(6 January, pp 7 and 10), we feel that some critics may have rushed to defend the forceps without reading our papers carefully. Of course, many different intrapartum events might generate neonatal morbidity in association with the use of Kielland's forceps. However, we clearly demonstrated an excess of babies with abnormal neurological behaviour even in the absence of preceding fetal asphyxia. Furthermore, we looked for an adverse influence of mechanical factors operating during labour, and, within the limits of a retrospective study, we did not find it.

We agree that a prospective controlled trial of different ways of managing malposition is necessary to assess accurately the neonatal morbidity caused by Kielland's forceps. Indeed, we stated that long-term follow-up studies of babies were needed to assess properly the role of caesarean section for malposition. Pending such investigations one should not dismiss any retrospective study that draws attention to neonatal problems associated with current obstetric practice—a sentiment that incidentally is supported by the tone of your leading article on the subject (10 February, p 362).

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* * * This correspondence is now closed.—ED,
BMJ.

"Doctors' orders"

SIR,—We should like to raise two points which arise from the interesting article by Dr D A Ellis and others (17 February, p 456).

Communication is a two-way process and, while written information may have improved the patient's reception and therefore understanding and recall of advice, the exercise of writing down the advice may have meant that it was given in a more lucid and concise way by the doctor. Whatever the mechanism, improving the communication of "doctors' orders" to patients is an important step forward.

During investigations of the circumstances leading up to the deaths of infants, both at home and shortly after admission to hospital, we have been disturbed by parents' lack of understanding of advice given by general practitioners during consultations in the children's terminal illness. Particular difficulties have arisen not so much with adherence to drug dose schedules (as these are usually written on bottle labels by the pharmacist) as with how the parents should recognise deterioration in the children's illness, how long medication can be expected to take to work, what constitutes deterioration, and what to do should deterioration in the condition occur. Clear instructions should be left with the parents that they should *not* wait for days for medication to be effective, and that deterioration in the child's illness—as marked by increasing severity or increasing numbers of symptoms—should be an indication for recalling the general practitioner.

We feel that written instructions would be as valuable in the primary care situation as in hospital. They would, in many cases, mean that "doctors' orders" were more concise and

adequate in themselves, thus helping parents' understanding of them, and also enabling them to check instructions at a later time. As a result of this, infants would receive earlier medical reappraisal of illness when necessary, with possible lowering of morbidity and mortality.

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Thrombocytopenia and splenic infarction

SIR,—Dr P T Wilmshurst and others (10 February, p 381) have reported thrombocytopenia in a patient with renal carcinoma which they think may have been related to splenic infarction. This is an interesting suggestion, as important splenic infarction is usually associated with a raised platelet count as a result of hyposplenism.¹ In the absence of disseminated intravascular coagulation, thrombocythaemia is also the more common platelet abnormality associated with carcinomas.²

As the patient had a palpable spleen, it is important that the possibility of hypersplenism should be considered. It would be helpful to know: (a) the weight of the spleen at necropsy and (b) should splenic infarction have occurred at a late stage in the patient's illness, whether terminally there was a palpable spleen and thrombocytopenia. The hypersplenism could be either idiopathic or secondary to splenic congestion.

I would be happier to accept the suggestion of Wilmshurst *et al* that the thrombocytopenia was due to splenic infarction if hypersplenism could be excluded, and if "the diffuse intravascular coagulation screen" ruled out the possibility of chronic consumption coagulopathy—the latter being very likely in the presence of a mass of necrotic tissue.³

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¹ Crosby, W H, *Annual Review of Medicine*, 1963, **14**, 349.

² Weatherall, D J, and Bradley, J, *Blood and its Disorders*, p 1387. Oxford, Blackwell, 1974.

³ Harker, L A, *Recent Advances in Haematology*, p 349. Edinburgh, Churchill Livingstone, 1977.

Is there a time factor in renal allograft rejection?

SIR,—The claim by Dr M S Knapp and others (13 January, p 75) to be able to accurately time rejection episodes in renal allograft recipients has interested us. We have therefore analysed 21 rejection episodes in 21 patients in the first postoperative month by extrapolating sequential measurements of plasma creatinine as described.¹ All blood samples were taken at approximately 0900. The rejection episodes were all well documented by abrupt changes in plasma creatinine concentration and in all cases there was additional clinical evidence. In many cases there was also histological evidence. We have found, contrary to Dr Knapp and his colleagues, a random distribution of rejection episodes throughout the day and night, with 11 episodes occurring between 2300 and 1100 and 10 episodes between 1100 and 2300.

We have recently, for other reasons, changed the time of administration of immunosuppressive agents in our renal allograft recipients

from 2000 to 0800. In the absence of any other changes in therapy, we have therefore been able to compare the influence of dose timing on allograft function. The number of rejection episodes and the severity of rejection as assessed by the total dose of methylprednisolone (given as bolus doses during acute rejection episodes) in the first six weeks after transplantation were analysed. Eight patients who received immunosuppressives at 2000 were matched as closely as possible with respect to age, sex, and degree of HLA matching with the same number of patients receiving immunosuppressives at 0800. All patients studied were transplanted within the same 12-month period.

It was clearly seen that there were no significant differences between the two groups with respect to age, sex, HLA matching, number of rejection episodes, total dose of prednisolone, and graft function at the end of six weeks after transplantation. We have therefore found no evidence of a circadian rhythm in renal allograft rejection and no influence of the time of administration of immunosuppressive agents on the frequency and severity of rejection episodes during the first six postoperative weeks. The accuracy of timing rejection episodes by the method described in the paper is in our opinion questionable because of the errors inherent in extrapolation.

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SIR,—Dr M S Knapp and others (13 January, p 75) have suggested that renal allograft rejection is a sudden event, basing their assertion on plots of the reciprocal of plasma creatinine concentration against time (since transplantation) for selected rejection episodes. These episodes were chosen because the values before and after rejection lay approximately on two straight lines, but they do not say how many rejection episodes do not look like this.

After transplantation (or after the kidney starts to function) the plasma creatinine falls from a very high concentration to a relatively stable low level. Rejection will be accompanied by a rise in creatinine concentration which may become very rapid. The fall after transplantation and the rise associated with rejection are two separate processes, and the effect described by Knapp *et al* will be apparent only if rejection starts before the creatinine level has stabilised. Otherwise the data will show a level plateau between the two periods of change (whether or not reciprocals are used). It would be interesting to know how many of their 22 rejection episodes occurred within a week of the kidney's starting to function or a previous rejection episode.

The method of fitting two straight lines and taking the extrapolated point of intersection as the time of rejection has several weaknesses. Firstly, extrapolation is a hazardous procedure. Secondly, there are problems of allocating points near to the intersection to the "up" or "down" phase. Thirdly, the points on the second line may well have been affected by the treatment (see their figure). Finally, the reciprocal transformation will produce values of unequal reliability—errors in low (raw) values will be magnified.

My figure shows that if the data in their