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Acute Dilatation of the Stomach in a Patient with Anorexia Nervosa

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It is generally believed that anorexia nervosa is due to an abnormal attitude of mind which results in an insufficient intake of food. As the term anorexia nervosa implies, many patients experience a loss of appetite, but sometimes this is not so, and vomiting is an alternative or additional device adopted by the patient to ensure a reduction in effective food intake. As a rule the patient induces vomiting by tickling her throat with her fingers, but sooner or later she becomes so proficient at vomiting that mechanical stimulation is no longer necessary. Nevertheless one should not assume that vomiting in anorexia nervosa is always under the control of the patient's will. In the patient described below vomiting became intractable and was found to be due to a physical disorder—acute dilatation of the stomach. Such a complication occurring in a severely undernourished patient is highly dangerous unless recognized early and treated effectively. The opportunity was taken to measure water absorption and gastric motility.

Case History

The patient was a 16-year old girl. When she was 12 her mother died from malignant disease after a painful illness with much loss of weight. The patient's periods began at about this time. Between 13 and 14 she began dieting, because she believed her hips and abdomen were too fat; her weight had previously been in the region of 10 st. 7 lb. (67 kg.). She avoided potatoes and slowly reduced her weight. When she was 15 her father remarried, and her relations with her step-mother were from the beginning unfriendly. Menstruation now ceased, and dieting became excessive, so that eventually the patient was eating only small quantities of meat, cheese and fruit. Moreover she took vigorous exercise with the object of reducing her weight still further. Sometimes she even tried to make herself vomit after meals, but apparently without success. Her weight loss had become precipitous by the time she was admitted.

On admission to the metabolic ward of the Maudsley Hospital the patient was emaciated and weighed only 35 kg. (5 st. 7 lb.). She had pitting oedema of the ankles, and her hands and feet were cold and cyanosed. The pulse rate was 44 per minute and the blood-pressure was 85/60 mm. Hg. The skin was scaly and dry with raised hair follicles. There were crusted abrasions on the elbows incurred while pedalling her legs in the air in an attempt to reduce her thighs. Fine downy hair was present over the cheeks and down the nape of the neck.

The patient's mental state was characterized by feelings of depression and obsessional thoughts about her body size. She was unable to get the idea out of her head that she was fat. On the one hand she recognized this to be silly, but on the other hand she firmly believed that her legs were colossal and her abdomen was too fat. Appetite and hunger were preserved: she wished she could have something which would let her eat and yet prevent her from gaining weight.

Haemoglobin was 12·1 g. per 100 ml. The following serum investigations were normal: sodium, 130·0 mEq. per litre; potassium, 5·4 mEq. per litre; chloride, 103 mEq. per litre; bicarbonate, 25 mEq. per litre. The 24-hour urinary excretion of 17-hydroxycorticosteroids was 11 mg.

After five days' observation, during which the patient's inadequate food intake was confirmed, treatment was begun by feeding a liquid diet containing Complan,* lactose and Nescafé,† and providing 1,500 Calories daily. This diet has been found to be useful in the treatment of patients with anorexia nervosa (Russell, 1960). From the first day she became distressed and complained of feeling nauseated by the diet. Two days later she began vomiting bile-stained fluid, and soon afterwards the diet was stopped. She was given 1 l. saline intravenously and lemon water to drink for five days. Thereafter she was persuaded with some difficulty to return to the liquid diet which now contained 505 g. solids in 1,000 ml. water and provided 2,176 Calories daily with an additional 1,500 ml. drinking water. She gulped it down and gained 2·3 kg. (5·1 lb.) in four days, there being no recurrence of the vomiting. The food content of the diet was next increased to 803 g. providing 3,450 Calories daily. Three days later, however, she complained of abdominal distension and nausea, and suddenly vomited: she appeared depressed and once again expressed her fears of becoming fat. Vomiting of large amounts (up to 880 ml.) of green coloured fluid recurred without any warning, and at times when she was observed by a nurse who was satisfied that vomiting was not self-induced. Throughout this time she expressed strong repugnance for her diet which she said tasted like disinfectant. Vomiting increased to 1½–3 l. daily and in three

*Glaxo, Limited. †Nestlé & Company Limited.

days her weight fell by 3·6 kg. (7·9 lb.). She was severely dehydrated and complained of thirst. The abdomen was distended and splashed on succussion. The vomitus contained bile but no free hydrochloric acid, and Candida albicans and Escherichia coli were cultured. The following serum levels were found; sodium, 136 mEq. per litre; potassium, 4·2 mEq. per litre; bicarbonate, 30 mEq. per litre; osmolality, 333 milliosmoles per litre. Blood urea was 34 mg. per 100 ml.

The patient's vomiting was recognized to be due to an acute dilatation of the stomach, and gastric aspiration yielded 2,560 ml. of bile-stained fluid. For five days aspiration was continued and intravenous infusions of glucose and saline were given. She was then able to retain milk and water, and when told she would not need to return to the loathed liquid diet accepted a mixed solid diet without demur. It was gradually increased over three weeks until she consumed 3,600 Calories daily. Thereafter her weight rose steadily to reach 49·8 kg. (7 st. 11 lb.) at discharge from hospital two months later, by which time she had also lost her morbid ideas about feeding. She recognized that her preoccupations with slimming had been abnormal and resolved never to lose weight again. She made excellent progress thereafter, and when last seen 15 months after discharge she weighed 53·4 kg. (8 st. 5 lb.) and was menstruating regularly.

Investigation of Gastric Function

Water Absorption

Water absorption from the gastro-intestinal tract was tested when the vomiting was at its height, before instituting treatment by gastric aspiration and intravenous infusions. The patient had received no food or fluids for twelve hours and was given 100 ml. water to drink, containing 0·5 millicurie tritiated water. Its rate of absorption was determined from measurements of the specific activity of plasma water obtained from venous blood samples taken 7, 22 and 50 minutes later. The respective specific activities were 0·67, 0·75 and 1·22 per cent. of the administered dose of tritiated water per litre body water, and are shown in Fig. 1 (A). The results are expressed per litre body water because it was not possible to determine the total body water owing to the recurrence of vomiting soon after the 50 minutes blood sample was taken, when the test was discontinued. Water absorption was measured again twelve weeks later when the patient had recovered. Blood samples were taken at the same times after drinking the tritiated water as before, with an additional sample beforehand in order to allow for tritium activity remaining from the previous test. The percentages

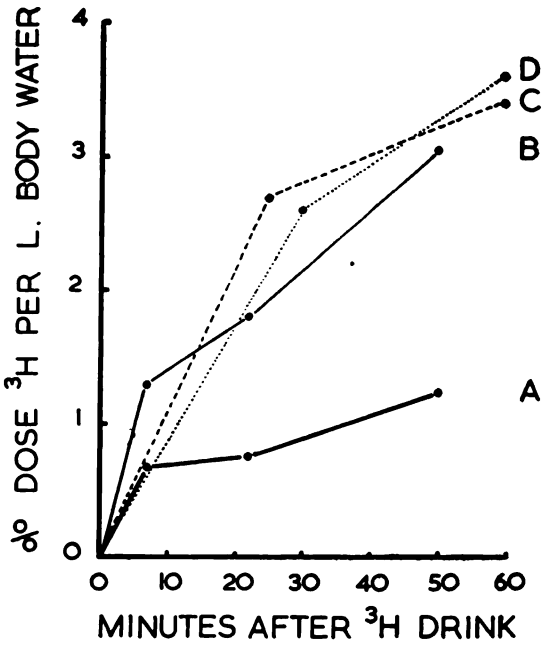

Fig. 1.—Absorption of tritiated water from the stomach.

A. In the patient at the height of the gastric dilatation.
B. In the patient after recovery.
C. In another patient with anorexia nervosa who did not vomit.
D. In a normal woman.

of the dose per litre body water at 7, 22 and 50 minutes were 1·28, 1·79 and 3·05 respectively, and are shown in trace B of Fig. 1. These results show that water absorption was much reduced at the time of the first test, shortly before treatment was initiated for the gastric dilatation.

As the results are expressed in terms of a specific activity, it must be remembered that differences might be due to changes in the patient's total body water between the two tests. She was indeed dehydrated at the time of the first test and had gained 15 kg. (33 lb.) when retested, so that the total body water must have increased substantially. But this increase would in fact lead to a reduction in the specific activities of plasma water so that the differences obtained are even more significant. Trace C shows the water absorption in another patient with anorexia nervosa who did not vomit, and trace D is that of a normal woman. These traces

approximate to that of the patient after recovery from her gastric dilatation. In these two control subjects the tritiated water was given in a drink of 1,200 ml. to establish that normally even a large draught of water is rapidly absorbed.

Gastric Motility

Gastric motility was recorded for 1 hour and 20 minutes five days after the diagnosis of gastric dilatation had been made, and as the patient was beginning to tolerate food again. A modification of the method of Lorber and Shay (1954) was used: a water-filled, open-ended polythene tube was passed nasally into the stomach, and the pressure waves were recorded by means of a Statham transducer connected to an AC-coupled amplifier and recording ammeter. Gastric activity was present for half the period of recording, waves reaching a pressure of 20 cm. water and some lasting up to 45 seconds.

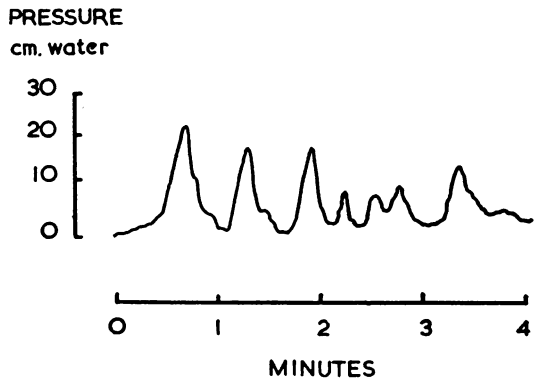

FIG. 2.—Gastric contraction waves 5 days after commencing treatment for the gastric dilatation.

Barium Meal

Ten weeks after recovery from the gastric dilatation, a barium meal X-ray examination showed a normal stomach.

DISCUSSION

The patient suffered from typical anorexia nervosa. When treated with a liquid diet which she disliked she developed intractable vomiting which culminated in acute dilatation of the stomach. This complication of anorexia nervosa has not hitherto been described. It is obviously important to recognize its occurrence in an illness which is thought to be psychogenic and in which vomiting, when it occurs, is most likely to be purposely induced by the patient in order to lose weight.

The cause of vomiting in anorexia nervosa may, however, sometimes be elusive. There may be an obvious explanation when a patient is discovered to tickle her throat with her fingers or an instrument. In other patients, however, there is no such ready explanation for the vomiting, and they are suspected of having learnt to vomit spontaneously (Bliss and Branch, 1960), and presumably at will. How this is achieved is not always clear. Some patients become nauseated and vomit after gorging themselves with food or drink in amounts or of a kind calculated to cause vomiting. Or perhaps nausea is engendered by distaste for the food, by fears of eating, or by dread of the consequent gain in weight.

However, simple physical causes must in the first place be considered as at least contributing to the gastric dilatation and vomiting in the present patient. For example, hypokalaemia is thought to lead to gastric dilatation (Hamburger and Leger, 1955), and is a known complication of anorexia nervosa (Wigley, 1960) This factor was excluded because the patient's serum potassium was normal. Yet there is no doubt that her physical health was impaired by severe malnutrition. Although gastric dilatation has not been reported in anorexia nervosa, it is known to have occurred in prisoners of war who had suffered privation and starvation (Markowski, 1947). These patients were suddenly given very bulky meals of low caloric value such as three litres of soup in a day taken with large amounts of bread. Other workers have stated that rapid refeeding in starvation is safe (Murray, 1947; Widdowson, 1951). In any case, the increased feeding was far less drastic in the patient with anorexia nervosa. Moreover the same dietetic treatment has been administered in the Metabolic Unit to 16 other patients suffering from anorexia nervosa, without any of them developing signs of gastric dilatation. Thus, although the existing malnutrition and attempts at refeeding may have been partly responsible for the gastric dilatation,

it is difficult to escape the conclusion that the patient's emotional distress may have played a significant part in its causation. Such a psychogenesis needs to be considered in view of her feelings of revulsion for the diet prescribed and her imagined need to avoid becoming fat. It is conceivable that this would lead to a disturbance of gastric function. That emotional factors might lead to dilatation of the stomach is of considerable interest because only rarely can it be demonstrated that an organic lesion follows in the wake of an emotional stress (Lewis, 1954). In experimental animals lesions cannot readily be induced as the immediate or delayed effects of emotional disturbances. MacLean (1960) has suggested that in man the experimental stresses are seldom sufficiently drastic or continuous. The requisite conditions may have been fulfilled in the patient described: the threat of feeding with a diet found to be unpleasant certainly constituted a prolonged and severe stress, and probably contributed to the vomiting and gastric dilatation. When the threat was removed and a solid diet offered she accepted it readily, her mental attitude improved and the vomiting did not recur.

An opportunity was taken of investigating water absorption in the patient when the gastric dilatation was developing. It was found to be markedly impaired, a finding of some interest because of the belief that the excess fluid in the distended stomach and duodenum is due to the accumulation of gastric, biliary, pancreatric and duodenal secretions as well as ingested water. Dragstedt *et al.* (1931) considered that this accumulation of secretions in the upper gastro-intestinal tract was due to gastric atony sometimes associated with partial obstruction of the duodenum by the mesentery or the dilated stomach. Malabsorption would thus be caused by failure of the secretions to reach the lower intestine. It is now known, however, that in man water is absorbed mainly from the duodenum but also from the stomach (Reitemeier *et al.*, 1957a and 1957b). The finding of impaired absorption of water in a patient with acute gastric dilatation therefore shows that the fault in the upper gastro-intestinal tract is one of absorption quite apart from one of propulsion of its contents. The additional observation that powerful gastric contractions were present as soon as five days after commencing treatment shows that return of function is extremely rapid.

Summary

A young girl with anorexia nervosa developed acute dilatation of the stomach when treated with a liquid diet which she found repugnant. This complication was thought to be at least partly psychogenic. Impaired absorption of water was demonstrated at the height of the gastric dilatation.

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