interview after an emergency referral she presented many biological symptoms and signs of depression. For three weeks, attempts were made to treat her with antidepressants as an outpatient, but she was admitted to inpatient psychiatric care after becoming acutely agitated and violent at home. Failure to respond to a combination of tetracyclic antidepressants and phenothiazines lead to the application of electric convulsion treatment four times. She made an excellent response, and was fit for discharge to outpatient care after six weeks as an inpatient. The social factors included profound language difficulties, leading to marked social isolation, aggravated by the lack of extended family support together with profound disappointment at the delivery of a fifth daughter.

There appeared to be a number of common features in these cases. Social isolation was marked and much aggravated by severe limitation in the use of the English language. Absence of an extended family network was an additional feature in two of the cases.

The sex of the child may have been of aetiological significance; certainly it is culturally of special interest. Jacob John and others have found that among native Indian women, those women who were particular about the sex were prone to suffer from depression during the postpartum period.[1] In the Camberwell Study Professor Kendell found that immigrant mothers (mainly Irish and West Indian women) had a higher rate of psychiatric consultation than women born in England.[2]

I do not think any conclusions can be drawn from the above case studies, but one needs to gather a large consecutive series of cases of both Asian immigrant women and native subjects with puerperal depression to establish: (*a*) whether immigrant Asians are any more at risk of puerperal psychosis than the indigenous population (or other immigrant groups); and, (*b*) whether there are more exogenous factors (linguistic differences, social isolation, and sex of the child) in Asian women than in other women with puerperal depression.

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[1] John J, Seethalakshmi, Charles SX, Verghese A. *Indian Journal of Psychiatry* 1977;**19**:40-3.
[2] Kendell R. *Psychol Med* 1976;**6**:297-302.

SIR,—We have read with interest the leading article by Professor Sydney Brandon (27 February, p 613) on this distressing condition, written from the viewpoint of the psychiatrist. As obstetricians we are grateful to our psychiatric colleagues for their help in caring for these unfortunate patients and their families.

We note, however, that Professor Brandon makes no reference to the possible use of progesterone in the treatment of some of these patients. That progesterone may indeed have a useful role in the prevention of postpartum depression is shown by the following case record of a patient recently under our care.

A 32-year-old housewife was booked for confinement at 14 weeks' gestation in her third pregnancy. In her first pregnancy in 1978 in another hospital labour was induced for postmaturity, and an emergency caesarean section was performed later for fetal distress. Thirty-six hours after the operation she developed signs of pulmonary embolism and was anticoagulated with heparin. She subsequently developed a wound haematoma and an episode of haematuria. At this time she became anxious, expressed paranoid thoughts, and began wandering in the early hours. She was sedated with chlorpromazine and seen by a psychiatrist. She improved enough to be allowed home after seven days, but unfortunately relapsed and required admission to a psychiatric hospital for two weeks' treatment of her puerperal psychosis; thereafter she made a complete recovery.

In her second pregnancy in 1980 in this hospital an elective caesarean section was performed at term under epidural analgesia. The puerperium was complicated by a wound infection and a widespread bullous rash. A dermatologist considered this to be an allergic reaction to amylobarbitone, which she had received as night sedation, and the rash cleared up on stopping this drug and giving chlorpheniramine. Ten days after the operation she became hypomanic and was once again seen by a psychiatrist, who felt that she was developing a puerperal psychosis. She became so restless and agitated that she was transferred to a local psychiatric hospital. Again she made a good recovery after two weeks' inpatient treatment.

The antenatal period of her third pregnancy was uncomplicated, but in view of her past history a psychiatric interview was arranged during that time, with the recommendation that she should be given haloperidol at the first signs of puerperal psychosis developing. The work of Dr Katharina Dalton had come to the patient's attention, meanwhile, and she asked if she might be given progesterone in the puerperium in an attempt to prevent a further puerperal psychosis. An elective caesarean section with sterilisation was performed on 26 January 1982, and on the advice of Dr Dalton a progesterone (Gestone) injection 100 mg intramuscularly was given at delivery with a further 100 mg intramuscularly daily for the next six days, followed by progesterone (Cyclogest) suppositories 200 mg twice daily until the first menstrual period. This time we were pleased to note that she made a good recovery from the operation, and there were no psychiatric problems during her eight days in hospital.

One week after returning home, however, she began to exhibit signs of hypomania, so on psychiatric advice treatment with haloperidol was started while continuing the progesterone suppositories. She gradually improved, and was pleased with the fact that this episode was much less severe than her previous ones and did not require hospital admission. At the same time she retained insight into her behaviour and was able to continue breast-feeding and caring for her baby.

We believe there may well be other clinicians with much greater experience of the use of progesterone in the puerperium than we have and would be interested to hear of their experiences in dealing with similar problems.

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Hyperosmotic non-ketotic diabetic syndrome precipitated by treatment with diuretics

SIR,—Diuretics make you thirsty. Too much sugary drink may lead to hyperosmotic non-ketotic coma.[1] It would be interesting to know if the patients of Drs Vivian Fonseca and David Phear (2 January, p 36) drank much Lucozade before their admission.

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[1] Hughes-Davies TH. *Lancet* 1966;i:822.

***We sent this letter to the authors, who reply below.—ED, *BMJ*.

SIR,—We were interested in Dr Hughes-Davies's comment that sugary drinks may cause the hyperosmolar syndrome in diabetes. None of the 11 patients in our series had drunk Lucozade. We have, however, also seen several patients who have aggravated their symptoms at the onset of diabetes, or even developed the hyperosmolar syndrome, by drinking Lucozade containing 22·4% glucose.

It is interesting to compare the 1966 series of hyperosmolar coma, on which Dr Hughes-Davies was commenting, with our series. In 1966 Halmos, Nelson, and Lowry[1] found major precipitating disease in three out of eight patients, and previous medication was not responsible. In 1982, the hyperosmolar state was precipitated by diuretics in eight out of the 11 patients and by prednisolone in another patient. The hyperosmolar non-ketotic diabetic syndrome is now an iatrogenic disease.

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[1] Halmos PB, Nelson JK, Lowry RC. *Lancet* 1966;i:675-9.

"Home brew" compared with commercial preparation for enteral feeding

SIR,—You published several letters in response to the article by Mr M R B Keighley and others (16 January, p 163), and it is significant that these various responses contained a similar tone of defensiveness. I submit that there is a classical case of defending the indefensible. Two simple facts are relevant. Fact 1: commercial preparations are either autoclaved, treated by ultra-heat treatment and aseptic filling, or undergo Tyndallisation. Minimal handling is likely to be accomplished in a practical sense and on a national scale. Fact 2: "Home brews" attract maximum handling and although these procedures may be accomplished in some specific units with well staffed and equipped dietetic departments, there is little likelihood, on a practical and national scale, of "home brews" matching commercial preparations in terms of lack of contamination on exhibition to patients.

There is now published evidence[1 2] showing pathological sequelae following contamination of tube feeds and strong in-vitro evidence that commercial preparations are substantially less likely to present contaminated product than "home brews" (paper presented by M D Bastow, S P Allison, and P Greaves at the third European Congress on Parenteral and Enteral Nutrition, 1981).

It is understandable that a minority of the dietetic profession retains a subjective desire to prolong the active life of "home brews." It is inevitable that this singularly British anachronism will probably, sooner rather than later, become subtotally superseded. There will always be a relatively low incidence of need for "specials."

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[1] Casewell MW, Philips I. *J Clin Path* 1978;**31**:845-9.
[2] Casewell MW, Cooper JE, Webster M. *Br Med J* 1981;**282**:973.

SIR,—I have followed with interest the correspondence (6 March, p 741) generated by the article of Mr M R B Keighley and his colleagues (16 January, p 163). While I agree that commercial preparations are easier to