cation of prostaglandin E_2 in various vehicles. Our own work involving over 500 patients who had a single insertion of 4 mg or 5 mg of PGE_2 tablets (Prostin, Upjohn Limited) vaginally showed that about 70% of patients went into labour without any other intervention.¹ These results, like those reported recently by Mr J H Shepherd and others (14 July, p 108), are not ideal and can be improved on by seeking the optimum dose regimen, as well as the best vehicle for release of PGE_2 from the many available.

Until these points are established it is too soon to claim that we have the best alternative to intravenous oxytocin.

M G ELDER

Institute of Obstetrics and
Gynaecology,
London W12 0HS

¹ Gordon-Wright, A P, and Elder, M G, *British Journal of Obstetrics and Gynaecology*, 1979, **86**, 32.

Hemiparesis after single minibolus of labetalol for hypertensive encephalopathy

SIR,—Your leading article of 28 July (p 228) suggested that a graded 50-mg minibolus or controlled infusion of labetalol may overcome some of the problems of rapid reduction of arterial pressure in malignant hypertension. We would like to report on a patient who developed left hemiparesis after a single minibolus of labetalol.

A 48-year-old West Indian negro housewife presented with a six-month history of intermittent headache, nausea, and blurring of vision, and described two episodes within the previous three hours suggesting grand mal attacks. A grand mal attack occurred 10 minutes after her arrival in the casualty department. She had a 14-year history of hypertension but was taking no regular medication. She did not smoke. There was no family or past history of epilepsy. She had a sinus tachycardia of 115 beats/min, blood pressure of 250/150 mm Hg, and clinical and electrocardiographic features of left ventricular hypertrophy. X-ray showed cardiomegaly. Fundoscopy demonstrated grade II hypertensive retinopathy. Ward testing revealed heavy proteinuria.

After her grand mal attack she received a minibolus of 35 mg of labetalol over two to three minutes. Two minutes later her blood pressure was 160/95 mm Hg. It remained at this level for three minutes and then rose over the next five minutes to 180/110 mm Hg, with a pulse rate of 90 beats/min, at which level it was maintained with oral therapy of atenolol 100 mg a day and hydralazine 25 mg twice daily. The next day she was drowsy and slightly confused and 36 hours after admission developed left hemiparesis with sensory inattention. Paresis of the left arm has persisted. Her renal function was normal and the proteinuria diminished. Computerised axial tomography revealed a low-density area in the right parietal region suggesting an ischaemic lesion.

Such a marked fall in arterial pressure after the administration of a small dose of labetalol has not been documented before. Cerebral complications have been noted previously,^{1,2} after reductions in arterial pressure of this magnitude. The data sheet for labetalol recommends a 50-mg bolus given over more than one minute, followed by repeated doses at five-minute intervals to a maximum of 200 mg. Alternatively an infusion at 2 mg/min may be given. A fall in blood pressure from 188/143 mm Hg to 90/64 mm Hg after 100 mg of labetalol was noted by Brown *et al*,³ and the same authors recommend graded intravenous infusions with continuous monitoring

of arterial pressure in hypertensive encephalopathy.⁴

This case supports slow infusion as the treatment of choice and indicates that there is no longer any place for the 50-mg minibolus in the treatment of hypertensive encephalopathy.

RICHARD SOLOMONS

St Mary's Hospital (Harrow Road),
London W9 3RL

¹ Love, D H, *et al*, *British Medical Journal*, 1979, **2**, 245.

² Ledingham, J G G, and Rajagopalan, B, *Quarterly Journal of Medicine*, 1979, **48**, New Series, 25.

³ Rosei, E A, *et al*, *Clinical Science and Molecular Medicine*, 1976, **51**, suppl p 49.

⁴ Brown, J J, *et al*, *Lancet*, 1977, **1**, 1147.

Mefenamic acid poisoning and epilepsy

SIR,—A 19-year-old woman took a well-substantiated overdose of 12.5 g mefenamic acid (50 250 mg Ponstan tablets) three to four hours prior to admission. (These tablets had been prescribed for headache by her GP on the morning of the day of admission and the bottle was later found by relatives to be empty—no other drugs were prescribed or found.) She was found approximately three hours after ingestion in status epilepticus, when it was not appreciated that self-poisoning had occurred. On arrival at the casualty department there were features of a generalised seizure, notably tonic-clonic movements of all four limbs, bilateral extensor plantar responses, lack of response to painful stimuli, and urinary incontinence. Paraldehyde 10 ml was administered intramuscularly by the casualty officer and the convulsive features rapidly subsided. Following transfer to the medical unit there was drowsiness but no neurological deficit. A drug screen was negative for salicylates and barbiturates. The laboratory was certain that an abnormal substance was present in the plasma but could not identify its nature. There were no further seizures and she subsequently admitted the above overdose. This patient had been previously well and there was no family history of epilepsy. The psychiatric diagnosis was personality disorder. An ECG three weeks later was normal.

There is little documented evidence that mefenamic acid poisoning may cause epileptic seizures. In one text the possibility of convulsions following massive overdosage is suggested,¹ but sources are not quoted. Current reports by Committee on Safety of Medicines mention only single episodes of generalised spasm and muscle twitching.

We would submit that there is good evidence from this case of a direct association between mefenamic acid poisoning and a generalised seizure. The possibility that such poisoning may be responsible for a previously fit patient presenting in status epilepticus should be borne in mind.

R J YOUNG

Neurological Unit and University
Department of Medical Neurology,
Northern General Hospital,
Edinburgh EH5 2DQ

¹ Cooper, P, *Poisoning by Drugs and Chemicals*, 3rd edn. London, Alchemist Publications, 1974.

What is to be done with the XYY fetus?

SIR,—With reference to Dr Simon Wilkinson's letter (14 July, p 131) concerning the control group used in our XYY study¹ we should like to bring the following points to your notice.

The induction rate in the control group was

45.6%, in the XYYs 50%, and in the hospital population from which both groups were drawn 42.6%; none of these figures differ significantly from each other. Hence there are no grounds for claiming that the controls were privileged in this respect. In line with changing obstetric policy, the present induction rate at these hospitals is around 28%.

No claim was made that the controls were representative of the general population. In our paper it was clearly stated that the hospital population from which both our controls and the XYYs were drawn differed significantly from the population of Scotland as a whole in that it contained a higher proportion of social classes I and II. However, the controls and cases matched closely in this respect and therefore the difference in mean IQ (98.4, SD ± 11.0, compared with 115.7, SD ± 16) indicated a small but significant reduction in the XYY child. Furthermore, comparisons in the three twin pairs were in each case adverse to the XYY twin, the differences being 23, 19, and 13 points. These results support the findings of the only other study of young XYY individuals ascertained outside institutions, where Army selection tests gave significantly lower scores for the XYY men.²

We wish to point out that we do not consider the small reduction of IQ to be grounds for selective abortion. As our longitudinal study has covered only the first 12 years of any child's life it would be premature to make any pronouncement on the rate of social deviance.

A full report of the obstetric and environmental background to the whole study will be published in the near future and will describe in more detail the numerous variables relevant to any analysis of human development.

SHIRLEY G RATCLIFFE
D G AXWORTHY

Medical Research Council Clinical
and Population Cytogenetics Unit,
Western General Hospital,
Edinburgh EH4 2XU

¹ Ratcliffe, S G, Axworthy, D, and Ginsburg, A, in *Birth Defects: Original Article Series*, vol 15, No 1, ed A Robinson, H A Lubs, and D Bergsma, p 243. New York, Alan R Liss Inc, 1979.

² Witkin, H A, *et al*, *Science*, 1976, **191**, 547.

Diverticular disease in urban Kenyans

SIR,—The Reverend H C Trowell and Mr Denis Burkitt in their letter (30 June, p 1795) on Dr John F Calder's article (2 June, p 1465) cite our paper on diverticular disease¹ as further evidence of the influence of diet on the emergence of diverticular disease in the urban African. They quote correctly that "60% of these Ghanaian patients belonged to the higher social classes"; the point of the article is lost, however, when the sentence in question is not completed—that is, "but all had lived on traditional African high residual food." The impression is thereby created that our patients may have been on Western-type diets.

It is particularly important to appreciate that in developing countries, and particularly among Africans, urbanisation is seldom accompanied by significant alterations in dietary habits. This principle has not been accorded its due place in the global diet-disease discourse.

Our data may be interpreted as suggesting that adoption of "Western habits" (60% had been in high public positions of trust)