Induction of labour

SIR,—Your leading article (18 August, p 407) reviewing the role of induction of labour in obstetric practice rightly points out that the controversies of recent years relate to orthodox induction by amniotomy and intravenous oxytocin and that these arguments are likely to change with the adoption of new methods of induction using prostaglandins. Unfortunately, however, scant justice is done to the prostaglandin developments, which extend over several years, and there is reference to only one recent study.

The cervical ripening effect of prostaglandin E_2 (PGE_2) was first observed in early studies using intravenous infusion in amounts insufficient to induce labour.¹ Intravenous therapy was soon superseded by local administration and in the Oxford unit it was shown that extra-amniotic infusion of PGE_2 ripened the cervix in primigravidae with poor induction prospects (low Bishop scores).² Later it was found that similar beneficial effects and improved prospects for induced labour could be achieved by a single administration of PGE_2 in a viscous cellulose-based gel into the extra-amniotic space.³

The most recent phase in the development of the use of prostaglandins in the induction of labour has been the adoption of vaginal administration of PGE_2 , either in a viscous gel or in a simple lipid-based pessary, or of tablets manufactured for oral administration.⁴ Fears that the larger dose of PGE_2 required for vaginal administration might lead to hypertonic uterine activity have not been substantiated, and there is now extensive experience of the efficacy and safety of the method.

The results of vaginal administration were first reported in 1977. Using PGE_2 gel in 168 primigravidae with low inducibility scores, the Oxford researchers⁵ reported that in almost 50% of patients labour ensued without further intervention; while in the remainder the Bishop score improved significantly, making subsequent amniotomy easy and progress in labour rapid. The method proved so satisfactory—and popular with patients and nursing staff—that its use was soon extended to patients with more favourable induction prognosis; and in the analysis of 803 patients⁶ it was found that when the cervix was ripe 65.9% of primigravidae and 87.5% of multiparae did not require formal induction of labour. The benefits are greatest when labour follows PGE_2 alone, and in such patients the frequency of caesarean section, epidural analgesia, and low Apgar scores were all reduced. In a continuing experience of using vaginal prostaglandin gels and pessaries their benefits in improving prognosis in labour have been substantiated. A recent paper from Oxford⁷ reported over 1500 patients safely treated with PGE_2 gel, including 78 women with fetal breech presentation and 54 multigravidae previously delivered by caesarean section.

The method has been adopted in many other units both in the UK and abroad.⁸ It would be still more widely used were it not for the disadvantage that no commercial product is available and the gel or pessary has to be prepared before clinical use. The chief reason for this is the inherent instability of PGE_2 compared with the less effective $PGF_{2\alpha}$; formulation in simple gels and pessaries does not provide a product with adequate long-term stability and this has discouraged commercial production.

No doubt these difficulties will be overcome, probably by the development of a better delivery vehicle; and the provision of a solitary stable PGE_2 pessary for induction of labour will have great potential. The prostaglandin regimen is attractive: it is simple, non-invasive, and highly acceptable to patients, causing minimal inconvenience while reducing analgesic requirements and improving the prospects of labour.

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Anti-D immunoglobulin and abortion

SIR,—The introduction of anti-D immunoglobulin injections to Rh-negative women delivered of Rh-positive infants brought about a dramatic fall in the incidence of Rh-haemolytic disease. However, in the last couple of years this welcome decline has halted and the reasons for this have been documented.^{1,2} In summary, new cases continue to occur because some mothers are sensitised during a pregnancy before the anti-D immunoglobulin can be administered (this is particularly serious in primigravidae) or because a mother, although eligible, fails to receive an injection after delivery—the so-called “administrative failure.”

If administrative failures could be completely eliminated this would reduce the incidence of new cases by half.¹ The purpose of this letter is to emphasise the importance of abortion as a sensitising factor in Rh-negative women. Of the 35 cases of administrative failures encountered in this region in 1977-8, no less than 14 (40%) appear to have been sensitised by a previous abortion where no anti-D immunoglobulin had been administered. At least four of these mothers had experienced a surgical termination of the pregnancy and it should be possible to avoid this type of omission. It is more difficult with the spontaneous abortion. The mother or her doctor (or both) may be unaware she is aborting, or the mother's blood group may not be known. However, if Rh sensitisation is to be reduced to a minimum these mothers should receive anti-D immunoglobulin.

The recent survey by the Medical Services Study Group on deaths from Rh disease³ has clearly shown that this is a national problem. Because of this I am hoping this letter will alert all doctors looking after mothers who are aborting to ensure that they obtain the mother's blood group and, if this is found to be Rh negative, administer a 50- μ g dose of anti-D immunoglobulin if the abortion is at 20 weeks or less and 100 μ g if it is after 20 weeks.⁴ If an abortion is clinically very probable but not certain, I would advise that an injection is given.

Many doctors do not appear to be aware that certain manoeuvres during pregnancy—for example, amniocentesis, external version—can also be sensitising factors. We encountered a Rh-negative woman recently who had an amniocentesis for genetic reasons early in the pregnancy, did not receive anti-D immunoglobulin, and developed antibodies later in the pregnancy. These cases should also receive 50 μ g anti-D immunoglobulin at the time of the episode.

If any doctor has any doubts about whether a patient should receive anti-D immunoglobulin or not, his local obstetrician, haematologist, or transfusion director should be consulted.

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Effect of beta-blockers on arrhythmias

SIR,—Dr J M Roland and others (1 September, p 518) maintain that their paper provides evidence suggestive that “serious” ventricular arrhythmias do not constitute an independent risk factor and evidently do little harm. The only point on which I take issue is that their results are based on data obtained 17-18 hours after the onset of symptoms. One cannot draw the same conclusions when these arrhythmias occur within the first hour or two.

Of the deaths from myocardial infarction 40% occur within one hour of the onset of symptoms.¹ It is likely that most of these deaths result from primary ventricular fibrillation (PVF).² Again, epidemiological studies of myocardial infarction from Teesside³ showed that over 50% of patients died within one month, half of these deaths being sudden deaths, and most of the other deaths (21%) were within the first two hours. Sudden deaths after coronary occlusion are frequently the result of PVF,⁴ suggested by the fact that in cases of sudden deaths without cardiac necrosis no coronary thrombi are found.^{5,6}

Lie et al,⁷ found that, out of 400 patients with acute myocardial infarction, 14 out of the 18 who developed PVF did so in the first six hours and that nine of these had warning symptoms, mostly vulnerable ectopics (R-on-T phenomenon), salvoes of ectopics, or ventricular tachycardia. Patients who entered PVF without warning did so therefore at the onset of disturbances which, had they persisted, were of a warning nature. The surprisingly large number who died suddenly with classical symptoms and had no thrombotic occlusion at necropsy is a point in favour of the argument that the ischaemia itself at the onset of infarction may be a potent arrhythmic stimulus,⁸ probably as a result of potassium loss from the injured cells⁹ causing a shortening of the action potential and refractory period and slowing of conduction. This favours further impulse re-entry and electrical dysynchronisation with rapid breakdown into ventricular fibrillation. It is significant therefore that the refractory period is very short in extrasystoles followed by multiple responses.¹⁰

The high initial and prehospital mortality