M.R.C.P.E., D.P.M.

Senior Registrar, Department of Psychological Medicine, King's College Hospital, London, S.E.5

Case Report

THE patient, a boy aged 14, was referred to the psychiatric department by a general physician who could find no physical cause for weight loss. He was first seen by one of us in February 1961, when he made little complaint except to say that he did not have his normal strength. His mother was concerned that he had been finicky with his food since April 1960; also that his appetite seemed very much below normal.

He was born in 1947 and is the second of three siblings. The father, a jovial, rotund chef, had a close relationship with the patient. The mother, a tense, worrying woman, appeared to be over-protective towards the patient. The parents are contentedly married. The patient's elder brother, aged 23, suffered with food faddiness until adolescence. The younger sister, aged 12, was said to have been jealous from an early age of the attention given to the patient by his mother and had been a notably poor eater until the age of five.

The patient shared a bedroom with the parents more or less consistently up until the age of 10. As a young boy he had always seemed lively, friendly, conscientious, methodical and concerned over personal cleanliness. There had been a phase of food faddiness around the age of four or five worsened, in the parents' view, by the mother's persistence in trying to get him to eat more. He had suffered with nocturnal enuresis since infancy except on one occasion when he had slept with his father alone for several weeks about three years previously. He had masturbated regularly and with feelings of anxiety and guilt from an early age. He had failed the 11+ examination but was now doing well at school with prospects of success in the G.C.E. At the age of eight he suffered with asthma and subsequently severe sinusitis for a year or more during which period he was said to have walked in his sleep.

The patient eventually revealed that, during the early summer of 1960, he thought that he was beginning to get plump and he resented being called 'fatty' once or twice by a friend and 'pop-bellied' by his brother (there was no obvious obesity in the photographs of the patient at that time). Subsequently he began to reject his father's continual efforts to 'build him up'; particularly he began to avoid hot foods and puddings. In July 1960 he attended a talk at school on the evil nature of masturbation and attendant risk of self-inflicted damage. This had a profound effect on him and for the next three months he strove to end the habit. In November 1960 he stopped masturbation completely. At the same time his nocturnal enuresis ceased.

In September 1960 the patient weighed 119 lb. In

February 1961 he was found to weigh 96 lb. and was admitted for general investigation and, ultimately, psychiatric opinion. A provisional diagnosis of anorexia nervosa was made and his progress was thereafter followed up in the psychiatric outpatient department. There was never at any time any evidence of schizophrenic symptoms or signs. Rapport was poor only that we were unable to make contact with him over the reality of his eating problem. He did not complain of depression and did not appear unduly depressed. He was pre-occupied with several worries; namely that he might miss too much school work if he came into hospital and also a persistent fear that he was too fat. During the next three months he was treated with supportive psychotherapy, small doses of chlorpromazine and norethandrolone. He was seen at weekly intervals. He began to complain that he was finding light exercise increasingly exhausting. Reluctantly he admitted that he was not eating his midday sandwiches which his mother gave him and he was refusing breakfast and eating a meagre evening meal under protest. At the end of March he lost over 3 lb. in one week. In April he suddenly developed a puffy swelling of the face and neck which was said to subside after a few hours. On April 17 he was found to weigh 88 lb. in his clothes, he looked, and admitted to feeling, very ill and was immediately taken into hospital.

On Examination

The patient weighed 80 lb. in his pyjamas. His height was 5 ft. 8 in. He appeared grossly under-weight; his skin was dry, cold, rubbery and had a marked bluish tinge over his abdomen and extremities. Pulse 56 per minute and B.P. 80/60 mm. Hg. (on his previous admission—pulse 52, B.P. 120/80 mm. Hg.) temperature 97°F. His mental state was unchanged. He was put to bed and treatment instituted immediately.

Treatment and Progress

He was given 5 units of soluble insulin and 250 mg. chlorpromazine on the first day (Dally and Sargeant, 1960). The insulin was increased by 5 units a day until a maximum of 35 units was reached. The chlorpromazine was increased by 100 mg. daily to 550 mg. This was given in the form of syrup to prevent the patient secretly rejecting the medication. He suffered distressing side-effects to the insulin and this was discontinued on the 18th day. He was seen daily and the basis of normal diet was frequently discussed with him; he was constantly reassured that the diet prescribed would not make him fat and it was explained to him that we were permitting

FIG. 1

FIG. 2

- (a) Patient at time of admission. (b) Patient 4 weeks later.

him to eat to the extent of restoring his weight to what would be regarded as normal for his age and height. The patient surprisingly tolerated large doses of chlorpromazine until the 15th day of treatment without any sign of side effects and then he began to complain of drowsiness which became increasingly apparent. On the 22nd day the chlorpromazine was reduced to 400 mg. a day and then again on the 26th day to 200 mg. a day. On the 31st day and thereafter he was given just 100 mg. at night.

For the first five days of treatment the patient's temperature was subnormal, varying slightly around 96°F. despite the fact that he was in bed. On the sixth day his temperature began to show a marked diurnal swing until on the eighth and ninth days this was from 95 to 99°F. It then settled for four days at 97°F and finally rose to around 98°F.

On the eighth day the patient's weight started to increase; this coincided with the end of the pronounced daily swing in temperature. During the next 12 days his weight increased by 24 lb. This weight increase was mainly towards the end of the 12-day period and amounted in all to 36% increase of his original weight. The period of rapid increase in weight coincided with an apparent change in the patient's attitude to his condition which was first noticed on the 15th day of treatment, a day or so before he became drowsy with the chlorpromazine; this coincided with the restoration of a more normal temperature pattern. The patient now frankly said that he had sometimes realized that his appetite had been poor but had always been convinced that his weight was satisfactory. He was able to describe how he had at times wanted food but had not dared to touch it. However, he was now aware that he was underweight, that he was hungry and able to eat. His nocturnal enuresis started again. During the period in which the weight increase occurred he remained in bed and received between 1,800 and 2,500 calories per day.

Since discharge from hospital the patient has been treated by supportive psychotherapy on an outpatient basis and has remained well.

Investigations

At the beginning of treatment the patient's daily urinary output of 17-ketosteroids and 17-hydroxy-corticosteroids was found to be towards the lower limit of normal. On the ninth day of treatment a B.M.R. was -39%, corrected for actual body weight; on the 23rd day it was -7%. Thyroid function assessed by ^{131}I uptake was within normal limits on the 13th day. Haemoglobin was 110% on admission, 93% on the third day and 91% on the 21st day. Total and differential white cell and platelet counts were normal on these occasions.

Discussion

Following Gull's and Lasegue's (1873, 1888) initial observations, interest in the psychological mechanism of the disease was mainly limited for many years to regarding it as a disease of hysterical young women reacting either with disgust to an over-threatening sexual situation or else with a perfectly straight-forward wish to lose weight. In 1940, Waller, Kaufman and Deutch elaborated a possible dynamic mechanism based on the individual's wish to reject sexuality and its implications. More recently, others (Kay, 1953) have attempted to assess the natural history of the disease and its relation to the premorbid personality. Untreated, it tends to a chronic and sometimes malignant course, unlike most psychoneurosis.

Many children and occasionally adults (e.g. Ghandi) use refusal of food as a weapon against authority, but this pattern alone does not seem to result in anorexia nervosa. Some authors (Fenichel, 1960) have suggested that the onset occurs in depression. In this context, perhaps the disease may be the equivalent of the aggressive 'no answer' suicidal act (Strauss, 1956). It has also been suggested that (Bliss and Branch, 1960) cumulative factors probably determine the ultimate intensity of the illness which is too difficult to understand in terms of a unitary psychopathology.

Although some authors insist that the disease is confined to females we have diagnosed it in the male. It would be an exceptional psychiatric disease if it were confined to the female, but on dynamic grounds a specific fear of pregnancy which some (Waller and others, 1940) indicate as the basis of the disease would support this view. Amenorrhoea is not a useful clinical sign in the male. If, however, the disease is regarded as an appetite withdrawal it is understandable that the female who, in our culture, is usually more conservative towards sexual and aggressive appetitive indulgence, should suffer more commonly in this way at a time of appetitive threat (e.g. puberty and adolescence) than the more adventurous male. The exception to this might be the inhibited or obsessional male, and our patient was of this type.

Our patient shared his parents' room at night and must have been frequent witness of their sexuality. This experience might explain his early masturbation and his nocturnal enuresis. The latter habit may have been re-enforced by the purpose it served in distracting his parents and in this context

it is interesting to note that the enuresis stopped when the patient slept with his father.

Regarding the onset of his illness there is a most untypical history (Schneider, 1947) of slight obesity followed by fear of getting fat and then of starting to diet.

The history in this case also reveals the conflict over sexuality which was brought to a head by a school lecture on sex just at the time of puberty and developing sexuality. The disease frequently occurs at this time of sexual development with its attendant threats from puberty through to prospective marriage. Various psychological mechanisms and symbolic interpretations have been suggested to try to explain why the withdrawal from sexual threats should spread to eating. The mouth, according to many (Fenichel, 1960), the first erotic zone. Later, eating of food and consequent enlargement of the abdomen have been compared (Bliss and Branch, 1960) to the act of impregnation which leads to pregnancy. Whatever the mechanism, it seems that our patient's need was to withdraw from appetitive indulgence in the presence of mounting anxiety and tension.

The abnormal metabolic state of these patients has been commented on before (Berkman, 1930). The initial degree of reduced metabolic activity in this patient was striking and we would like to emphasize the almost step-like change in his metabolic recovery during treatment.

At the beginning of treatment the patient was surprisingly able to tolerate large doses of chlorpromazine. Dally and Sargeant (1960) stated that they gave similar doses of chlorpromazine to many of their patients. Our patient's recovery coincided with a change in his tolerance to chlorpromazine as indicated by the onset of drowsiness. Tolerance to large doses of chlorpromazine has been mainly commented on in relation to the psychoses (Belar, 1958).

A Drive-reduction Theory

One of us (Crisp, 1962) has previously suggested that the habit of not eating may be particularly rewarded in this illness since the consequent persistent hunger in the early stages of the illness inhibits sexual drive which then no longer threatens the individual. That is to assume that (1) these patients need to escape from sexual threat, and (2) the drive to satisfy hunger is more fundamental than, and over-rides, the normal sexual drive in humans, as it does in rats (Warden, 1931). It may be that the motor over-activity and sleeplessness sometimes seen in these patients is a physiological concomitant of this chronically unsatisfied drive. There may be some similarity here to the experience of undernourished prisoners-of-war who have reported (personal communications) that their sexual drive was lost during their state of malnutrition and, indeed, often did not return for some months after repatriation. Unlike the prisoner-of-war, however, the patient with anorexia nervosa needs to be in such an abnormal state and may adopt it more fully. If this is so, then modified insulin treatment would

not be expected to be of much therapeutic value, since a disease process starting in the state of needing to feel hungry for food is unlikely to be influenced by a state of artificially induced hunger for food.

This theory would be consistent with the neurophysiological concept of an arousal homeostatic mechanism in which stimuli causing intolerable arousal, e.g. sexual threats—are inhibited, by various mechanisms, from awareness. In anorexia nervosa, inhibition of this awareness might be maintained by ensuring a state of chronic hunger for food which effectively attracts and fixes maximum total tolerable arousal. This particular homeostatic mechanism might be facilitated in these patients because of some special symbolic or associative meaning that food has for them; or because they happen to be already dieting. Perhaps, eventually, this latter arousal, because of its ineffectiveness in relieving hunger, becomes intolerable; then hunger itself becomes inhibited and the patient is then cut off from all his drives, collective and individual, towards survival. These changes may be reflected in diminishing measurable specific hypothalamic activities.

Experimentally in animals chlorpromazine has been found to work in varying specific ways (Bradley and Key, 1959; Killam and Killam, 1956; Preston, 1956; Olds, Killam and Eidusen, 1957).

Mostly it has been shown to raise the arousal threshold to indirect stimuli. If this mechanism holds for human beings then its action might be to reduce the total arousal without inducing sleep to the extent that stimuli (in this case sexual appetite) which previously provided intolerable arousal, could once more be integrated into consciously acceptable experience. Also, in this connection, it is interesting to note that the patient's BMR and body temperature returned to normal despite the specific tendency of the drug to often reduce these functions. This would seem to indicate that it is having its greatest effect by some other mechanism perhaps by the effects described above. Improvement of appetite and some weight gain are also described as fairly common effects of the drug (Kalinowsky and Hoch, 1961) in a variety of patients, but whether this is due to some specific effect on the hunger mechanism or whether it is the consequence of increased tranquillity is not clear.

These suggestions perhaps serve to qualify at least some cases of anorexia nervosa as being closer in form to a psychotic process than a maladaptive but reality related neurosis. They are based on observation of the nature of the condition and speculation regarding the underlying physiological mechanisms.

We would like to thank Dr. K. Freudenberg for permission to publish this case.

REFERENCES

- BELAK, L., and BENEDICT, P. K.: 'Schizophrenia', p. 489. New York: Logos Press.
- BERKMAN, J. M. (1930): Anorexia Nervosa, Anorexia, Inanition and Low Basal Metabolic Rate, *Amer. J. ment. Sci.*, **180**, 411.
- BLISS, E., and BRANCH, C. (1960): 'Anorexia Nervosa', pp. 66 and 73. New York: Paul B. Hoeber.
- BRADLEY, P., and KEY, B. (1959): A Comparative Study of the Effects of Drugs on the Arousal System of the Brain, *Brit. J. Pharmacol.*, **14**, 340.
- CRISP, A. H. (1962): Psychogenic Anorexia, *Middlesex Hospital Journal*, **62**, in press.
- DALLY, P., and SARGEANT, W. (1960): A New Treatment of Anorexia Nervosa, *Brit. med. J.*, **i**, 1770.
- FENICHEL, O. (1960): 'Psychoanalytic Theory of the Neuroses', pp. 62, 138, 189, 412. London: Routledge and Kegan Paul.
- GULL, W. W. (1873): Anorexia Hysterica, *Brit. med. J.*, **ii**, 527.
- (1888): Anorexia Nervosa, *Lancet*, **i**, 516.
- KALINOWSKY, L., and HOCH, P. (1961): 'Somatic Treatments in Psychiatry', p. 17. New York and London: Grune and Stratton.
- KAY, D. W. (1953): Anorexia Nervosa—a Study in Prognosis, *Proc. roy. Soc. Med.*, **46**, 669.
- KILLAM, E., and KILLAM, K. (1956): A Comparison of the Effects of Reserpine and Chlorpromazine to those of the Barbiturates on the Central Afferent Systems in the Cat, *J. Pharmacol.*, **116**, 35.
- LASEGUE, C. (1873): De L'Anorexie Hysterique, *Med. Times and Gaz.*, **2**, 265 and 367.
- LEHMAN, E. (1949): 'Psychoanalytic Study of the Child', Vols. III and IV, p. 461. London: Imago.
- OLDS, J. D., KILLAM, K., and EIDUSEN, S. (1957): 'Psychotropic Drugs', p. 235. Amsterdam: Elsevier.
- PRESTON, J. B. (1956): Effects of Chlorpromazine of the Central Nervous System of the Cat, *J. Pharmacol.*, **118**, 100.
- SCHNEIDER, R. (1947): Störungen der Ernährungstrieb, *Schweiz. Arch. Neurol. Psychiat.*, **58**, 315.
- STRAUSS, E. (1956): Suicide, *Brit. med. J.*, **ii**, 818.
- WALLER, J. V., KAUFMAN, N. R., and DEUTCH, F. (1940): Anorexia Nervosa, A Psychosomatic Entity, *Psychosom. Med.*, **2**, 3.
- WARDEN, C. J. (1931): 'Animal Motivation Studies'. New York: Columbia University Press.