nervosa. She was subject to recurrent hypoglycaemia and on one occasion had a grand mal fit. After a joint medicopsychiatric admission she was followed up in both clinics in conjunction. She remained at about 42 kg (standard 50 kg) until just after taking O levels at 16; she lost 4 kg in a few weeks, and was subsequently found dead in bed.

A few missed periods and fluctuations in weight are commonplace in adolescence. Primary anorexia nervosa is liable to arise when lack of menses and weight loss come specifically to assuage high levels of adolescent anxiety. The combination of the illusion of self control, the safety of emotional regression, and reaffirmation of parental concern may be rewarding enough to maintain and intensify weight-reducing behaviour. In our second patient the strains of puberty and adolescence were added to the psychological disturbance common in unstable diabetes[3]—leading to overdose, wristcutting, and finally anorexia nervosa. The reason for her death was uncertain but she did not have autonomic neuropathy, which can predispose to cardiorespiratory arrest in diabetes.[4] It seems that the association between anorexia nervosa and diabetes mellitus is neither excessively rare nor particularly common, but what would be expected by chance. However, since the secondary forms of anorexia nervosa merge into various neurotic reactions it is probable that a search among diabetics would produce an appreciable number with neurotic behaviour which included an obsession with weight.

JOAN GOMEZ
PETER DALLY

Department of Psychiatry

A J ISAACS

Department of Medicine,
Westminster Hospital,
London SW1P 2AP

[1] Dally P, Gomez J, Isaacs AJ. *Anorexia nervosa*. London: Heinemann, 1979.
[2] Jarrett RJ, Keen H. *Lancet* 1976;ii:1009-12.
[3] Gale E, Tattersall R. *Hospital Medicine*. 1979;**22**: 589-97.
[4] Page M McB, Watkins PJ. *Lancet* 1978;i:14-6.

Guar crispbread in the diabetic diet

SIR,—In 1978, largely from hospital metabolic ward studies, we concluded that guar crispbread was likely to be a useful adjunct to diabetic treatment.[1] Later, Cohen and Martin reported that they had failed to find any beneficial effect when feeding supplements of guar or bran to poorly controlled obese diabetics.[2] We replied[3] that the form of supplement and its relationship to the meal may be all important—that is, the fibre should be taken mixed with the carbohydrate portion of the meal.

Subsequently Mr Nathan Mantel (15 December, p 1589) stated that he found some "disturbing aspects" in our original report with "several numerical discrepancies in the data."

Firstly, he notes that the large mean reduction in urinary glucose output (38%, $p < 0.002$) was due largely to a fall in one individual. However, exclusion of this individual still results in a mean fall of 33% ($p < 0.01$) for the remaining eight patients. Secondly, he comments that smaller urinary glucose concentrations were seen in a patient prior to hospital admission than were measured on the metabolic ward. It is well recognised that some patients, especially those who lead active lives, show greatly increased glycosuria when such activity is restricted by a period of hospitalisation. Thirdly, he suggests the possibility that our very limited follow-up study was terminated when individual patients showed an improvement. The follow-up data in our original paper were preliminary and we gave the most up-to-date figures we had at the time. Not all patients were started synchronously; hence the disparity in times.

We are also asked for data on "the entire course of the results for each patient," and these have now been published (7 June, p 1353).

Though we acknowledge deficiencies in our studies, we would not wish Mr Mantel's comments to obscure what we consider to be a major problem of ensuring that guar is distributed throughout the day with the carbohydrate portion of the meals. To this end, if guar is to be used successfully, further guar products will have to be developed to supplement the crispbread.

DAVID J A JENKINS

Department of the Regius Professor of Medicine,
Radcliffe Infirmary,
Oxford

RODNEY H TAYLOR

Department of Gastroenterology,
Central Middlesex Hospital,
London NW10

THOMAS M S WOLEVER

University Laboratory of Physiology,
Oxford

SUSAN BACON

Nuffield Orthopaedic Centre,
Oxford

T DEREK R HOCKADAY

Radcliffe Infirmary,
Oxford

D J REYNOLDS

Department of Chemical Pathology,
Royal Postgraduate Medical School,
London W12

[1] Jenkins DJA, Wolever TMS, Nineham R, *et al*. *Br Med J* 1978;ii:1744-6.
[2] Cohen M, Martin FIR. *Br Med J* 1979;i:616.
[3] Hockaday TDR, Jenkins DJA, Wolever TMS, Nineham R, Taylor RH, Bacon S. *Br Med J* 1979;i:1353.

Dietary management of maturity-onset diabetes

SIR,—Ms E Anne Wilson and others have recently described their very considerable experience in Belfast with low-carbohydrate diets for diabetic patients (7 June, p 1367). Such diets seem to have been recommended almost universally for diabetics in Western countries at least since the eighteenth century; and many authorities, at least in the United Kingdom, are clearly still recommending them. We would agree with Ms Wilson and her colleagues that achieving ideal body weight (preferably by means of a "simple approach") is undoubtedly the most important aim of a diet for maturity-onset (type II) diabetes, and that if it is achieved this obviates the need for drug therapy in the great majority of cases. However, we wonder whether in 1980 a low-carbohydrate diet is still the best means of achieving this.

Research done elsewhere[1] and in Oxford[2 3] has suggested that diets relatively high in total carbohydrate (derived chiefly from cereal and vegetable sources) and dietary fibre result in lower fasting and preprandial blood glucoses and lower cholesterol levels than a low-carbohydrate diet. There is still much to be learned about the long-term acceptability of such diets, but in Northern Ireland, where intake of starches is usually high, it may be that a simple diabetic diet could be based on the "high" rather than the "low" carbohydrate principle.

J I MANN

University Department of Community Medicine and General Practice,
Oxford 0X1 3QN

H C R SIMPSON

Diabetes Research Laboratory,
Radcliffe Infirmary,
Oxford 0X2 6AE

[1] Kiehm TG, Anderson JW, Ward K. *Am J Clin Nutr* 1976;**29**:895-9.
[2] Simpson RW, Mann JI, Eaton J, Moore RA, Carter R, Hockaday TDR. *Br Med J* 1979;i:1753-6.
[3] Simpson RW, Mann JI, Eaton J. *Br Med J* 1979;ii: 523-5.

Pseudomonas septicaemia

SIR,—The failure of antibiotic therapy to reduce the mortality of *Pseudomonas* bacteraemia (discussed in your leading article of 24 May, p 1240) could be the result of too enthusiastic therapy, producing too much endotoxin for debilitated subjects.

This possibility was first put forward, even before the discovery of penicillin, in 1936 by the late James Reilly[1] in the course of his researches into typhoid fever and confirmed following the introduction of chloramphenicol.[2] In several instances patients receiving loading doses of this antibiotic suffered severe cardiovascular collapse, which in some cases was fatal. The possibility that this might arise from endotoxin released from the typhoid bacilli was suggested by Galpin in 1949[3]. A few years later Reilly confirmed his hypothesis by showing that animals infected with Gram-negative organisms that received a weight-equivalent loading dose of chloromycetin consistently underwent fatal cardiovascular collapse although all the organs were sterilised.[4]

A summary of this work appeared in 1978,[5] a plea being made for discontinuing the use of loading doses of antibiotics in treatment of Gram-negative bacteraemia and for reducing dosage or withdrawing it if signs of toxaemia appear. It is unfortunate that there is no easy method of estimating blood endotoxin levels. However, as function of the kidney is always affected before that of any other organs by endotoxin,[6] a rise in blood creatinine could provide an adequate warning sign.

After all, typhoid is a Gram-negative infection and the pathological features, so carefully studied by Reilly and his colleagues, were shown by them to be a constant feature of all Gram-negative infections.[7]

D A BUXTON HOPKIN

London SW3

[1] Reilly J, Rivalier J, Compagnon A, Phan HC, Friedman E, Du Buit H. *Ann Med* 1936;**39**:138.
[2] Reilly J, Compagnon A, Tournier T, Bastin R, Du Buit H. *Ann Med* 1950;**51**:597-654.
[3] Galpine JF. *Br Med J* 1949;ii:1047-8.
[4] Reilly J, Compagnon A, Tournier P, Du Buit H. *Ann Med* 1954;**55**:22.
[5] Hopkin DAB. *Lancet* 1978;ii:1193-4.
[6] Reilly J, Compagnon A, Laporte A, Du Buit H. *Le role du systeme nerveux en pathologie renale*. Paris: 1942: 1, 2, 15, 16.
[7] Hopkin, DAB, Laplane, R. *Ann R Coll Surg* 1978; **60**:108-16.

SIR,—A shift of emphasis away from newer antibiotics that are only impressive in vitro towards consideration of provision of appropriate antibody is not only welcome, as