elsewhere in Europe have reported a high incidence of CJD in dairy farmers. The apparent excess of recent cases of CJD in the UK occurring in people exposed to cattle affected with BSE has increased speculation that CJD may result from the transmission of BSE to human beings, although the similarly increased incidence of CJD reported in dairy farmers from European countries in which BSE is rare[5] is reassuring. There may be ascertainment bias; concern that individuals occupationally exposed to BSE may be at higher risk of contracting CJD leading to the increased recognition of the disease in farmers.

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BCG revaccination against tuberculosis

SIR—The Karonga Prevention Trial Group (July 6, p 17)[1] report a controlled trial of single and repeated BCG against tuberculosis and leprosy in a population of over 120 000. In this trial, individuals lacking a BCG scar were allocated to BCG alone or BCG plus killed *Mycobacterium leopard*, and individuals with a BCG scar to either placebo, BCG alone, or BCG plus killed *M leprae*. Previous results had shown that a single BCG dose protected against leprosy but not tuberculosis in this population. The trialists concluded that a second BCG dose added appreciable protection against leprosy but not against tuberculosis.

The trial group presented a subset of the data for HIV-infected patients with tuberculosis (table). Cases of tuberculosis were defined as probable if they had a positive sputum bacteriology with more than ten bacilli per 100 fields and as certain if they had at least two positive sputum tests, at least one of which was by culture. They comment that the difference in distribution of certain tuberculosis in this subset may be due to chance. This is a sensible reaction to any unexpected result, especially one based on so few cases. By contrast, Reider in his accompanying commentary[2] states that the results show "a distinct possibility that . . . BCG may harm [HIV infected persons] by increasing further their risk of tuberculosis". He goes on to discuss the implications for policy and the research agenda. He ignores the probable cases and overinterprets an isolated, unexpected finding based on 15 cases. His conclusions could affect research and policy.

The table shows a very small data set, in which the frequency of tuberculosis is the same among recipients of BCG and of placebo (RR=1·25, p=0·51), but in which recipients of BCG seem more likely to be classified as certain. If we were pushed to interpreting these data as causal, we might suggest that BCG revaccination causes HIV-infected persons with tuberculosis to be more likely to excrete bacilli, therefore shifting them from the probable to the certain tuberculosis category. This explanation has the added virtue of clarifying why there are fewer BCG vaccinated cases in the probable group. Who are the probable cases? Excluding the possibility of laboratory cross-contamination, and the odd atypical mycobacteria, most are likely to be true cases of tuberculosis.

Cases of HIV+ TB	Placebo	BCG	RR
All cases	15	19	1·25 (0·64–2·48)
Certain cases	1	14	13·98 (1·82–107)
Probable cases	14	5	0·36 (0·13–1)

RR=relative risk

Table: **Cases of tuberculosis in HIV infected persons with a previous BCG scar, by BCG, or placebo status**

The appropriate response to the data is to take note and to wait for further studies addressing this question with larger numbers; with a recommendation that new results be presented in a comparable way.

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1 Karonga Prevention Trial Group. Randomised controlled trial of single BCG, repeated BCG, or combined BCG, and killed *Mycobacterium leprae* vaccine for prevention of leprosy and tuberculosis in Malawi. *Lancet* 1996; **348:** 17–24.
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A new treatment of anorexia nervosa

SIR—We have suggested that analyses of anorexia nervosa should concentrate on reduced food intake and enhanced physical activity.[1] With use of a method based on this view, we have treated eight women aged 14–23 (median 17) years, consecutively referred to us with anorexia nervosa diagnosed by the referring physician, who had been treated up to twice before without success.

The patient eats from a plate placed on a balance and the weight loss of the plate is recorded by computer. At 1 min intervals a scale from none (0) to maximum (10) appears on a monitor and the patient records her perception of satiety. This device, Mandometer, determines eating rate and satiety. A linear curve, steeper than that generated by the patient is then displayed on the monitor and the patient adapts her rate of eating, which emerges on the monitor during a meal, to the curve. Successive 20% increases in meal size and curve slope are presented. Similarly, the patient adapts her perception of satiety to less steep linear curves. Daily physical activity is monitored with the Actigraph,[2] which records arm movements per 16 s, and the patient is taught to reduce her activity at the times of its peaks. No drugs are used.

After a median of 7 months (range 3·5–14) of treatment the body mass index (BMI) was normal (19·6 [18·8–22·1] *vs* 14·7 [11·1–16·8] kg/m,^{2} p<0·01, Wilcoxon test) and the amount of food consumed had more than doubled compared with before treatment (335 [250–465] *vs* 135 [50–150] g daily, p<0·01). The perception of satiety (4·9 [3·0–5·2] *vs* 9·5 [8·0–10·0], p<0·01) and anxiety[3] (3·5 [1·0–6·5] *vs* 15·5 [5·5–20·5], p<0·01) had decreased by at least 50%. All patients had bradycardia and were amenorrhoeic before treatment. Heart rate was normal and 75% menstruated either during or after treatment. The increased eating and decreased satiety and activity is shown in the figure. The patients were re-examined at 3-month intervals. There were

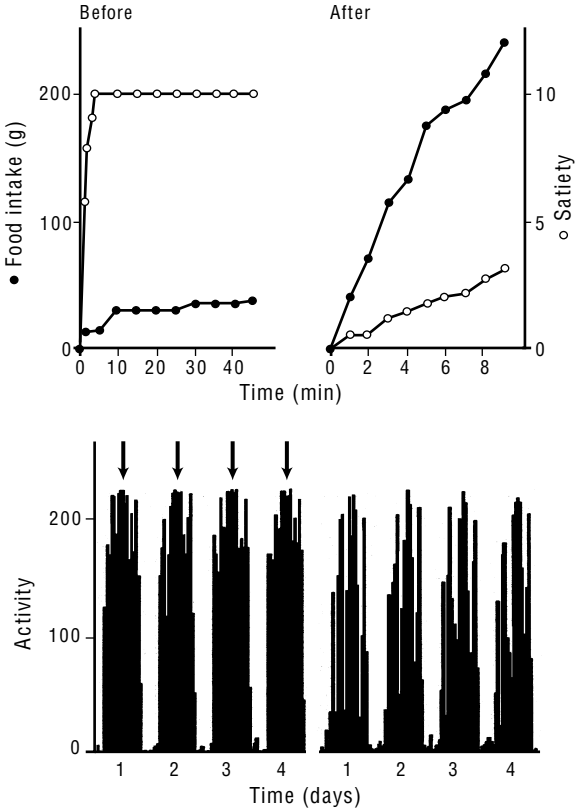

Figure: **Cumulative food intake and satiety (top) in an anorexia patient and physical activity (bottom) in another patient before and after treatment**
Times of peak activity (arm movements per 16 s) before treatment are indicated by arrows.

no relapses in 12 (9–24) months. Two patients have remained healthy for 24 months. No complications (such as bulimia), have developed and all patients are back to educational or professional activities.

These results show that Mandometer and the Actigraph may return eating, satiety, and physical activity in anorexic patients to normal. Although interindividually stable, these measures vary between individuals and it is not known whether patterns typical of anorexics exist. The decrease in anxiety, a concomitant to the behavioural normalisation, supports the suggestion that "psychopathology" is an effect, not a cause, of starvation.[1] Our method must be further evaluated and the patients followed up. However, it is unlikely that we have treated a "mild" form of anorexia nervosa, because the patients had previously been treated without success and two were severely emaciated (BMI 11·2 and 12·7) at the start of treatment.

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Improvement of stiff-man syndrome with vigabatrin

SIR—Stiff-man syndrome (SMS) is a neurological disorder characterised by progressive stiffness and painful spasms often provoked by stress and being startled.[1] We report improvement of a man with SMS after treatment with vigabatrin.

A 42-year-old man with insulin-dependent diabetes mellitus for 12 years complained of paroxysmal painful muscle spasms in his left leg. Over the years these spasms extended to his other leg, lower back, and finally his complete trunk. When these spasms appeared almost continuously during the day, running, cycling, and even bending forward became impossible. Clinical examination revealed a lumbar hyperlordosis, and board-like rigidity of the trunk and proximal legs. He walked with a hobbling, stiff-legged gait. Loud noises provoked involuntary contractions lasting several minutes. Electromyography showed continuous motor-unit activity at rest in all affected muscles, which decreased after diazepam. In serum, autoantibodies against glutamic acid decarboxylase (GAD) were present. These findings are compatible with SMS.[1] Diazepam had to be discontinued because of drowsiness and impotence. Carbamazepine was ineffective. Soon after we started vigabatrin, the patient greatly improved. He was again able to run, cycle, and perform one of his favourite hobbies, dancing, even in crowded places. His improved condition has now been stable for 4 years on vigabatrin 3 g daily without side-effects.

SMS is considered to be an autoimmune disease, with autoantibodies directed against (gamma-aminobutyric acid) (GABA)-ergic neurons, present in 60% of these patients.[2] The principal autoantigen is thought to be GAD,[2] which catalyses the conversion of glutamate to GABA, and is found in the central nervous system and in pancreatic β-cells. GAD antibodies are associated with SMS and also with insulin-dependent diabetes mellitus, and may prevent GABA production. Since vigabatrin increases GABA concentrations by inhibiting its breakdown, we hypothesised that it may diminish muscle spasms and rigidity in patients with SMS.

Improvement of spasms and rigidity in SMS have been reported after treatment with diazepam, carbamazepine, baclofen, sodium valproate, plasmapheresis, and intravenous immunoglobulin.[3–5] The improvement in our patient's neurological condition, which has lasted for more than 4 years, may be caused by treatment with vigabatrin rather than a remission by chance. This observation has to be the subject of further investigation before definitive conclusions can be drawn.

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