

Mandometer treatment of Australian patients with eating disorders

John Court, Cecilia EK Bergh and Per Södersten

TO THE EDITOR: The Mandometer treatment for patients with eating disorders (Box 1), developed at the Karolinska Institute in Stockholm, was brought to the attention of the public and the profession in Australia through Norman Swan's Health Report in 2003.¹ Since then, 40 patients from Australia have received treatment using this method, 29 of whom were treated in Stockholm, seven in Melbourne and four in San Diego.

In a randomised controlled trial, Mandometer treatment brought 75% of patients into remission in an average 14 months, with a relapse rate of about 10% during 5 years of follow-up.² There are seven criteria for remission, including normal eating behaviour, normal body mass index (BMI) and physical parameters, and remission of psychiatric symptoms. Patients must have returned to school or work, be comfortable with their body weight, and have avoided binge eating and vomiting for at least 3 months. Patients fulfilling five of these criteria are considered in partial remission.

Of the 40 (39 female) patients reported here, 27 patients (68%) were diagnosed according to criteria of the *Diagnostic and statistical manual of mental disorders*, 4th edition, with anorexia nervosa, seven (17%) with bulimia nervosa, and six (15%) with an

eating disorder not otherwise specified. The median age of patients was 18.7 years (range, 10.7–39.3 years). On admission, they had been ill for a median period of 5 years (range, 1–25 years) and they had had up to 20 previous unsuccessful episodes of treatment (median, 3 episodes).

Fourteen patients (35%) fulfilling five remission criteria have returned to Australia for follow-up, but have not yet returned to school or work. Eight patients (20%) are in full remission, 11 (28%) are in treatment, and seven (17%) withdrew from treatment before completion, some after only a few days. All patients who are treated in Stockholm and San Diego are followed up in Melbourne, and outpatient treatment is also offered at the Melbourne clinic.

Of the 27 patients with anorexia, 16 have entered full or partial remission, with marked improvement in all parameters (Box 2). Six achieved remission and 10 partial remission in 220 (range, 129–257) and 220 (range, 168–570) days, respectively. The five anorexic patients who dropped out of the treatment had low BMI (11.3, 13.5, 13.8, 14.6 and 15.7 kg/m²), had been ill for 4, 5, 8, 14 and 25 years, and had had up to nine previous episodes of treatment.

This report concerns severely ill Australian patients who had previously undergone many episodes of care. These preliminary results are encouraging, given the known resistance of these disorders to treatment.^{4,5} Although the outcomes reported here appear to be better, comparison with other

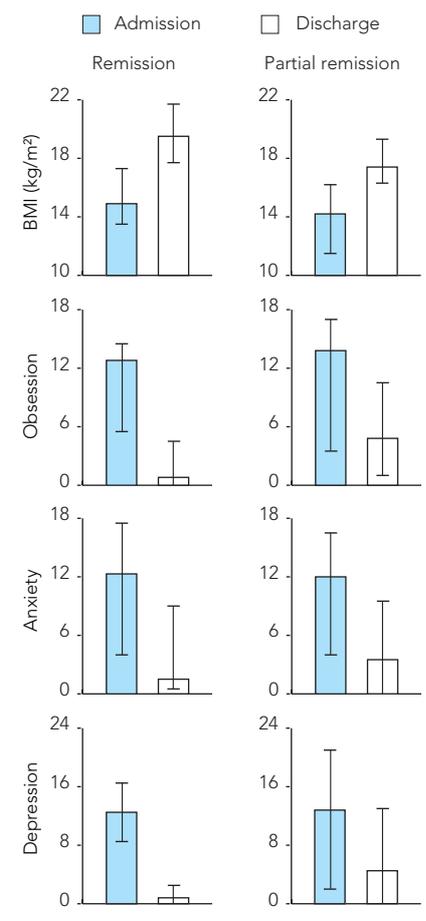
methods used in Australia should be made in a randomised controlled trial. The Melbourne Mandometer Clinic is willing to participate in such a trial.

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Competing interests: John Court is employed by Mandometer Melbourne and receives a salary. Cecilia Bergh and Per Södersten are part-owners of AB Mando, Sweden.

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2 Body mass index (BMI) and obsession, anxiety and depression scores in six Australian anorexic patients treated to remission and in 10 anorexic patients treated to partial remission

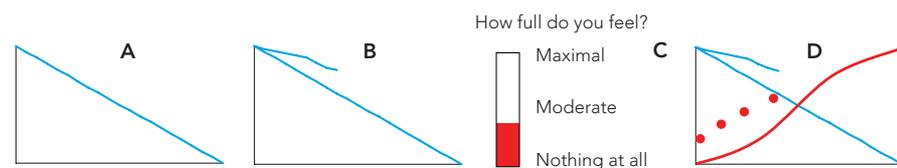


Psychiatric symptoms were evaluated by the Comprehensive Psychopathological Rating Scale.³ Values are medians and ranges. There were no significant differences between the groups. ♦

1 Mandometer treatment has four interventions

1. The patient re-learns how to eat using Mandometer, a computerised procedure that provides feedback during meals (Figure). In about 5–6 months, anorexic patients are able to eat a normal meal and perceive a normal level of satiety after practising eating gradually larger meals. Bulimic patients are similarly trained, but they are able to eat normal amounts of food from the beginning of treatment. After using Mandometer, patients are trained to eat socially.
2. Patients rest in warm rooms after each meal. Warmth has an anxiolytic effect and prevents compensatory hyperactivity and vomiting after the meal.
3. Physical hyperactivity is prevented by use of wheelchairs and warming jackets between meals.
4. Patients are trained to re-learn social skills.

Manuals describing the details of treatment are used at all clinics.



The patient can adapt her or his eating rate to a linear curve displayed on the monitor (A), because she or he sees her or his own eating rate emerging (B). At regular intervals, the patient rates her or his feeling of fullness on a scale that appears on the monitor (C) and adapts her or his ratings to an s-shaped curve (D). The axes have no numerical values during training. ♦

LETTERS

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Anorexia nervosa and senna misuse: nephrocalcinosis, digital clubbing and hypertrophic osteoarthropathy

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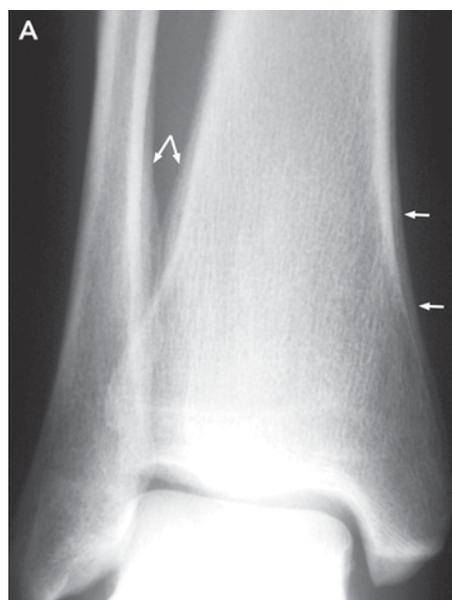
TO THE EDITOR: Senna is widely used in laxatives, but the results of its misuse are not inconsequential. We describe a 36-year-old woman admitted with hypercalcaemia and renal failure. She had a 6-year history of anorexia nervosa and ingestion of 50–100 senna tablets daily for weight loss. Examination revealed clubbing of the fingers and toes, a body mass index (BMI) of 17.7 kg/m² and postural hypotension. Laboratory findings on admission are shown in Box 1. Results of autoimmune studies and protein electrophoresis, and the serum angiotensin-converting enzyme level were normal. Parathyroid hormone-related peptide was absent. She had a bland urinary sediment, trace proteinuria (150 mg/24 h; reference range, < 150 mg/24 h) and a urine pH of 5.0 (physiological range, 4.5–8.0).

Computed tomography scans did not detect malignancy or infection, but showed bilateral medullary renal calcifications. Renal biopsy confirmed extensive nephrocalcinosis and the absence of primary glomerular disease. A skeletal survey showed prominent periosteal reaction and new bone formation at the ends of long bones (Box 2, A). A bone scan revealed increased tracer uptake in a pattern consistent with hypertrophic osteoarthropathy

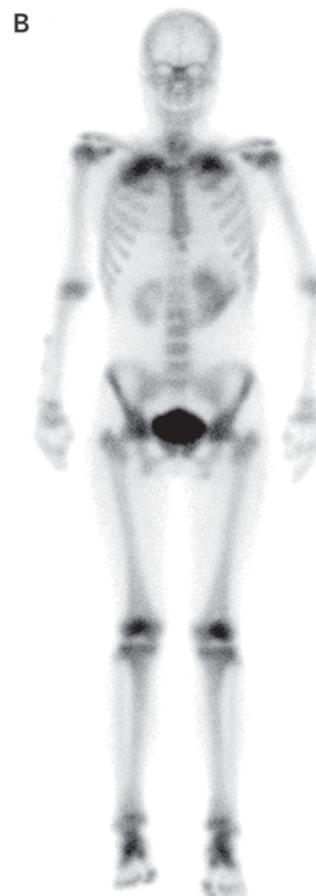
1 Biochemical parameters on admission before treatment

Parameter	Patient value	Reference range
Serum concentrations of:		
Sodium (mmol/L)	139	135–145
Potassium (mmol/L)	3.2	3.5–5.0
Magnesium (mmol/L)	0.74	0.80–1.50
Chloride (mmol/L)	102	95–107
Bicarbonate (mmol/L)	24	21–30
Urea (mmol/L)	8.6	2.5–7.8
Creatinine (μmol/L)	166	40–120
Albumin (g/L)	28	35–45
Corrected calcium (mmol/L)	2.93	2.20–2.60
Ionised calcium — pH adjusted (mmol/L)	1.51	1.14–1.29
Phosphate (mmol/L)	2.58	0.80–1.20
25-hydroxyvitamin D (nmol/L)	23	55–108
Parathyroid hormone (pmol/L)	1.0	1.1–7.7
Rates of:		
Creatinine clearance (mL/min)	20	90–150
Urine sodium excretion (mmol/day)	28	40–100
Urine potassium excretion (mmol/day)	29	50–140
Urine calcium excretion (mmol/day)	1.56	2.0–7.5

2 Ankle x-ray and full bone scan



A: Ankle x-ray showing periosteal reaction and new bone formation along the ends of the tibia and fibula (arrows).



B: Bone scan showing symmetrical increased tracer uptake at both the proximal and distal ends of long bones, hands and feet. Disease activity is more prominent in the lower limbs. Lung uptake is probably the result of metastatic calcification, which can be predominantly apical¹ and not visible on x-ray or conventional computed tomography.² ♦