patients continue to exhibit an unfavourable cervix resistant to induction post term and most of these benefit from local prostaglandins.

With careful adjustment, the highly sophisticated cardiotocographic monitors available today allow very satisfactory recordings to be made. Perhaps in our critical age induction with a PGE₂ pessary should constitute the only permissible form of "stress test." After all, if a potentially compromised fetus shows distress at this early stage, then expedited delivery by the most appropriate route would obviously be in the best interests of the fetus.

In the future, different doses of PGE₂ pessaries will be available, depending on the requirements of the individual patient. It may be that 1 mg, 3 mg, and 5 mg pessaries will prove necessary, and perhaps when the drug companies concerned are more interested in the commercial aspect of their production this will be possible. Each pessary costs approximately £6, but surely this, or \$14, is not a large sum when compared with the potential benefits gained, especially if caesarean section and its attendant price are avoided, as our reported series showed possible. Given the choice, patients prefer this method of "non-invasive" induction, and if they had to most would be willing to pay for it. At present, in many centres in America, the caesarean section rate is much higher than that in Great Britain. There are many reasons for this, but quite definitely, if the United States Food and Drug Administration were to recognise the tremendous potential and advantages of locally administered prostaglandins, then prolonged inductions ending in failure and inevitable caesarean section could be avoided in the USA too. This would be especially important in a country where the maxim "Once a section, always a section" is very widely applied.

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¹ Shepherd, J H, Pearce, J M F, and Sims, C D, *British Medical Journal*, 1979, 2, 108.

² Campbell, S, *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1970, 77, 1057.

Postpartum haemolytic uraemic syndrome

SIR,—It may be worth while thinking of the haemolytic uraemic syndrome (21 July, p 179) on the assumption that it derives from loss of equilibrium within the interlinked system¹ of proteolytic control.

Each condition predisposing to or precipitating the syndrome (HUS) has within it an element of proteolytic stress: postpartum HUS may be related to the intense involutional proteolysis of the puerperium²; HUS in young women on oestrogen-containing oral contraceptives may be related to protease-dependent acute-phase effects of hyperoestrogenism³; HUS after an endotoxic condition⁴ may be related to activation of coagulation and complement systems plus release of tissue proteases from damaged cells; the HUS of severe preclampsia may have a similar basis.⁵ The allied aspects of the Schwartzman reaction, mentioned by Dr Gwyn Williams, are also likely to have their basis in the upset of proteolytic equilibrium in endotoxic shock.⁶

If the loss of control is overwhelming several proteolytic cascades may be released from control and become hyperactive, including the kallikrein, renin, complement, and coagulation cascades. The exact pattern will vary in different patients but on this basis it may not be so surprising if the HUS is heralded first by permeability (kallikrein) or hypertensive (renin) upsets rather than by complement or coagulation changes.

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¹ Heimbürger, N, Haupt, H, and Schwick, H G, in *Proceedings of the International Research Conference on Proteinase Inhibitors*. Berlin, Walter de Gruyter, 1971.

² Schoolwerth, A C, et al, *Archives of Internal Medicine*, 1976, 136, 1978.

³ Laurell, C B, and Skanse, B, *Journal of Clinical Endocrinology and Metabolism*, 1963, 23, 214.

⁴ Baker, N M, et al, *British Medical Journal*, 1974, 2, 84.

⁵ Stark, J M, *Lancet*, 1978, 1, 417.

⁶ Stark, J M, et al, *Journal of Clinical Pathology*, 1978, 31, 1177.

Neonatal care

SIR,—The impressive work of Dr Malcolm L Chiswick and others for the babies of Lancashire (28 July, p 247) is difficult to assess on the figures given. Superficially the first-week mortality of about 7 per 1000 at St Mary's Hospital, Manchester, in 1978 does not differ much from the 1978 perinatal figure of 15.4 for England,¹ over half of which is likely to be due to stillbirth. However, the overall neonatal rate for individual hospitals is bedevilled by fluctuations in the distribution of babies by weight and by differences in reporting habits for very small babies. Babies born dead before 28 weeks are not reported; those alive should be, no matter how small. Thus in one hospital in 1975 of 18 deaths in the first week six were in infants weighing 450 to 470 g, and their omission would have lowered the mortality from 10 to 6.7.

Perhaps the time has come for editors to insist that, as a minimum, figures on small babies are reported by the number born and the number dying at each weight. Only so can we compare—and perhaps dispute.

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¹ Macfarlane, A, *Lancet*, 1979, 2, 255.

Breast or bottle

SIR,—I greatly enjoyed Dr Paula H Bolton-Maggs's paper "Breast or bottle" (11 August, p 371) and agree entirely with her that, as a nation, we appear to consider breast-feeding unusual and maybe even socially rather unacceptable. Perhaps, women doctors, knowing the undeniable medical advantages of breast-feeding, should themselves be given special encouragement to breast-feed so that they may in turn be better qualified to advise others.

Unfortunately, the medical mother wishing to breast feed may find herself faced with two major areas of difficulty. Firstly, as Dr Bolton-Maggs suggests, attending staff often seem to assume that doctor patients "know it all." Indeed, in some instances, this may be reinforced by the doctor mother's reticence to admit what she may consider her own inadequacies. No matter how well medically qualified a woman is, motherhood is at first

strange and stressful and the doctor mother is not immune to the problems associated with establishing breast-feeding. At this critical stage she needs to be actively offered the help and advice she may feel reluctant to seek for herself.

Secondly, a medical mother wishing to pursue her career in what is a male-dominated profession may feel under pressure to "put the baby on the bottle" as soon as possible so that she can return to work early and thus minimise the inconvenience to her male colleagues and subsequent adverse comment from them.

Of course, neither of these problems is restricted to doctor mothers. But, given that they are likely to form a highly motivated and, hopefully, articulate group, should we not try to make breast-feeding easy and enjoyable for them, in the hope that we may produce within the profession a core of individuals experienced in breast feeding and enthusiastic about it to counteract the prevailing ambivalence which Dr Bolton-Maggs describes?

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SIR,—Like Dr Paula H Bolton-Maggs (11 August, p 371), I left medical school believing that determination was the vital factor in successful breast-feeding. I too was determined, and yet my hungry baby failed to thrive, and I eventually had to give in and give bottles.

Too late, I discovered Penny and Andrew Stanway's book *Breast is Best*,¹ with its refreshingly simple conclusion that to boost an inadequate milk supply one must increase the total sucking time. Obvious this may be, but it is far from being a widely taught truth. Instead one is advised to limit sucking time to "10 minutes a side" lest cracked nipples develop. Frequent feeding is discouraged, lest mother becomes tired—though it is far more tiring to pace the floor with a hungry, crying babe. Again, feeding one's infant more often than four hourly is considered to be evidence per se of an inadequate milk supply and an indication to start complementary feeds, when in reality it is just what the hungry babe needs. The weekly weighing often advised can induce such anxiety that relaxed feeding becomes impossible. The deficiencies of maternity units in failing to encourage early suckling, in proffering complements at the slightest excuse, and in separating mother and baby are but the beginnings of one's problems.

It is easy to make lack of maternal determination the scapegoat for all breast-feeding failures; but surely bad advice and unhelpful customs are often the real culprits?

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¹ Stanway, P, and Stanway, A, *Breast is Best: A Commonsense Approach to Breast-feeding*. London, Pan Books, 1978.

SIR,—I should like to put the other side of the coin in the discussion "Breast or bottle." Like Dr Paula H Bolton-Maggs (11 August, p 371) and Anne Chase (25 August, p 492) I also was determined to breast-feed my first baby. Unlike these two, however, I failed.

On being discharged from hospital after the birth, I was fully breast-feeding my infant, and he was gaining weight. On arriving home,

however, he seemed to completely change in personality from being a sleepy, placid baby to being a restless, demanding horror. He demanded feeds as often as hourly or two hourly—even through the night—and was very difficult to settle after feeds. Of course, I got the usual advice from my mother-in-law—“It’s your milk, dear, you haven’t got enough, or it’s too thin.” But I knew my physiology—all I had to do was let him suck, and Nature would do the rest. Nature did not.

With increasingly sore nipples and a baby continually screaming for something—apparently hungry, although he was rapidly gaining weight—I began to yearn for the simplicity of a bottle. At least I could then see what he had taken, and would know whether he was hungry or just bad tempered. The deciding point came after eight weeks of what can only be described as a nightmare, when my husband had to go to Malta on business, and I could accompany him. The decision was either to stay at home and battle on with the breast-feeding or to wean him on to a bottle, leave him with my mother, and go to sunny Malta for four days. Needless to say, the latter won and I have never regretted it.

I returned to my difficult baby with renewed energy, and I needed it. The change to bottle feeding had not altered my infant’s behaviour in the slightest—but it had altered mine. He still screamed all day long, but I no longer bared a breast at each scream but rather took him out for a walk, or carried him around strapped to me in a sling, as I knew from his milk consumption that he was unlikely to be hungry. I also discovered a new sense of freedom—I could go out shopping and leave my husband to feed him, and I could plan my return to part-time general practice. Night feeds also could be shared out, and my husband rapidly started to develop a relationship with his son which before had been totally absent.

It was five-and-a-half months before my child’s behaviour improved, and now, at almost a year, he is a delight. I have learnt many lessons from this experience—I still believe that “breast is best,” but I realise that in some situations bottle is more suitable. I feel that in our enthusiasm to promote breast-feeding it is important not to invoke a feeling of guilt in our patients who, like me, fail to breast-feed.

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Enuresis

SIR,—With reference to your leading article (17 March, p 705) and the article by Dr A J Cronin and others (p 722), if enuresis is treated as casually as you suggest, it shows little understanding of its main cause. If the child is otherwise normal the cause of wet beds is a small bladder capacity. If you take the trouble to ask the mother to measure the amount of urine the child can produce after holding on as long as possible you will find that there is a very small capacity. The treatment of enuresis is to increase that capacity. If this is done the condition cures itself. My normal policy is to get the child over the next school holiday to have a drink every time he passes a tap and to hold on five minutes after it starts to hurt. Within two weeks or so the problem has normally solved itself. The condition is caused by mothers and grandmothers who, on seeing the child naturally squirming at play,

say, “Go and wee before you wet yourself.” This results in a small bladder and a wet bed. Bells, buzzers, and drugs are very rarely required.

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Respiratory obstruction by epiglottitis

SIR,—I read with interest the report by Mr M Hardingham and Dr P N Young (4 August, p 309) of respiratory obstruction by the flaccid epiglottitis, and their comments on the phenomenon. A similar case illustrated my report¹ on the fiberoptic laryngoscope as a “steerable guide-wire,” rather than as an optical instrument, for difficult or trauma cases.

This patient had attempted suicide by cutting his throat with a pair of scissors, resulting in a narrow wound penetrating the thyrohyoid membrane and amputating the epiglottis. The epiglottis, held only by mucosal strips, had impacted over the laryngeal inlet, while the patient breathed through the neck wound. Conventional oral intubation with topical anaesthesia was possible only by means of the extreme manoeuvrability of the fiberoptic laryngoscope, while the patient breathed via an endotracheal tube through the neck wound.

There was structural rather than functional damage to the epiglottis in this patient, so that patients with both respiratory obstruction and neck wounds may be doubly at risk—which emphasises the dangers of inexpert anaesthesia mentioned in the report.

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¹ Davies, J R, *British Journal of Anaesthesia*, 1978, 50, 511.

Travel and health risks

SIR,—Professor Brian Macgraith’s suggestion (18 August, p 443) that the DHSS printed warnings against malaria be enclosed within travellers’ cheque folders is admirable enough, but surely as a week’s prophylaxis against malaria costs less than half a penny (pyrimethamine 25 mg) might it not be more effective for airline stewardesses to hand them out before flights through endemic areas instead of the customary glucose sweets?

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Measles and vaccine protection

SIR,—Dr G A Jackson’s survey (4 August, p 332) of measles in children who had been vaccinated against the condition failed to take into account the population at risk: the number of children susceptible to measles (having no history of measles and being exposed to the possibility of infection) who were in the specified area during 1978. If that number were 1470 and they were vaccinated and then exposed to infection with measles the fact that 147 developed the illness would indicate a protection rate by the vaccine of 90%.

Without the number of susceptibles, the conclusion that the vaccine had lost potency is more than these data will support—a point

that is well illustrated by Canadian workers in WHO’s *Weekly Epidemiological Record* (1979, 54, 210).

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Unusual manifestation of hypocalcaemia

SIR,—As Dr K Graham and others have recently pointed out (2 June, p 1460), the clinical manifestations of hypoparathyroidism in the elderly can be atypical. I would like to draw attention to an unusual physical sign of hypocalcaemia in an elderly person.

The patient, an 81-year-old man, had been confused and restless during the three weeks before he was brought into hospital. On admission, he was disorientated and uncooperative. He had a marked dorsal kyphosis and the head was stretched forward. The trunk was so rigid that his head and neck did not touch the pillow when he lay on his back. Ankylosing spondylitis was suspected but there was no x-ray evidence for this. Laboratory investigations showed: serum calcium 1.25 mmol/l (5.0 mg/100 ml); phosphorus 2.8 mmol/l (8.5 mg/100 ml); alkaline phosphatase 3.7 Bodansky units; urea 3.9 mmol/l (20 mg/100 ml); albumin 36 g/l; globulin 28 g/l. After treatment, the calcium rose to 2.2 mmol/l (8.8 mg/100 ml), while the patient became fully conscious; his back and head assumed a normal posture and the spasticity disappeared. Idiopathic hypoparathyroidism was diagnosed.

There have been several reported cases of idiopathic hypoparathyroidism presenting with a clinical picture reminiscent of ankylosing spondylitis.^{1 2} In these cases, however, there were paraspinal calcifications on the radiograph and the condition was not reversible when the serum calcium returned to normal.

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¹ Adams, J E, and Davies, M, *Postgraduate Medical Journal*, 1977, 53, 167.

² Chaykin, L B, Frame, B, and Sigler, J W, *Annals of Internal Medicine*, 1969, 70, 995.

Chronic ambulatory peritoneal dialysis

SIR,—I read with interest your leading article (28 July, p 229) on chronic ambulatory peritoneal dialysis. May I draw your attention to two points?

The correct name of the technique you described is continuous ambulatory peritoneal dialysis or CAPD. By describing it as chronic you miss one of the major points of this technique—that is, it is continuous, which is a unique characteristic of this treatment.

The continuous ambulatory peritoneal dialysis was first described by Popovitch and his colleagues in 1976, as you have mentioned, but these authors were using dialysate in glass containers, resulting in a high incidence of peritonitis. The technique that you describe in your editorial in which “while the dialysate is in the peritoneal cavity the plastic tubing set and the bag are folded into a small container which is held around the patient’s waist” is the Toronto Western Hospital technique for CAPD, which was first presented at the meeting of the American Society for Artificial Internal Organs in 1978.¹ Using this technique we have now trained 95 patients and our