"It is no longer justifiable for one physician to spend twice as much money as another on treating patients with similar diseases,"³ yet it is almost embarrassing to report that only the private sector seems to be using well researched, humane, and acceptable techniques which reduce the duration of opiate withdrawal from the usual two to three weeks to two to three days or less.^{4,6}

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Fibre and patients with diabetes

SIR,—Ms V A Beattie and others (23 April, p 1147) state that viscous fibres have no therapeutic benefit for type II diabetic patients when added to a low energy, high carbohydrate, low fat diet. In their study the lack of washout periods between different treatments is unwise and could lead to misleading results, especially of concentrations of glycated haemoglobin. The method of giving the guar gum is not clear. Was it added to the main meals or given otherwise? Fasting blood glucose concentrations at the end of the run in periods were not grossly raised, an important factor for a beneficial effect from guar.

We have recently conducted an eight week randomised, non-crossover study of 15 g of guar mini tablets given daily before meals compared with placebo in 20 overweight non-insulin dependent (type II) diabetic patients who failed to respond to diet. Guar was well tolerated, although eight of 12 patients receiving guar experienced mild gastrointestinal upsets, which settled in one to two weeks in all but one patient.

Guar significantly reduced the fasting plasma glucose concentration (14.05 (SD 2.61) before treatment v 11.01 (2.02) mmol/l after treatment), postprandial glucose concentrations (17.50 (3.10) v 14.32 (3.60) mmol/l), glycated haemoglobin concentrations (9.96 (1.17) v 7.86 (1.00)), total cholesterol concentrations (7.44 (1.90) v 6.78 (2.20) mmol/l), and triglyceride concentrations (3.8 (2.18) v 2.99 (1.47) mmol/l) (in each case $p < 0.05$). No changes were observed in these variables with placebo.

Six out of nine patients in the guar group lost their symptoms of hyperglycaemia during the study compared with one of eight in the placebo group. Our results show a therapeutic benefit conferred by guar gum, and we conclude that it is effective in patients whose blood glucose concentrations remain high despite dietary measures.

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SIR,—Non-viscous, non-digestible materials in the diet have no important influence on the meta-

bolic control of diabetes. In contrast, viscous fibre, when intimately mixed with food, has been shown to delay carbohydrate absorption and lower serum concentrations of cholesterol. Many papers have been published to this effect.¹ Ms V A Beattie and others (23 April, p 1147) do not contribute usefully to them.

It is well recognised, for example, that guar, like many other treatments, must be started slowly. We prescribe half a satchet (2.5 g) with the evening meal for the first week of treatment and increase this slowly to a maximum of 10 or 15 g daily. If this is done over three to six weeks minimal bowel symptoms result and compliance is good. In contrast, a high proportion of patients begun on 15 g a day all at once will suffer from severe bowel symptoms.

For the best effect the gum needs to be mixed with the food.^{2,4} This is presumably because its effect is achieved through increasing the viscosity of the gastric chyme, preventing ingress of digestive enzymes and increasing the unstirred layer in the small intestine, both effects being likely to delay the absorption of nutriment. Indeed, drinking guar with a glass of water before the meal seems designed to frustrate its effective action by reducing mixing of guar with the food. The authors did not describe their mode of giving guar—a surprising omission.

If the optimal fasting blood glucose concentration is taken as 5 mmol/l the two groups on high fibre diets at 20 weeks were above this target by 1.8 and 1.5 mmol/l, while the low fibre group was above it by 2.8 mmol/l. This difference could be biologically important, and the lack of statistical significance might reflect the design of the trial and the small numbers of patients studied rather than a true lack of effect.

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Neisseria gonorrhoeae resistant to tetracycline

SIR,—Dr M A Waugh and others (26 March, p 898) discuss the isolation of tetracycline resistant *Neisseria gonorrhoeae* in the United Kingdom. We have also encountered infections caused by these organisms in patients attending the Praed Street Clinic at St Mary's Hospital. Since our original report of eight such infections¹ we have isolated a further six strains of tetracycline resistant gonococci, all from heterosexual patients.

Recently we have encountered a strain of *Neisseria meningitidis* that also has high resistance to tetracycline (minimum inhibitory concentration > 8 mg/l). This was isolated from the pharynx of a homosexual man during screening for pharyngeal gonorrhoea. Analysis showed the presence of a plasmid of approximately 25.2 megadalton. A similar isolate of *N. meningitidis* (received from Dr M S Sprott at the Newcastle Public Health Laboratory) showed a plasmid of identical size. In gonococci the determinant for high level resistance

to tetracycline is integrated into the conjugal plasmid to give a plasmid of 25.2 megadalton.²

Plasmids are unusual in meningococci, although low molecular weight cryptic plasmids have been described.^{3,4} To our knowledge there has been only one report of plasmid mediated antibiotic resistance in a clinical isolate.⁵ This strain carried both a penicillinase encoding plasmid of 4.5 megadalton and a conjugal plasmid of 24.5 megadalton. Transfer of penicillinase plasmids between gonococci and meningococci has been achieved in vitro, but acceptance of the conjugal plasmid by the meningococci has not been shown.^{6,7}

The presence of a 25.2 megadalton plasmid in these two isolates of meningococci, which both exhibit high level resistance to tetracycline, is alarming. This plasmid may have been acquired from gonococci. If this is true the plasmid will be capable of self transfer,² allowing further dissemination of tetracycline resistance. Both of these isolates were associated with asymptomatic carriage. This means that the presence of the 25.2 megadalton plasmid could easily remain undetected. While tetracycline resistance is not of concern in meningococcal disease, the presence in the pharynx of meningococci that carry self transmissible plasmids is worrying. Meningococci should be screened for tetracycline resistance, particularly in areas where the 25.2 megadalton plasmid has been found in gonococci.^{1,8}

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Meningococcal meningitis

SIR,—Dr G Venkat Raman (23 April, p 1141) stated that rifampicin is the prophylactic drug of choice for meningococcal meningitis. A 14 year old girl was admitted in the last week of March 1988 with what proved to be meningococcal meningitis. Rifampicin prophylaxis was given to all of her close contacts. It was shown subsequently by disc diffusion tests that this isolate was resistant to the sulphonamides and rifampicin, and these results were confirmed by the Meningococcal Reference Laboratory.

This strain was sensitive to the tetracyclines and ciprofloxacin, and ciprofloxacin was given prophylactically to the home contacts. We were told that this had been used successfully at a naval establishment (N Gaunt, personal communication) to protect adolescents. No adverse effects were reported, and so far no evidence of secondary meningococcal disease has occurred.