Intraregional variation in treatment of end stage renal failure

SIR,—Although the rates of acceptance of patients with end stage renal failure for dialysis and transplantation are rising in the United Kingdom, there is a shortfall by comparison with European and North American figures. The recent paper by Dr Maureen Dalziel and Mr Chris Garrett (30 May, p 1382) suggests that the sparse geographical distribution of renal units may be part of the explanation, and Dr I T Wood and colleagues (6 June, p 1467) suggest the provision that will be necessary for a comprehensive balanced programme.

Using computer records we have reviewed our experience of 334 patients admitted between 1981 and 1986. They were referred from 10 district general hospitals, covering a population of about two million in West Yorkshire. Though there are minor overlaps with referrals to two other renal units from the districts concerned we found a similar negative correlation between the number of patients admitted per million of the population and the distance from the referring centre (1981 $r = -0.66$, $p = 0.023$; 1986 $r = 0.70$, $p = 0.012$) (population figures for 1986 from the Office of Population Censuses and Surveys refer to super-seded area health authorities). Only three of the district health authorities have achieved referral rates greater than 40 per million population, but this is influenced by cross referral. The highest acceptance rate represented 55 new patients per million population yearly.

Further analysis showed an interesting variation in early mortality. There was a bimodal distribution of the interval between starting treatment with dialysis and death, with a break point after 12 months. The 12 month mortality in patients accepted from districts with the highest referral rates was 11-17%, but from one district it was 27% (figure). In the centres with low referral rates the mortality was less than 16%. In addition, the distribution of 12 month mortality reflected the age structure of the population admitted for treatment.

Number of early deaths as percentage of number of referrals from each area health authority 1981-6.

Interestingly, the district referring patients with a high early mortality had been heavily exposed to our attempts to increase referral rates. Excellent local contacts ensured this, as did the rotation of a senior registrar trained in renal treatment through the local hospitals in alternate years.

This high early mortality, if confirmed by the experience of other centres, has important implications. It suggests the existence of a very high risk group, which will greatly affect projections about future resources. In addition, it suggests that the announcement of targets for treatment of end stage renal failure is only part of the strategy that should be used to increase referral to renal centres.

Clinicians who are open to influence from

nephrologists and who are already referring standard and high risk patients (as defined by Dr Wood and coworkers) may refer increasingly handicapped patients with very limited possibilities for survival and rehabilitation, whereas those who are unaware of the changes in criteria will continue to send only standard risk cases. A more comprehensive strategy than the announcement of the intention to treat to a target level is required, and the setting of targets will be ineffective without specific attempts to inform all the referring agencies.

If it is accepted that new initiatives are required there is no obvious focal point in current administrative structures. On a day to day level it is uncertain who is primarily responsible for the systematic encouragement for referral of patients with chronic renal failure—the renal unit clinicians or regional community doctors. There may well be a place for national initiatives to complement the announcement of targets, with some more coherent central planning, finance, and publicity. It is regrettable that there is no agency comparable to UK Transplant that could coordinate such a strategy. Perhaps it is time for the convening of more than a subcommittee of the Renal Association to deal with these problems.

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Changes in serum lipid concentrations after myocardial infarction

SIR,—We agree with Dr Rodney Jackson and colleagues (20 June, p 1588) that serum cholesterol concentrations in patients who have suffered myocardial infarctions may fall rapidly after the first 24 hours. Dr Jackson and coworkers have not, however, compared their values on admission with figures after discharge, and thus their assumption that the best time for measuring lipid concentrations is on admission to the coronary care unit may be overconfident.

In a study of serum lipoproteins in patients admitted to the coronary care unit in this district we confirmed the fall in serum cholesterol concentration during the first few days after myocardial infarction¹ but observed clinically important increases over concentrations at admission in several patients three months after discharge. Of 35 consecutive patients under 65 years of age admitted to the coronary care unit with myocardial infarction in 1986, 27 had higher cholesterol concentrations at three months than at admission. The range of serum cholesterol concentrations was 4.75-9.70 mmol/l on admission and 5.08-10.55 mmol/l after three months. In 13 of the 35 patients the increase in serum cholesterol concentration after three months was in excess of 1 mmol/l. Six of these patients already had raised cardiac enzyme activities on admission; one had had a cardiac arrest but had normal enzyme activities; 10 of the 13 patients had gained weight after discharge, and in four this may have been related to stopping or reducing smoking. Thus a number of factors may operate after discharge to increase serum cholesterol concentrations.

The reason for measuring lipid concentrations, particularly in younger patients admitted to coronary care units, is to detect continuing high risk due to lipidaemia and to identify families with hyperlipidaemia or familial hypercholesterolaemia. The best time to screen would be when fewest patients would be missed.

We are inviting younger patients to keep an appointment for a lipid check 10 weeks after

admission to the coronary care unit. Patients with symptomatic coronary artery disease are usually highly motivated to have health checks if given the opportunity.

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1 Fyfe T, Baxter RH, Cochran KM, Booth GM. Plasma lipid changes after myocardial infarction. *Lancet* 1971;ii:997-1001.

Atrial natriuretic peptide concentrations in pre-eclampsia

SIR,—The pathophysiology of atrial natriuretic peptide is slowly unfolding, as shown by the report by Dr Jørn Kvist Thomsen and colleagues (13 June, p 1508), which describes increased plasma concentrations of atrial natriuretic peptide in women with pre-eclampsia.

Raised plasma concentrations of any substance may result either from increased release of that substance into the bloodstream or from impaired degradation of the substance, and thus it is surprising that Dr Thomsen and colleagues firmly attribute the raised plasma concentrations that they observed to an increased release of the peptide from the heart into the circulation. Clearly, an alternative explanation is that the raised atrial natriuretic peptide concentrations were caused by impaired clearance of the peptide from the circulation in the presence of normal, or even reduced, secretion from the heart. Dr Thomsen and coworkers mention this possibility, but in both their concluding comments and their abstract they seem to discount it.

The kidneys receive a large proportion of the total cardiac output at rest. Renal vein plasma atrial natriuretic peptide concentrations are much lower than renal arterial concentrations.¹ This suggests that the kidneys are an important site of removal of the peptide from the circulation (as they are for many other peptide hormones²). Impairment of renal function is a well recognised manifestation of pre-eclampsia, and renal impairment from a wide variety of causes has been shown to produce enormously increased plasma atrial natriuretic peptide concentrations.^{3,4} A reduced capacity to clear atrial natriuretic peptide from the bloodstream seems to contribute to these high concentrations in non-pregnant patients with renal impairment, as correction of sodium and water overload by dialysis does not fully return plasma concentrations to normal.^{3,6}

Unfortunately, Dr Thomsen and colleagues do not present data on the renal function of their patients with pre-eclampsia. As frank renal failure causes such huge increases in plasma atrial natriuretic peptide concentration it is quite possible that even small changes of renal function in patients with pre-eclampsia might produce the modest changes in plasma atrial natriuretic peptide concentrations observed by the authors. It certainly seems rather rash to conclude that "these findings are clear evidence that atrial pressure may not be the principal determinant of the release of the natriuretic peptide in pre-eclampsia."

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1 Crozier IG, Nicholls MG, Ikram H, Espiner EA, Yandle TG, Jans S. Atrial natriuretic peptide in humans: production and clearance by various tissues. *Hypertension* 1986;8(suppl II):11-5.

2 Katz AI, Immanuel DS. Metabolism of polypeptide hormones by the kidney in uraemia. *Nephron* 1978;22:69-78.