offending drug. If the diagnosis of acute SLE is correct, I see no evidence of nomifensine being implicated in the patient's illness.

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1. Sokol RJ, et al. Fatal immune haemolysis associated with nomifensine. *Br Med J* 1985; **291:** 311–12.
2. Hamm H, et al. Alveolitis associated with nomifensine. *Lancet* 1985; i: 1328–29.
3. Garcia-Mortes O, et al. Lupus-like syndrome during treatment with nomifensine. *Arthr Rheum* 1983; **26** (no 7).

HAZARD OF POTASSIUM CHLORIDE SOLUTION

SIR,—Professor Rendell-Baker and Professor Meyer write (Aug 10, p 329) about the concern of the secretary of the Medical Defence Union—which they share—about deaths due to concentrated potassium chloride being injected intravenously instead of the harmless sodium chloride. My interest also was aroused during a discussion with the secretary when writing a book (*Mishap or Malpractice?*) to celebrate the centenary of the MDU this year. A visit to our pharmacy confirmed that the ampoules are identical in size, shape, lettering, and colour of solution; it surprised me that no steps had been taken by manufacturers or others to solve the problem by colour coding or changing the shape of the ampoules. Mr S. R. Potter (pharmacy, Queen Elizabeth Hospital, Birmingham) and his colleagues took the matter up and now an ampoule is available (Antigen International Ltd) that cannot possibly be mistaken, for it has a fixed black plastic cap over its neck. It is used throughout the West Midlands region and has been approved by anaesthetists, nursing authorities, and others, and the old ampoules have been withdrawn by the pharmacists. The extra cost is trivial. The doctor should still read the label but it provides an added safeguard—like a seat-belt even though the driver knows the highway code by heart.

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ETHYLENE GLYCOL POISONING

SIR,—Your editorial (Aug 3, p 254) on the clinical and biochemical findings in and treatment of ethylene glycol poisoning stated that most such poisonings are of the suicidal type. Probably, most cases of ethylene glycol poisoning are accidents not suicide attempts. This point was made in 1975,[1] and I doubt if the pattern today is different. The chemical is sweet and pleasant tasting and can be readily ingested by children and others who mistake it for a beverage. To prevent both ingestion (accidental and intentional) and wine adulteration, an unpleasant tasting substance should be added to all marketed ethylene glycol.

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1. Winer CL. Ethylene glycol poisoning. *N Engl J Med* 1975; **292:** 928–29.

IN-VITRO FERTILISATION FOR ENDOMETRIOSIS

SIR,—Dr Wardle and colleagues (Aug 3, p 236) report a reduced fertilisation rate of mature oocytes aspirated from the ovarian follicles of women whose infertility was attributed to pelvic endometriosis. There were too few pregnancies to permit a useful statistical comment on the potential of such cases to conceive but they suggest that the chance of pregnancy would be the same as that for other couples if embryos were achieved and transferred.

The pregnancy rate has recently been analysed for patients managed in the PIVET Laboratory's in-vitro fertilisation (IVF) programme in Perth, Western Australia. Since 1981 more than 60 infants have now been delivered in the PIVET programme[1] and in May, 1985, we reported data on 140 pregnancies arising from IVF.[2] The pregnancy rate for 592 couples, who had a mean of 1·66 treatment cycles, was 23·7%. The pregnancy rate of endometriosis patients (9·1%) was significantly reduced ($p<0·01$) from the mean pregnancy rate for all categories of infertility and for those with tubal disease (23·3%). In part this was attributable to a reduction in oocyte recovery and fertilisation rates but this did not provide the entire explanation since other groups with similarly reduced fertilisation rates (eg, where there was oligospermia and asthenospermia) maintained a high pregnancy rate, comparable with the overall mean.

Women without a recognised cause for their infertility, including those couples with unexplained infertility, and women with failed artificial insemination treatment with donor sperm, had significantly higher pregnancy rates (47·6%, $p<0·001$) when compared with those with tubal disease.

Oocyte recovery in women with pelvic endometriosis can be very difficult because the ovaries are often part concealed and difficult to mobilise from behind a fixed retroverted uterus. Endometriotic cysts within the ovary are often inadvertently aspirated during attempted oocyte recovery and the aspiration needle and catheters may become clogged. These factors apart, there seems to be another reason, related to the failure of successfully developed embryos to implant—but, as with other aspects of endometriosis, the reason remains obscure.

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1. Yovich JL, Puzey A, De Atta R, Roberts R, Reid S, Grauaug A. In-vitro fertilisation pregnancy with early progestagen support. *Lancet* 1982; ii: 378–79
2. Yovich JL, Stranger JD, Yovich JM. In-vitro fertilization in Western Australia: a viable service program. *Med J Aust* 1984; **140:** 645–49.
3. Yovich JL, Yovich JM, Tuvik AI, Junk S, Bootsma B, Matson PL. In-vitro fertilization applied for tubal and non-tubal causes of infertility. *Asia-Oceania J Obstet Gyaecol* (in press).

RISK FACTORS FOR HEPATOCELLULAR CARCINOMA

SIR,—Zaman et al[1] conclude that cirrhosis is the major risk factor for the development of hepatocellular carcinoma (HCC) and that the hepatitis B virus (HBV) infection and HCC are linked solely because HBV is a common cause of cirrhosis.

The two major high-incidence areas for HCC are the Western Pacific and Subsaharan Africa.[2] Of Zaman's 613 patients with cirrhosis only 4 (0·7%) came from the Far East and 21 (3·4%) came from Africa (of which an unmentioned proportion was from Egypt). The conclusions drawn by Zaman et al can at best be applied to a small proportion of the world's HCC and cannot be extrapolated to the two high-incidence areas.

Zaman et al point out that Obata et al[5] did not consider the sex of be subclinical.[3] This is borne out in a study of 211 HCC patients in Hong Kong.[4] Zaman et al studied patients who presented with cirrhosis. In our view patients with HCC and symptomless HBV-associated cirrhosis would probably die of HCC without ever presenting with symptoms of cirrhosis whilst patients presenting with cirrhosis may die of complications of cirrhosis before HCC develops. A follow-up of patients who present with HBV-associated cirrhosis will thus miss many patients with HCC.

Only 42 patients had developed HCC, a small number and a very small proportion of the study population (6·9%). The conclusions drawn by subdividing these 42 patients into different categories are thus subjected to type II statistical error.

Zaman et al point out that Obata et al[5] did not consider the sex of their patients in concluding that HCC developed significantly more in patients who were seropositive for the hepatitis B surface antigen (HBsAg). In a necropsy study from Hong Kong, of 545 cases of cirrhosis and HCC,[6] HBsAg was identified in the liver in 83% of male cases of HCC with cirrhosis and in 87·5% of the female cases. As many as 183 of 353 (52%) cases of HBsAg-positive cirrhosis were complicated by HCC (55% for males, 37% for females) while only 36 of 143 (25%) cases of HbsAg-negative cirrhosis had HCC (27% for males, 14% for females). The incidence of HCC was therefore significantly higher in HBsAg-positive than HBsAg-negative