constituted an error of judgment. If it is untrue, then the authors of the book should give high priority to putting the record straight.

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Large-bowel polyps and colonoscopy

SIR,—Your leading article "large bowel polyps and colonoscopy" (16 June, p 1587) emphasises the importance of screening in early detection and treatment of colonic cancer, yet comes to an odd conclusion.

Surely, before introducing a sigmoidofibroscope into outpatient clinics it would be simpler to ensure that every patient who attends outpatients with bowel symptoms has a guaiac test for occult blood in the stool obtained from a rectal examination. Then those who are positive can be investigated further—barium enema and endoscopy on a prepared bowel. For it is my experience that patients' symptoms are often ignored or misdiagnosed as piles until the chance for curative surgery is missed. Selective screening in the form of a guaiac test would help us to look at the right population with possible colonic cancer and, perhaps, to see the tumour earlier. A sigmoidofibroscope, however, for unprepared bowels, in an outpatient clinic, would be little more than toys for the boys.

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Diverticular disease in urban Kenyans

SIR,—Dr John F Calder (2 June, p 1465) has reported 15 urban Kenyan patients with diverticular disease in the Kenyatta National Hospital, Nairobi. This report should be compared with other reports on diverticular disease in urban Africans, such as that from Ghana Medical School, in which 14 cases of diverticular disease were observed in 360 barium enema examinations during 1974-6. Sixty per cent of these patients derived from Ghanians of higher social class.¹ Also from Johannesburg the first 16 Bantu patients suffering from diverticular disease were reported in 1977 by the physicians and radiologists in the 2000-bed University Baragwanath Hospital during 14 months of 1974-5.² Experience over many years led their Johannesburg group to entitle their article "Emergence of diverticular disease in the urban South African Black."

Development in Kenya has brought increasing affluence to urban Kenyans, with Westernisation of the diets, shown as increased consumption of sugar, while low-fibre sifted maize flour has increasingly replaced high-fibre maize meal, as mentioned by Dr Calder. Britain has been the first country to report dietary fibre intakes.³ In other countries these are not known but trends are certainly not only towards far lower intakes of cereals as Westernisation of diets occurs but in particular towards a reduction in cereal fibre. This is of importance as it contributes much more than does the fibre of fruits and vegetables to bulk and softness of stools.⁴

The World Health Organisation has reported in 85 countries dietary changes that accompany increasing affluence and Westernisation.⁵ Among the 750 million poor in-

habitants of developing countries 75% of the daily energy is derived from starch, whereas among the 500 million affluent inhabitants of the developed countries only 25% of daily energy is derived from starch. In the latter fibre-free fat has increased from under 10% to 40% and fibre-free sugar from under 5% to 20%. At the same time fibre-rich cereal meals, such as wholemeal bread and maize meal, have been replaced often by fibre-depleted refined white bread and refined, sifted, maize flour. For instance, wheat wholemeal bread contains dietary fibre 8.5 g/100 g, white bread 2.7 g/100 g.⁶ No data have been published yet concerning the dietary fibre content of maize meal and maize flour, but a comparable decrease of dietary fibre is anticipated. If in Western communities starch consumption decreases threefold and its associated fibre also decreases almost threefold, then wherever Western-type diets are consumed cereal dietary fibre intake certainly decreases considerably.

Workers in the urban areas of Africa have for many years been reporting increased incidence of appendicitis. Only recently, however, have reports of diverticular disease begun to appear. The former is among the first and the latter among the last of characteristically Western diseases to emerge following the impact of Western culture. Since Dr Calder states that these Africans are still on relatively high-fibre diets one cannot but wonder whether these figures are representative of urban Africans as a whole. A frequency of 7% of diverticular disease in an African group of average age 41 is comparable to Western figures; yet British vegetarians with their marginally higher fibre intake, compared with Africans, have a significantly lower frequency of diverticular disease than the community as a whole.⁷

Quantitative data are needed on all aspects of this problem, and also comprehensive dietary surveys.

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¹ Archampong, E Q, Christian, F, and Badoe, E A, *Annals of the Royal College of Surgeons of England*, 1978, **60**, 464.

² Segal, I, Solomon, A, and Hunt, J A, *Gastroenterology*, 1977, **72**, 215.

³ Southgate, D A T, Bingham, S, and Robertson, J, *Nature*, 1978, **274**, 51.

⁴ Cummings, J H, *Journal of the Royal Society of Medicine*, 1978, **71**, 81.

⁵ World Health Organisation, *Energy and Protein Requirements*, Technical Report Series, No 522, p 20. Geneva, WHO, 1973.

⁶ Paul, A A, and Southgate, D A T (editors), *McCance and Widdowson's The Composition of Foods*. London, HMSO, 1978.

⁷ Gear, J S S, *Journal of Plant Foods*, 1978, **3**, 57.

Maturity-onset diabetes

SIR,—I read with interest the article by Drs V Anne Ropner and J Anderson (7 April, p 938), as I am concerned at the possible diabetogenic effect of propranolol. One of our patients, who developed maturity onset diabetes, was taking 960 mg of propranolol daily for angina, and, on stopping this drug, his blood sugar returned to normal and he required no further hypoglycaemic agents.

The *BMJ* (20 January, p 159) had a report from Birmingham detailing the, what seems to me, significant rise in blood sugar concentration of some patients on 160 mg of

propranolol daily, and, although the makers of this drug deny that it can cause any significant hyperglycaemia, I shall be cautious in prescribing propranolol in future for patients with a history of diabetes or overt diabetes.

I thank Dr P de V Meiring, principal physician, for his encouragement and the Director of Hospital Services, Natal, for permission to report this case.

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Absorption of enteric-coated prednisolone

SIR,—We were interested to read the observations concerning the absorption of enteric-coated prednisolone tablets (9 June, p 1534). We have been unhappy about the reliability of their absorption for a considerable time and would like to refer to a case which nearly resulted in the loss of a renal transplant.

A 58-year-old man had a cadaveric renal transplant on 13 August 1975. He was started on enteric-coated prednisolone tablets and azathioprine. A few days later the patient showed evidence of a rejection and was given conventional treatment with pulse doses of methylprednisolone. However, 24 hours later he developed classical signs of an acute intestinal obstruction. A plain x-ray film of the abdomen showed aggregation of the enteric-coated tablets which had not been absorbed and were causing the obstruction. Intravenous fluid therapy and nasogastric suction were instituted and laparotomy was avoided. Parenteral prednisolone and azathioprine were commenced for the maintenance of immunosuppression. Approximately three days later the patient had relief of the intestinal obstruction and was able to take fluids orally. His medication was then changed to normal prednisolone and azathioprine.

This case illustrates that, although in some patients enteric-coated tablets are absorbed, in others they may be passed unabsorbed into the small intestine. In conditions in which the plasma levels of prednisolone need to be stable and predictable their use is unsatisfactory. We would agree that it is doubtful whether the coating of the prednisolone tablet prevents peptic ulceration and suggest that this effect may be due to non-absorption of the medication, with potentially dangerous effects.

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SIR,—In the paper by Dr R G Henderson and others (9 June, p 1534) the authors' use of enteric-coated prednisolone tablets to reduce peptic ulceration is referred to an observation reported 20 years ago.¹ I would like to make two comments.

Firstly, the observation applied only to a particular enteric coating that dissolved soon enough for the hormone to be absorbed before it was degraded. The BP enteric coating did not allow this, as others rediscovered to their cost years later.

My second comment is that I gave up such use of enteric-coated prednisolone tablets in favour of prednisolone phosphate tablets, taken dissolved in water, in 1963 and, following studies of the speed and completeness of their absorption, suggested their use for all children