SIR,—With reference to the article "Postnatal depression: does no one care?" (8 December, p 1487), I would like to point out that the subject of postpartum psychiatric disorders has been scrutinised and been the subject of a lot of time and energy of many researchers for over a century.¹ Needless to say, numerous papers and research projects have been published during this time. These concern many aspects of the whole spectrum of these disorders—from acute psychotic disorders² to the "three-day blues syndrome,"³ including postpartum depression.⁴

With this history in mind I would like to reassure the author that the lack of "appropriate hormones" to treat people like his wife is not the result of people "not having a care" to answer the queries he poses but is much more likely to be a direct reflection of the complexity of the biochemical and endocrinological state of the psychiatrically ill puerperal women.

As far as the "lack of warning" that patients will be affected is concerned, epidemiological studies and research into the aetiology of these disorders are plentiful⁵⁻⁷ and concern in this respect is in an ever-evolving state.

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¹ Savage G. *Guy's Hosp Rep* 1875;20:83-117.

² Karnosh LJ, Hope JM. *Am J Psychiat* 1937;94:537.

³ Pitt B. *Br J Psychiat* 1973;122:431-3.

⁴ Pitt B. *Br J Psychiat* 1968;114:1325-35.

⁵ Nilsson A, Kaij L, Jacobson L. *J Psychosom Res* 1967;10:341-7.

⁶ Kaij L, Nilsson A, Jacobson L. *J Psychosom Res* 1967;10:317-25.

⁷ Jacobson L, Kaij L, Nilsson A. *Br Med J* 1965;1:1640-3.

SIR,—I read with interest "Postnatal depression: does no one care?" (8 December, p 1487).

As a practising health visitor, I found it strange that the couple concerned did not seek or were not offered support from a health visitor during this stressful event in their lives. The author describes the care of the mother in the puerperium as one of the few remaining deficiencies in an otherwise magnificent and comprehensive health service. The care of the mother in the puerperium, both physically and emotionally, has always been and will continue to be a health visiting priority.

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SIR,—Your contributor's wife has come through a severe postnatal depression and lived to tell the tale, but only just. Instead of "Does no one care?" could I ask what went wrong?

I thought that the article was a little short of background. There was no mention of postnatal visits from the midwife, no mention at all of the health visitor—surely one of the first people to turn to. Could a health visitor or general practitioner restrict visits to one a week when the mother and baby are in danger? Surely an attempted suicide merits either daily visits or referral to a psychiatrist for inpatient or outpatient treatment.

Your contributor feels that postnatal depression has a hormonal cause but surely other factors are just as important: responsibility, the feeling of being alone with an unpredictable baby, lack of sleep, loss of previous role,

and possibly lack of preparation. It is the job of all concerned to treat postnatal depression actively, but we have a bigger part to play in preventing problems in the neonatal period developing into a full-blown depression.

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SIR,—As a psychotherapist, I read with great interest the article "Postnatal depression: does no one care?" (8 December, p 1487). First of all, let me express my admiration for the charming and sensitive way in which it was written. Of course, lots of people do care and many are concerned—fortunately one being this gentleman's doctor.

Regarding the responsibility of hormones, I have it from the horse's mouth that profound changes in the production and levels of various hormones—particularly oestrogens and progesterone—occur during and at the end of every woman's pregnancy. It is therefore clear that these changes alone cannot be held responsible for the postnatal depression which affects only a small proportion of women who have undergone pregnancy. Other factors must be involved and it is reasonable to suppose that these include the woman's emotional reactions to her past and present experiences.

It is curious that no mention is made of the central figure—namely, the baby. Who was caring for baby, mother, and home, for instance? Where were the grandmothers (important people, these)? We are not told the duration of the illness, or the effect of the relationship on the mother and her child, to say nothing of that between the husband and his wife. Although it is made out that all is now well, I find it hard to believe that your correspondent can feel free from the anxiety of a recurrence of emotional problems should there be another moment of critical life experience.

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Kidney transplants and long-term immunosuppression

SIR,—The observations on our paper (18 August, p 421) made by Dr B Jones (22 September, p 735) and by Dr H Graf and others (13 October, p 936) deserve some additional comment.

Since the mechanisms involved in graft acceptance or rejection are many and specific for every tissue,¹ in-vitro tests have been often disappointing as predictors of graft rejection.² We wonder therefore if reliable data on the well-being of an allograft may be obtained by performing some in-vitro tests on peripheral blood cells such as the mixed lymphocyte culture or lymphocyte-mediated cytotoxicity test.

Moreover, it was not our aim to correlate the alterations of the immune parameters studied with the acceptance of the graft. We have detected and reported only the concomitant existence of these events. We personally think that non-specific immunosuppression induced by corticosteroids plus azathioprine and antilymphocyte globulin is one but perhaps not the most important factor in graft acceptance or rejection.

With regard to the second letter, we must state that, in the absence of side effects, we have never withdrawn immunosuppressive treatment in transplant recipients because we are aware of the possibility of rejection. These data are reported in the literature,³ and we also had the opportunity to see in a kidney transplant recipient an irreversible rejection crisis after the spontaneous suspension of immunosuppressive treatment. None the less, we consider our case report is interesting because it raises many questions. We hope that answers will rapidly follow.

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² Dosssetor JB, Myburgh JA. *Transpl Proc* 1978;10:661-670.

³ Dandavino R, Trunet P, Descamps B, Kreis H. *Transpl Proc* 1978;10:655-657.

Donor kidneys and restrictions on their supply

SIR,—Guidelines now published by the DHSS on organ donation and transplantation (15 December, p 1596) promise to lend a questionable authority to the oversimplified thesis that the supply of kidneys could easily be increased if, as well as more understanding from the public, there was better co-operation from doctors. Much is written of the difficulties met by the transplant surgeon in harvesting kidneys, even by those with an essentially lay interest,¹ but less is heard of the practical problems encountered in setting up kidney donation from ventilated cadavers. May we try to redress the balance?

The neurosurgical intensive care ward in Swansea has over several years contributed not only to satisfying our Cardiff transplant colleagues' own requirements but to making Cardiff a major "net exporter" of kidneys, so that we can fairly be counted as positively motivated providers. The three cardinal clinical donor groups are patients with severe head injury, primary intracranial haemorrhage, and terminal primary intracranial tumour. All patients in these three categories who fulfil the royal colleges' criteria for brain death² while being ventilated are regarded as potential donors and it is the clinician who has care of the donor patient who approaches the family for consent. During 1978 and 1979 there have been 86 such cases. In most instances we have supplemented these criteria with the evidence of an electroencephalogram: an isoelectric record is a great "reassurance" to nurses and doctors as well as to relatives.

The potential donors are, however, whittled down by various exclusions, which we have classified as age and cardiorespiratory factors, infection and other medical reasons, refusal of consent, and miscellaneous circumstances. Exclusion by age implies a limit of 60 years, though the age factor has more recently become a biological rather than a chronological concept. Cardiorespiratory factors include obvious arteriopathic disease, known hypertension in the donor, prolonged refractory renal underperfusion, and sudden cardiac

arrest or respiratory obstruction during "life support." The category of other medical factors includes known or presumptive renal disease, some types of long-term drug therapy, and various constitutional diseases with a nephropathic component. Where the next-of-kin categorically refuse consent or after private discussion withdraw it, we make no attempt to persuade or cajole but let the matter drop. The "miscellaneous" category of exclusion contains instances where the donor's death had such peculiarly painful and distressing overtones that to seek consent from distraught relatives would have seemed inhuman; where the donor's wishes were known in life; where no recipient could be found; and where (an important obstacle in the context of acute surgical practice) theatre time, space, and expertise could not all be stage managed at the optimal moment. Such cases were rare. Donation has never been interdicted by the coroner, but this is always a possibility. As a result of these various exclusions, only 15 (17%) of our potential donors became actual donors.

Our figures suggest that, with the best will in the world on the part of activist donor clinicians, it still cannot be expected that the kidney harvest will readily be doubled. Even if it were, the demands of recipients would not be met. Our data also give rise to speculation about why accident victims provide proportionately so many more donors and so many fewer refusals of consent than tumour and haemorrhage victims. Of the accident victims, 30% became donors, 38% being excluded because of age or cardiorespiratory factors and 8% because of refusal of consent; for the patients with intracranial tumours the corresponding proportions were 6%, 50%, and 25% and for patients with intracranial haemorrhage 7%, 60%, and 16%. Some of the reasons are obvious, others not.

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¹ Osborne DJ, Gruneberg MM. *Injury* 1979;11:3-9.

² Conference of Medical Royal Colleges and their Faculties in the United Kingdom. *Br Med J* 1979;i:332.

Renal impairment and lithium

SIR,—Diane Kimbrell and her colleagues (3 November, p 1145) have joined the argument concerning the possibility of lithium nephrotoxicity with more data on serum creatinine levels. We feel that it should be stated again that serum creatinine concentration is an inaccurate measure of renal function and that creatinine clearance at least should be quoted if at all possible. The inferences about the degree of renal impairment and the cause-effect relationship of renal impairment with lithium assessed by this means are even more tenuous. We would agree with the recent leading article in the *Lancet*¹ that there are grounds for cautious optimism in interpreting results so far obtained on the effects of lithium on the kidney.

The only satisfactory way to determine this issue is to compare a series of patients receiving prophylactic lithium with another group of patients who are matched with the lithium patients at least for age and preferably for diagnosis and also for concomitant administration of other psychotropic drugs. So far

as we know, a study of this type has not been done.

Finally, we would agree with points raised in the final paragraph. The recommended lithium concentrations quoted of 0.6-1.6 mmol/l may well be too high and may well result from earlier studies in which lithium was used as a therapeutic agent. Indeed, in our study of a group of unipolar and bipolar patients we found that successful prophylaxis could well occur at much lower concentrations, although we would point out that there seems to be a cut-off point in that our responders had an average serum lithium level of 0.69 mmol/l whereas the non-responders had an average serum concentration of 0.58 mmol/l, this difference being significant.

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¹ *Lancet* 1979;2:1056-7.

² Poole AJ, James HD, Hughes WC. *J Roy Soc Med* 1978;71:890-4.

Decline of mortality from cardiovascular diseases in North Karelia

SIR,—Dr J T Salonen and his colleagues, in their report (10 November, p 1178) on changes in morbidity and mortality during the community programme to control cardiovascular diseases in North Karelia, stated that mortality from cardiovascular diseases started to fall around 1970 both in the programme area (North Karelia) and in the control area (Kuopio county). We would like to supplement this interesting observation with findings from our recently completed studies on the development of mortality in the counties of Finland in 1961-75.

The decline of mortality from cardiovascular diseases was observed in all counties of Finland. In the western counties the decline began in the late 1960s—that is, about three years earlier than in the eastern counties such as North Karelia and Kuopio (see figure). The

beginning of the decline also was clearly earlier in urban than in rural areas.

The decline of mortality from cardiovascular diseases may be connected with changes in some exogenous or behavioural factors such as changes in dietary or other life habits, although improved treatment of cardiovascular diseases may also be involved. Our results suggest that these changes—whatever they are—have first taken place in the more urban western Finnish counties and somewhat later in more rural eastern counties such as North Karelia and Kuopio.

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Intravenous N-acetylcysteine: still the treatment of choice for paracetamol poisoning

SIR,—In their indignation at the title of our recent paper on the use of intravenous N-acetylcysteine for paracetamol poisoning (3 November, p 1097), Dr J A Vale and his colleagues (1 December, p 1435) have overlooked some important points.

They should know better than to perpetuate the commonly held but erroneous belief that patients with plasma paracetamol concentrations above our treatment line are inevitably "destined for severe hepatotoxicity." In attempts to justify this misconception (which gives a misleadingly optimistic impression of the efficacy of any treatment) they referred¹ inappropriately to studies we carried out years before our first description of a treatment line for paracetamol poisoning.² We stated clearly in our paper that severe liver damage occurred in only 58% of such patients receiving supportive therapy—survival without hepatotoxicity is not necessarily proof of efficacy of treatment. It is for this reason that we emphasised that efficacy is best assessed in patients at high risk (plasma paracetamol concentrations above a line joining semilogarithmic plots of 300 µg/ml at four hours and 45 µg/ml at 15 hours after ingestion), in whom the incidence of severe liver damage was 89% with supportive therapy alone. Only one of our 33 high-risk patients treated with intravenous N-acetylcysteine within 10 hours developed severe liver damage and it occurred in none treated within eight hours, in whom the mean maximum alanine aminotransferase concentration was only 27 IU/l. In contrast, Dr Vale and his colleagues reported severe liver damage in no less than six similarly poisoned patients given oral methionine within 10 hours—but, inexplicably, they failed to mention the total number in this group. How then can they seriously suggest that oral methionine is as effective as intravenous N-acetylcysteine? We should also point out that one of their patients developed acute renal failure despite oral methionine given within 10 hours. We have never encountered this complication with any specific intravenous therapy started within this time.

We also find it difficult to accept the apparent low incidence of vomiting before treatment (16%) in the patients described by Dr Vale and his colleagues, since in a recent prospective survey of patients requiring treatment we found that vomiting occurred before admission in 62% and in a further 15% subsequently. We wonder if this discrepancy reflects the difference between the indirect

Age-standardised mortality from cardiovascular diseases per 10 000 among males aged 35-64 in two eastern and two western counties of Finland and in the whole country in 1961-75 (three year moving averages).