swelling. Most symptoms were rated on a four-point scale from nil to severe. Women who rated moderate or severe were included in the trial provided that their periods were regular and they were not taking oral contraceptives or sex hormones.

Thirteen patients completed the trial. It had a double-blind, crossover design, and patients were allocated at random to receive either bromocriptine (2.5 mg) or identical placebo tablets. The patients were instructed to take one tablet (bromocriptine 2.5 mg or placebo) daily 10 days before the start of their next period. Ten days before the subsequent period they were given the other tablets.

The total scores for each course of treatment are shown in the table. Statistically there was no significant difference between the two treatments or order of treatments.

Mean age and total scores ($\pm$ SE) of patients taking bromocriptine and placebo for premenstrual syndrome (PMS)

Group	Mean age in years	PMS total scores	
		Placebo phase	Active phase
Placebo first (n=5)	34.2 $\pm$ 3.5	6.0 $\pm$ 1.0	6.4 $\pm$ 1.4
Bromocriptine first (n=8)	33.2 $\pm$ 3.4	6.6 $\pm$ 1.1	5.0 $\pm$ 0.8
Combined series (n=13)	33.6 $\pm$ 2.5	6.4 $\pm$ 0.7	5.5 $\pm$ 0.7

Comment

Our results clearly do not support the theory that prolactin is related to premenstrual mood disturbances or to the symptoms of swelling or headaches. They do not agree with those of Benedek-Jaszmán *et al.*,³ who observed significant alleviation of the premenstrual syndrome after bromocriptine treatment. The dosage used in their study, however, was 2.5 mg twice daily, which is double the dose used in our study. We decided to use a smaller dosage after a pilot study had shown that most of the women were unable to tolerate 2.5 mg twice daily. We also found that a single 2.5 mg oral dose was sufficient to reduce plasma prolactin to a significantly low concentration after two hours, and that the effect lasted for six to eight hours.⁴

We thank Drs E Saphier, S V Kar, O Meerapfel, and G Wills for invaluable help in the trial, and Mr J Bailey for the statistical evaluation of the data. We are grateful to Sandoz Products Limited for supplying the bromocriptine and placebo tablets.

¹ Horrobin, D F, *Prolactin: Physiological and Clinical Significance*. Lancaster, Medical and Technical Publishing Company, 1973.

² Coppen, A, and Kessel, N, *British Journal of Psychiatry*, 1963, **109**, 711.

³ Benedek-Jaszmán, L J, Lequin, R M, and Sternthal, V, *Acta Endocrinologica (København)*, 1975, Suppl No 193, p 29.

⁴ Ghose, K, and Coppen, A, unpublished observations.

(Accepted 19 October 1976)

Medical Research Council Neuropsychiatry Laboratory, West Park Hospital, Epsom, Surrey

K GHOSE, MRCP, senior research registrar

A COPPEN, MD, FRCPsych, consultant psychiatrist and member of MRC external staff

Symptomatic magnesium deficiency after small-intestinal bypass for obesity

The first reported successful massive intestinal resection was in 1881.¹ Since then small intestinal bypass operations have gained a limited place in the management of obesity. Common complications of such operations² include vomiting, diarrhoea, malabsorption, liver failure, pancreatic insufficiency, and disturbances of potassium and calcium balance. Magnesium deficiency, however, is rare^{3 4}; indeed, Shibata⁵

found no overt signs of magnesium deficiency in his series. I report a case of symptomatic hypomagnesaemia occurring after jejunoileal anastomosis for obesity.

Case report

A 40-year-old woman who weighed over 160 kg was admitted to hospital for a small-intestinal bypass operation. Results of all preoperative haematological, biochemical, and endocrine investigations were normal. A 30 cm-30 cm jejunoileal anastomosis was performed, from which she recovered uneventfully, and she was discharged two weeks later. When seen at six weeks as an outpatient she had lost 20 kg but was having five bowel movements daily.

Sixty-two days postoperatively she was readmitted as an emergency case. She complained of weakness and dizziness, with "pins and needles" in her hands and feet. On examination Chvostek's sign and Trousseau's phenomenon were seen, and latent tetany due to hypocalcaemia was diagnosed. Serum calcium, magnesium, and potassium concentrations were 1.35 mmol/l (5.4 mg/100 ml), 0.35 mmol/l (0.85 mg/100 ml), and 2.2 mmol (mEq)/l respectively (see table). She was given 10 ml 10% calcium gluconate intravenously and oral calciferol 1.25 mg (50 000 IU) three times daily, but the serum calcium continued to fall. On day 68 she again developed paraesthesiae and was given a further 10 ml 10% calcium gluconate. The serum potassium was 2.50 mmol (mEq)/l and magnesium was 0.40 mmol/l (0.9 mg/100 ml) and a new diagnosis of hypomagnesaemia was made. Calcium supplements were stopped the next day and intramuscular injections of 2 ml 50% magnesium sulphate five times daily were started. This rapidly controlled her symptoms and corrected her electrolyte imbalance within five days.

Serum electrolyte concentrations in a patient before and after treatment with 10% calcium gluconate, calciferol, and 50% magnesium sulphate

Day	Calcium (normal range 2.25-2.65 mmol/l)	Magnesium (normal range 0.7-0.9 mmol/l)	Potassium (mmol/l)
0	2.22	1.05	3.75
62	1.35	0.35	2.20
65	1.42		2.50
68	1.20		
69	1.22	0.40	2.50
72	1.62	1.40	3.10
76	2.15	0.80	3.30

Conversion: SI to traditional units—Calcium: 1 mmol/l $\approx$ 4 mg/100 ml. Magnesium: 1 mmol/l $\approx$ 2 mg/100 ml. Potassium: 1 mmol/l $\approx$ 1 mEq/l.

She was started on Lomotil (diphenoxylate hydrochloride and atropine sulphate), which controlled her diarrhoea, and then discharged.

At follow-up after six months she was asymptomatic with normal electrolyte concentrations without any further treatment.

Comment

This woman presented with calcium-resistant latent tetany due to magnesium deficiency after jejunoileal anastomosis. Dietary magnesium is uniformly absorbed throughout the small intestine with little or no absorption in the large intestine. The acute magnesium deficiency was probably caused by increased faecal loss of magnesium. After a massive resection the small intestine adapts by dilatation, elongation, and villous hypertrophy. In this case the increase in available surface area for absorption because of adaptation together with control of the diarrhoea presumably resulted in magnesium and calcium homeostasis.

I thank Mr J Leslie for permission to report this case, and Mrs J Cragg for help in preparing the report.

¹ Haymond, H E, *Surgery, Gynecology, and Obstetrics*, 1935, **61**, 693.

² Bondar, G F, and Pisesky, W, *Archives of Surgery*, 1967, **94**, 707.

³ Fletcher, R F, *et al*, *Lancet*, 1960, **1**, 522.

⁴ Swenson, S A, jun, *et al*, *American Journal of Surgery*, 1974, **127**, 250.

⁵ Shibata, H R, MacKenzie, J R, and Long, R C, *Archives of Surgery*, 1967, **95**, 413.

(Accepted 7 October 1976)

Leicester General Hospital, Leicester

A LIPNER, MB, ChB, house surgeon (present appointment: senior house officer in orthopaedics, Frenchay Hospital, Bristol BS16 1LE)