# Hypoglycaemia and anorexia nervosa

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*J R Soc Med* 1995;88:191–195

**Keywords:** *anorexia nervosa; bulimia; hypoglycaemia; hypothalamus*

## INTRODUCTION

Ever since it was first described, the majority view has been that anorexia nervosa has a psychological cause. The biological aspects of the disorder have been largely regarded as secondary to chronic malnutrition, although Russell did suggest that they might induce a symptom perpetuating vicious circle[1].

On the other hand, there are at least 10 reports of patients who appeared to be suffering from classical anorexia nervosa but were later found to have cerebral tumours involving the hypothalamus[2,3]. The first convincing case was one of our early patients who died in hypoglycaemic coma and was found at autopsy to have a small hypothalamic astrocytoma[4]. The tumour was later shown to contain substantially more catecholamine-secreting neurones than the normal surrounding tissue.

We are not suggesting for one moment that structural changes are present in every case of anorexia nervosa, but the fact that infiltrating tumours of the hypothalamus can mimic this condition does raise the possibility that it may be due to functional disturbances in this region of the brain.

## HYPOTHALAMIC ABNORMALITIES

In many cases the most obvious disorder, amenorrhoea, precedes any significant weight loss, and is associated with low serum gonadotrophins and a blunted response to gonadotrophin-releasing hormone[5]. Other abnormalities include the inappropriate secretion of the antidiuretic hormone, arginine vasopressin[6], increased secretion of corticotrophin-releasing hormone (CRH) leading to adrenocortical overactivity[7], elevated fasting serum growth hormone levels and a paradoxical rise in these levels following oral glucose[8].

It has been proposed that anorexia nervosa is caused by increased neurotransmitter activity in the hypothalamus. For example, Morley and colleagues[9] have suggested that in constitutionally predisposed individuals nonspecific environmental stresses act to increase serotonin turnover in the paraventricular nucleus. In turn, this leads to the release of CRH which inhibits feeding. Dopamine, noradrenaline and opioids have also been implicated[10–12].

These theories are supported by reports that neurotransmitter antagonists may be effective in treatment. Over 30 years ago, Sargant and his colleagues found that the broad spectrum neurotransmitter antagonist, chlorpromazine, could induce a remission in some cases[13]. Similar claims were later made for the more specific dopamine antagonist, pimozide[14], and the α-adrenergic blocker, phenoxybenzamine[11]. Naloxone and pizotifen, which block the central effects of opioids and serotonin, respectively, have also been described as being effective[12,15].

We have reported the beneficial effects of chlorpromazine and pimozide in 71 patients, and observed that rapid weight gain usually began after a latent period of about 14 days and was preceded by a rise in the resting pulse rate[2]. At the same time, the skin became warm and lost its pallor, and the morose and withdrawn often became charming and self-assured. Supression of abnormal neurotransmitter activity in the hypothalamus could have accounted for these changes. Our results are comparable to those obtained using behaviour therapy, and weight gain was achieved more rapidly than is usually the case with psychotherapy.

If we accept that hypothalamic overactivity is involved in the pathogenesis of anorexia nervosa, what is the most likely cause? Any explanation must take into account that it follows strict dieting and is largely confined to adolescent girls and young women.

Merimee and Tyson[16] fasted normal adults for 72 h and found that the premenopausal women had lower mean plasma glucose levels than the men throughout. This sex difference was apparent after only 24 h. The lowest plasma glucose concentrations recorded during the fast are shown in Figure 1. Prepubertal children had glucose responses during fasting comparable to those of the women. Similar results were obtained by Fajans and Floyd[17] in healthy adults fasted for 72 h. The lowest level in the women was 36 mg/dl (2.0 mmol/l), compared to 55 mg/dl (3.1 mmol/l) in the men.

It would therefore appear that young women are at much greater risk of developing hypoglycaemia after fasting than young men, and that this is probably due to differences in body composition[18,19]. Studies involving families and twins have shown that genetic factors play an important role in anorexia nervosa[20]. It has been suggested that in an

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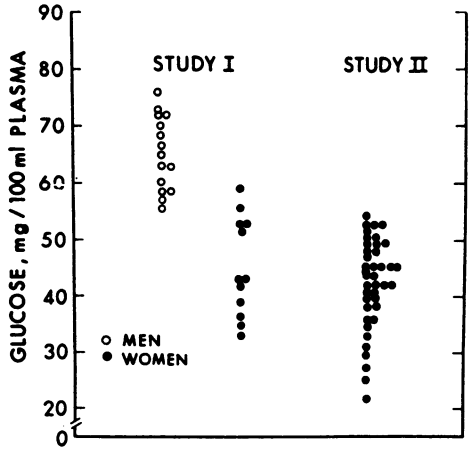


*Figure 1* **Lowest plasma glucose concentration recorded in a 72 h fast for each subject. Solid circles indicate women, and open circles men. Three of 12 men were fasted twice. Conversion factor: mg/dl × 0.056=mmol/l. (Reprinted, by permission of the *New England Journal of Medicine*, 1974;291:1276)**

environment in which dieting behaviour, and hence weight loss, is encouraged, those who are genetically predisposed are highly likely to develop this disorder. Could prolonged hypoglycaemia be the trigger which converts strenuous dieting in these vulnerable girls and young women into a pathological obsession?

## HYPOGLYCAEMIA IN ANOREXIA NERVOSA

Hypoglycaemia is often defined as a plasma glucose concentration of less than 2.5 mmol/l, a level at which acute signs and symptoms are likely to appear. However, this definition implies that concentrations above this level are of little clinical significance. We take the view that any plasma glucose level below the normal overnight fasting range is indicative of hypoglycaemia and may evoke some hormonal response.

Severe hypoglycaemia, with plasma glucose levels as low as 1.0 mmol/l, does occur in anorexia nervosa but is rare and often fatal[21]. On the other hand, milder forms of hypoglycaemia are common and usually asymptomatic. In 91 anorectics that we studied[2], the mean overnight plasma glucose concentration was only 3.9±0.06 (SEM) mmol/l, the lowest concentration being 2.0 mmol/l. Two-thirds had levels below our normal overnight fasting range of 4.2–6.5 mmol/l. None of our patients had symptoms of hypoglycaemia, but hypoglycaemia unawareness is not confined to anorectics. It is also seen in children with chronic protein-calorie malnutrition[22], and in diabetics on insulin therapy.

There is good evidence that the plasma glucose levels at which the glucose counter-regulatory systems are activated are higher than the threshold for symptoms, even in the healthy individual. Schwartz and her colleagues[23], using an insulin clamp technique, found that they lay within, or just below, the physiological range. They suggested that their findings provided further support for the concept that glucose counter-regulatory systems are involved in the prevention, as well as the correction, of hypoglycaemia. Symptoms appeared in their patients when their plasma glucose levels fell below 53 mg/dl (2.9 mmol/l). Thus, even mild hypoglycaemia in an anorectic is likely to activate the neurotransmitters in the hypothalamus which stimulate corticotrophin (ACTH) and growth hormone secretion.

Insulin-induced hypoglycaemia in primates, including man, leads to increased growth hormone and ACTH secretion[24]. In Rhesus monkeys, the chemoreceptors controlling growth hormone secretion are situated in the lateral hypothalamic area[25], but the neurotransmitters involved have not been identified.

On the other hand, neuroglycopenia is known to be a potent stimulus to noradrenergic and serotoninergic neurones in the rat hypothalamus[26–28]. Increased noradrenergic activity is associated with stress-induced ACTH release[29] and evokes spontaneous feeding in the satiated animal[26,30]. By contrast, activation of the serotoninergic pathways in the rat hypothalamus stimulates growth hormone secretion[31], and inhibits feeding[32].

If similar biochemical responses occur in humans, chronic hypoglycaemia could account for many of the endocrine abnormalities found in anorexia nervosa. There is some evidence that dieting increases brain serotonergic activity in women but not in men[33], whilst the increased melatonin secretion observed in some anorectics provides indirect evidence of hypothalamic noradrenergic overactivity[34].

Hypoglycaemia is a major stress, and the events it sets in train include behavioural as well as physiological effects. Voluntary starvation can cause irrational thoughts and bizarre behaviour, including binge eating[35], but the anorectic's conviction that normal eating is unnecessary or even harmful amounts to a psychotic state.

## SUPPORTING EVIDENCE

Similar endocrine abnormalities, such as adrenocortical overactivity and raised serum growth hormone levels, have been described in cases of gross malnutrition from other causes[36,37]. Hypoglycaemia could be the common factor here.

Epilepsy is not uncommon in anorexia nervosa, and a substantial proportion of patients have an abnormal electroencephalogram (EEG). No consistent explanation has emerged, but the EEG appearances are very similar to those seen in hypoglycaemic patients. In both conditions the commonest findings are a generalized slowing of background activity, and heightened sensitivity to hyperventilation[38–40].

There are now many reports of apparently asymptomatic cerebral atrophy in anorexia nervosa, revealed by computed

tomography (CT) and magnetic resonance imaging[41,42]. The main findings consist of ventricular dilatation and widening of the sulci. We scanned 23 patients using CT and in 10 we found these abnormalities[2]. There are considerable doubts as to whether these changes are completely reversible even after satisfactory weight gain.

It has been suggested that this cerebral atrophy is due to increased adrenocortical activity, since similar changes have been described in Cushing's syndrome[43]. However, in anorexia nervosa the cortisol secretion rates are reported to be within the normal range, and the plasma cortisol levels are only moderately increased[8], so that this seems unlikely. Glucose deprivation is an alternative explanation, for it has long been recognised that prolonged hypoglycaemia can cause cerebral atrophy[24,44].

## BULIMIA NERVOSA

When Russell first described bulimia nervosa he regarded it as an ominous variant of anorexia nervosa[45]. Some psychiatrists believe it to be a separate entity, but it is now acknowledged that many patients with eating disorders have features of both conditions[46].

In bulimic patients fasting is followed by gross over-eating so that little, if any, weight is lost. Nevertheless, there is evidence of reduced blood sugar and its consequences. Mean overnight and 24-h plasma glucose levels have been found to be significantly lower in bulimics than in healthy women of comparable age[47,48]. Similar endocrine disturbances may occur in both anorexia nervosa and bulimia[49], and cerebral atrophy is becoming increasingly recognized in normal weight bulimics[50].

## EATING DISORDERS AMONG DIABETICS

The commonest cause of hypoglycaemia in the young is the use of insulin in diabetes. Three of our anorectics had been treated for insulin-dependent diabetes mellitus (IDDM) before the onset of their anorexia nervosa. All three had a history of poor control, heavy glycosuria and frequent hypoglycaemic episodes[51].

In the past 10 years there have been numerous reports of a possible association between IDDM and anorexia nervosa or bulimia, but this is not universally accepted. Fairburn and his colleagues[52], in a controlled study, concluded that there was no evidence that clinical eating disorders were more prevalent in young women with diabetes than in non-diabetic women, but agreed that disturbed eating, poor control of glycaemia, and the misuse of insulin to influence body weight is common in these patients.

Those who believe that this relationship is more than coincidental have provided various psychological explanations[53]. Our hypothesis, however, suggests that neuroglycopenia could have been responsible. In most of the reported cases the eating disorder was preceded by the onset of the diabetes, and the latter had been poorly controlled[54].

## WEIGHT LOSS ASSOCIATED WITH INSULINOMA

This rare disorder is one of the few known causes of chronic neuroglycopenia. Accordingly to Marks and Rose[24], hunger is a symptom in only some 14% of cases. Contrary to common belief, obesity is not usually a feature of these tumours and occurs only in those patients who are aware of the relationship between attacks and lack of food. Overall, weight loss is as common as weight gain. Odd behaviour or disturbances of consciousness are the commonest causes of referral. Almost any psychiatric condition may be mimicked, including anorexia nervosa[55].

## VEGETARIAN DIETS

It has been claimed that many vegans persistently run low blood glucose levels, but we have not found any published evidence to support this. The known complications of lacto-vegetarianism include deficiencies of iron, vitamin $B^{12}$ and vitamin D, but hypoglycaemia has only been reported as a complication in a child with the rare disorder of systemic carnitine deficiency[56].

In a recent study of young Chinese Buddhist vegetarians the female monks did have significantly lower overnight plasma glucose levels than the male monks and omniverous female medical students[57]. However, this sex difference was not found in other studies carried out in North America and Sweden which specifically looked at glucose metabolism on a vegetarian diet[58,59].

We can only conclude that vegetarians with persistently low plasma glucose levels are likely to be suffering from undiagnosed anorexia nervosa, since many patients with this disorder avoid eating meat as well as carbohydrates. In a recent retrospective study of 116 cases, about half the anorectics had become vegetarians at the onset of their illness[60].

## CONCLUSIONS

Many cases of anorexia nervosa are precipitated by psychological stresses, but our experience of 155 cases[2] suggests that in some patients strict dieting alone may be sufficient. Whatever the reason, fasting for more than a day or two will alert the glucose counter-regulatory systems, and may stimulate or inhibit the food-seeking centres in genetically vulnerable subjects. The former will encourage binge eating, whilst the latter will prolong the hypoglycaemia and produce the vicious circle proposed by Russell[1].

Delusional ideas result from stimulation of other pathways and further disturb the attitudes toward eating. Finally, overactivity of the autonomic nervous system[61] may

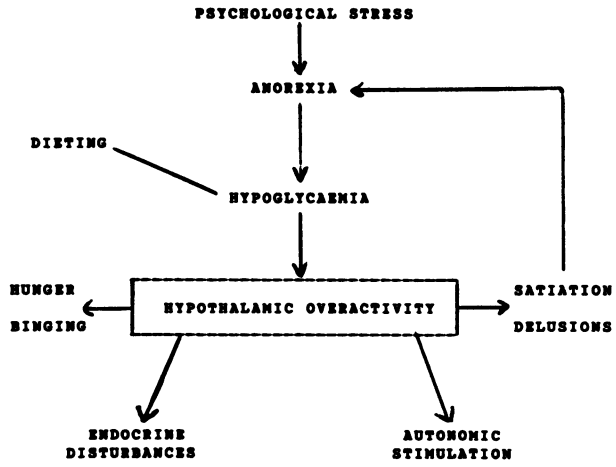


*Figure 2* **Proposed relationship between fasting, hypoglycaemia, hypothalamic stimulation and anorexia nervosa**

lead to such features of anorexia nervosa as acrocyanosis, bradycardia and changes in the electrocardiogram (Figure 2).

Once this train of events has begun, the hypothalamic overactivity triggered off by chronic hypoglycaemia is likely to persist, even if the blood sugar returns to normal at times. As with other causes of chronic neuroglycopenia, the adverse effects can only be corrected by the permanent restoration of normoglycaemia.

In the rat, hypothalamic noradrenergic activity is inversely related to the blood glucose level and is inhibited by hyperglycaemia[27]. To achieve sustained hyperglycaemia in patients would require the administration of glucose intravenously for several days. Neurotransmitter antagonists may also help to restore hypothalamic function to normal by blocking overactive pathways.

Anorexia nervosa is a serious illness with significant morbidity and mortality. Similarly, many bulimics fail to respond to treatment. Whether hypoglycaemia is the cause of these eating disorders in genetically vulnerable subjects, or merely the result of chronic malnutrition is open to debate, but we hope that our ideas concerning its possible role will stimulate greater medical interest in these unfortunate girls and young women, and may lead to more rational treatment and a better outcome.

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*(Accepted 2 November 1994)*