motor accident, illness, or divorce. I should welcome the views of other doctors as to whether one should yield to the request for sterilization in such young women.—I am, etc.,

BRUCE ETON

Buchanan Hospital,
St. Leonards on Sea,
Sussex

Starvation Therapy in Obesity

SIR,—Total fasting as a treatment for obesity has lately been severely judged and recently referred as "dangerous,"[1] because of serious side-effects such as episodes of sudden death.[2] Dr. J. Runcie and Dr. T. J. Thomson on the contrary, in their article on prolonged starvation (22 August 1970 p. 432), express a favourable opinion on this therapy. We agree with them. Fasting is a drastic measure indeed, but in our opinion it should keep its own place in the treatment of obesity. We have practised starvation on 19 selected obese patients for periods varying from 7 to 104 days, and have obtained satisfactory results with only minor complications. Glomerular filtration rate (G.F.R.) and daily diuresis were controlled in 13 of these subjects and a decrease of these parameters during fasting with a return to normal values after refeeding were observed in five of them, as has been reported by others.[3]

In particular, starvation of one patient weighing 128 kg was undertaken, in spite of a persistent microhaematuria consequent to a previously diagnosed glomerulonephrictis. He was confined to bed owing to a serious and progressive osteoarthritis of the left big toe with destruction of the phalanges, marked osteoporosis of the neighbouring bones, and oedema of the soft tissues. A striking improvement of the condition resulted, with a loss of 19 kg in 29 days. During the starvation period daily diuresis and G.F.R. decreased, but returned to basal values after refeeding and remained normal afterwards. Another favourable outcome of fasting that we have observed is the lowering of blood pressure in hypertensive patients. Blood pressure did not rise again after refeeding, provided the subjects did not regain the weight lost.

Fasting therapy, if applied to subjects on the basis of their willingness to co-operate and satisfactory conditions, presents some positive features. Once a patient has decided to fast, he usually does so correctly; we have seen many patients either disregard a diet or follow it in a wrong way but very few cheat with fasting. Weight loss is quick and, therefore, encouraging to the patient. Finally, hunger usually causes less discomfort than a diet and, at the refeeding, patients can easily tolerate an 800 calorie regimen.

In conclusion, while it is true that starvation is to be avoided in patients with heart failure, renal, or hepatic insufficiency, it does represent an effective tool for weight reduction and should not be regarded with hostility, since it appears to be no more hazardous than many other therapeutic procedures.—We are, etc.,

F. CAVAGNINI
M. PERACCHI
G. BIANCHI PORRO

Institute of Medical Pathology,
University of Milano,
Italy

1 *Lancet*, 1970, **1**, 1094.
2 Spencer, I. O. B., *Lancet*, 1968, **1**, 1288.
3 Edgren, B., and Wester, P. O., *Lancet*, 1970, **1**, 613.

Ileorectal Anastomosis for Ulcerative Colitis

SIR,—The leading article "Ileorectal Anastomosis for Ulcerative Colitis" (5 December, p. 572) made a plea for publication of results of ileorectal anastomosis in ulcerative colitis. This procedure has been used in our department during the past 20 years, and the results were summarized at the fourth World Congress of Gastroenterology, Copenhagen 1970. Subtotal colectomy was performed in 127 patients and in 48 an immediate or delayed ileorectal anastomosis was constructed. There was only one early operative death, and two anastomoses failed early because of rectal bleeding. Two patients had a late conversion to ileostomy because of activation of rectal disease, and five were late deaths, one of these from rectal cancer.

Thirty-eight patients still have their anastomosis on an average seven years after the operation. Twenty-six have four or less bowel movements a day. Sigmoidoscopy revealed no sign of activity in 27 cases. Of these 38 patients only three were considered as having an unsatisfactory result, but it was not so bad that the patients wanted the rectum removed.

Most of the ileorectal anastomoses were performed between three months and four years after removal of the colon (37 out of 48). In the interval the rectum was observed for regression of the inflammatory disease, which was occasionally aided by local treatment with steroids or simple irrigation. The anastomosis was performed if there was none or only slight activity on sigmoidoscopy and the rectum was soft and pliable. —We are, etc.,

M. SPRECHLER
H. BADEN

Surgical Department F (Gastroenterology),
Bispebjerg Hospital, Copenhagen,
Denmark

Antibacterial Agents in Renal Failure

SIR,—Referring to your leading article "Antibacterial Agents in Renal Failure" (20 March, p. 621), we recently managed a diabetic with renal failure who developed a severe polyneuropathy while being treated with intramuscular colistin which recovered rapidly on withdrawal of the drug.

The patient was a 26-year-old female, who had been an insulin-dependent diabetic for 14 years. She underwent a termination of a two-month pregnancy for hypertension and renal failure. Prior to operation, there was no clinical evidence of a neuropathy; her blood urea was 117 mg. Unfortunately, during evacuation of the uterus the cervix was split and there was a perforation into the pouch of Douglas. Five days postoperatively, she developed a high swinging temperature. Blood cultures were negative but *Pseudomonas pyocyanea* sensitive to colomycin was isolated from the urine. One daily intramuscular injection of 1 mega unit of colistin sulphomethate B.P. was started. The creatinine clearance at this time was 11 ml/minute.

On the fourth day of therapy, she developed a flaccid paralysis of the lower limbs; all reflexes and all modalities of sensation were absent. The power and sensation in the upper limbs were normal but the reflexes were depressed. The diabetes at this stage was well controlled. The blood colistin level on the fifth day of treatment was markedly elevated at 160 μg/ml. Colistin injections were, therefore, stopped. A day later the blood colistin level was 40 μg/ml, and the day after this the level had dropped to 20 μg/ml. All evidence of infection had cleared by this time. Four days after the cessation of colistin therapy the legs were still areflexic, but there was some slight movement. At seven days there was improvement in all forms of sensation except for vibration. Within three weeks she could walk with help. Three months later she was able to walk quite well by herself, and sensation was normal. The ankle jerks were, however, impaired.

Colistin has been reported as causing apnoea, perioral paraesthesia, ataxia, muscular hypotonia and halucinations.[1-3] These effects improved after the drug had been stopped. The respiratory paralysis causing apnoea was thought to be a curare-like effect of the drug and reversible by neostigmine.

Although the patient was a diabetic, there is strong incriminating evidence that the drug was responsible for the neuropathy, in that there was no clinical evidence of a neuropathy before this episode, its development coincided with high blood levels of colistin, and recovery took place on withdrawal of the drug. It would seem that even with small doses of the drug in renal failure and in the presence of other debilitating illnesses we should be wary of the possible neurotoxic effects of colistin.—We are, etc.,

J. F. BRIDGMAN
S. M. ROSEN

St. James's Hospital,
Leeds

1 Perkins, R. L., *Journal of the American Medical Association*, 1964, **190**, 421.
2 Fekety, F. R., Norman, P. S., and Cluff, L. E., *Annals of Internal Medicine*, 1962, **57**, 214.
3 Wolinsky, F., and Hines, J. D., *New England Journal of Medicine*, 1962, **266**, 759.

Bacteriuria Again

SIR,—In the leading article "Bacteriuria Again" (13 February, p. 361) gentamicin is described as being notoriously toxic to the eighth nerve. While this toxic effect is undisputed, the notoriety may be due to overdosage or inadequate control of treatment.

Ototoxicity has been shown to occur in animals treated with gentamicin[1 2] and is greater than that produced by streptomycin or kanamycin[3] on a dose/weight ratio.

Few controlled studies have been carried out in man defining the incidence of ototoxicity, but most workers have related this to reduced renal function.[4-7] However Wright[8] has shown in guinea pigs that the cochlear toxicity is independent of the level of blood urea. The significant ototoxicity reported by Arcieri *et al.*[7] following original work by Jackson[9] in two studies involving 1,327 patients was 2·3%. The second study showed a fall in toxicity that was not statistically significant but was associated with an increase in dosage. More recently Meyers[10] reports that 10 of 40 patients treated with gentamicin developed ototoxicity but eight of these may have had serum levels of over 10 μg/ml and a similar number had a raised blood urea. Arcieri *et al.*[7] found that only 13 of the 31 recorded cases of ototoxicity had had serum levels carried out and eight of these had levels of 10 μg/ml or more. Bulger *et al.*[4] in reporting one case of severe vestibular dysfunction found only one of the six