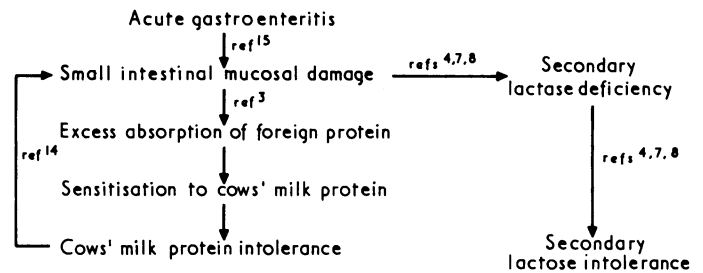


activities resulting in lactose intolerance secondary to cows' milk protein intolerance. The rise seen in the number of immunoglobulin-containing cells of the lamina propria after a positive milk challenge supports this suggestion, details of which will be given in another paper.⁹

Insufficient attention has been paid to this relation between lactose intolerance and cows' milk protein intolerance, resulting in underdiagnosis of the protein intolerance. This also explains why breast milk with its lactose content of 194 mmol/l (7 g/100 ml) may fail in treatment. A lactose-free preparation is recommended in the treatment of cows' milk protein intolerance.

The association between coeliac disease and cows' milk protein intolerance is well known.^{10,11} Three children with possible coeliac disease were included in this study. They illustrate the difficulty of distinguishing between the two conditions and the need for serial small intestinal biopsies coupled with accurate dietary information. They probably had cows' milk protein intolerance, but it is essential that a final biopsy is performed two years after the reintroduction of gluten to exclude coeliac disease, as cows' milk protein intolerance may be a precursor of coeliac disease.¹¹

Why did sensitisation to cows' milk occur in these few children, when most recover uneventfully from gastroenteritis? A partial explanation may come from the observation that 40% of the 20 infants tested had a serum IgA level below the lower limit of normal for age at the time of diagnosis and that clinical recovery tended to coincide with a return of the IgA level to normal. Gerrard *et al*¹² postulated that sensitisation to foreign protein would be apt to occur in the newborn when the infant's rate of synthesis of serum and secretory IgA is low. Taylor *et al*¹³ reported that IgA deficiency at three months is associated with the development of atopy and cows' milk protein intolerance in the offspring of reaginic parents. A family history of atopy was a notable feature in this study, although a control group was not included, and probably IgA deficiency may be a predisposing factor in the development of cows' milk protein intolerance.



Hypothesis on possible association of gastroenteritis and lactose intolerance with cows' milk protein intolerance.

A hypothesis based on the findings in this study, concerning the possible association of gastroenteritis and lactose intolerance with cows' milk protein intolerance, is indicated in the figure.

References

- Freier, S, and Kletter, B, *Australian Paediatric Journal*, 1972, **8**, 140.
- Burgess, E A, *et al*, *Archives of Disease in Childhood*, 1964, **39**, 431.
- Gruskay, F L, and Cooke, R E, *Pediatrics*, 1965, **16**, 763.
- Burke, V, Kerry, K R, and Anderson, C M, *Australian Paediatric Journal*, 1965, **1**, 47.
- Holzel, A, *Paediatric Clinics of North America*, 1965, **12**, 651.
- Kuitunen, P, *et al*, *Archives of Disease in Childhood*, 1975, **50**, 350.
- Matsumura, R, Kuroume, T, and Amada, K, *Journal of Asthma Research*, 1971, **9**, 13.
- Harrison, M, Wood, C B S, and Walker-Smith, J A, *Archives of Disease in Childhood*, 1975, **50**, 746.
- Kilby, A, *et al*, in preparation.
- Visakorpi, J K, and Immonen, P, *Acta Paediatrica Scandinavica*, 1967, **56**, 49.
- Fällström, S P, Winberg, J, and Anderson, H J, *Acta Paediatrica Scandinavica*, 1965, **54**, 101.
- Gerrard, J W, *et al*, *Acta Paediatrica Scandinavica*, 1973, suppl No 234.
- Taylor, B, *et al*, *Lancet*, 1973, **2**, 111.
- Kuitunen, P, *et al*, *Acta Paediatrica Scandinavica*, 1973, **62**, 585.
- Barnes, G L, and Townley, R R W, *Archives of Disease in Childhood*, 1973, **43**, 343.

Explanations for weight loss after ileojejunal bypass in gross obesity

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Summary

Twenty grossly obese patients underwent ileojejunal bypass operations. Measurements of calories lost in faeces showed that the malabsorption could not account for the weight loss. Furthermore, the malabsorption was not decreased two years after bypass, when weight was no longer being lost. Dietary restriction is therefore largely responsible for the weight loss and increased food intake for weight maintenance.

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Introduction

Payne and De Wind¹ in 1969 pioneered ileojejunal bypass for the treatment of gross obesity, a condition which can only rarely be treated by other means. After this operation about 40 kg of weight is lost over two years and the resulting lower weight is then easily maintained. It has usually been assumed that the weight loss is due to the malabsorption of fat and protein.² This explanation, however, has never been checked quantitatively. We report here the measurement of faecal calories before and after bypass.

Patients and methods

Seventeen women with a mean weight (\pm SD) of 117.71 \pm 14.77 kg and three men weighing 142.33 \pm 30.54 kg had a bypass operation in which 4 in (10.2 cm) of the proximal jejunum was anastomosed to 10 in (25.4 cm) of terminal ileum.³ Before operation the patients were admitted for metabolic study. The women were given diets supplying 9.489 MJ (2270 kcal) and the men diets supplying 11.620 MJ (2780 kcal) on each of five days. The energy content of the diet was verified by calorimetry and the metabolisable energy derived by the equation

TABLE II—Mean (\pm SD) energy content (MJ) of faeces each day

	Before operation	After operation*		
		4 months	12 months	24 months
Women (n = 17)	0.571 \pm 0.240 (17)	2.01 \pm 0.697 (13)	2.18 \pm 0.893 (11)	1.78 \pm 0.648 (10)
Men:				
Case 1	0.478			3.78
Case 2	0.451	3.09	1.99	
Case 3	0.344	2.95	2.53	3.84

*Values after operation did not differ significantly from each other, but all were highly significantly different from the preoperative value.
Conversion: SI to traditional units—Energy: 1 MJ \approx 239 kcal.

TABLE III—Comparison of weight loss with energy lost in faeces

	Excess energy* in faeces (MJ/day) (A)	Equivalent weight loss (kg/month) (B)	Actual weight loss (kg/month) (C)	Weight loss due to diet restriction (C-B) (kg/month)	Equivalent diet restriction (MJ/day)
<i>Women</i>					
At 4 months	1.44	1.47	5.03	3.56	3.47
At 12 months	1.61	1.65	2.04	0.39	0.380
At 24 months	1.21	1.24	0.24	-1.0	-0.974
<i>Men</i>					
At 4 months:					
Case 2	2.64	2.7	7.6	4.9	4.78
Case 3	2.61	2.6	6.4	3.8	3.70
At 12 months:					
Case 2	1.54	1.6	2.1	0.5	0.49
Case 3	2.18	2.2	1.1	-1.1	-1.19
At 24 months:					
Case 1	3.30	3.4	0.2	-3.2	-3.61
Case 3	3.49	3.6	1.7	-1.9	-1.85

*Energy after bypass - energy before bypass. Month = 30 days. 1 kg adipose tissue = 29 MJ (7000 kcal).

of Miller and Payne.⁴ These quantities were judged to be close to amounts required to maintain weight. All the diets were prepared at the beginning of the study and deep frozen. At 4, 12, and 24 months the patients were asked to repeat identical dietary studies. On each occasion faeces were collected for five days, homogenised, and aliquots dried. Calorimetry was performed in triplicate on 0.5 g of the dried samples in a bomb calorimeter⁴ using benzoic acid as standard.

Results

Cumulative weight loss is shown in table I. The most rapid loss of weight occurred in the first six months, but during the second year very little was lost. All patients found it very difficult to eat the diet at four months, but seemed to manage more easily at 12 and 24 months.

Table II shows the energy content of faeces. In the women there was a nearly fourfold increase in faecal calorie output after bypass and no significant change between four, 12, and 24 months. The faecal calorie loss in the men was larger and showed the same trend, but the numbers were too small to draw justifiable conclusions. Clearly differences in malabsorption—that is, energy loss in faeces—could not have accounted for the different rates of weight loss during the two years of study.

In table III we have compared the weight loss with the energy lost in faeces. For these calculations we assumed that after the bypass the energy requirements of our patients did not decrease; this assumption seems justifiable since they all became more active. We also assumed that the weight loss was due to the catabolism to adipose tissue with an energy value of 29 MJ (7000 kcal/kg). At four months weight loss far exceeded that accounted for by faecal calorie loss. Reduced dietary intake must have been responsible for the difference, and it amounted to 3.47 MJ/day (830 kcal/day) among the women. At 12 months the rate of weight loss was much reduced and was largely accounted for by the faecal energy loss. When weight was stable at 24 months the faecal calorie loss had to be made good by a dietary increase. Dietary

histories taken by a dietitian confirmed that after operation the patients ate less than before and that by two years their food intake had increased again. Nevertheless, the calorie deficits calculated from dietary histories were much less than those calculated in table III. This was not surprising in view of the inaccuracy of such dietary surveys.

Discussion

These studies show that the assumption that ileojejunum bypass produces weight loss predominantly through malabsorption is unfounded. The calories lost in faeces do not decrease with time and amount to about 1/7 of the 9.489 MJ eaten by the women and between 1/3 and 1/4 of the 11.620 MJ eaten by the men. These findings suggest that the malabsorption depends on the amount of food taken. Further studies are required to clarify this relation. In no case, however, can the faecal loss of calories account for the amount of weight lost.

These results also show that adaptation does not work primarily by reducing the malabsorption of food. It follows that both weight loss and eventual weight maintenance must occur through dietary regulation: weight loss by reduction of food intake and maintenance by an increase of food intake. Our patients invariably found the experimental diet difficult to take in the early postoperative months. Vomiting was common at this time. The reason for this difficulty is most probably the slowed transit time through the small bowel which Quaade *et al*⁵ have shown. This results in uncomfortable distension of the proximal small bowel and stomach, and in this way the bypass seems to modify eating behaviour. Some patients adapt to this situation very quickly and lose only small amounts of weight, and some never adapt: they vomit frequently, lose too much weight, and eventually require gut reconstitution. Most, however, take about six to 12 months to learn how to eat amounts sufficient to maintain weight. During this time they lose much weight.

TABLE I—Mean cumulative weight loss (\pm SD)

	Women		Men	
	No	Weight loss (kg)	No	Weight loss (kg)
4 Months	16	20.12 \pm 6.29	2	27.90 \pm 3.54
6 Months	17	27.11 \pm 8.74	3	31.9 \pm 10.94
12 Months	15	39.35 \pm 10.54	3	41.37 \pm 13.96
24 Months	14	42.26 \pm 13.09	3	50.23 \pm 14.50

References

- Payne, J D, and De Wind, L I, *American Journal of Surgery*, 1969, **118**, 141.
- British Medical Journal*, 1971, **4**, 247.
- Gazet, J-C, *et al*, *British Medical Journal*, 1974, **4**, 311.
- Miller, D S, and Payne, P R, *British Journal of Nutrition*, 1951, **13**, 501.
- Quaade, F J, *et al*, *Scandinavian Journal of Gastroenterology*, 1971, **6**, 537.