and that we have as much to learn from Europe as vice versa.

The Hospital Doctors' Association has campaigned for two years for a single training grade for all branches of specialist medicine, in which the trainee would spend a shorter time and be more closely supervised than at present. This idea is now being examined by the Royal College of Obstetricians and Gynaecologists and piloted by the Royal College of Surgeons. I believe that Brearley is mistaken in seeking to extend the numbering system currently operated by the Royal College of Surgeons. It cannot be right to have two people in essentially the same job on the same rota, with one preselected for promotion and accreditation and the other denied this. Such a system will eventually fall foul of the courts in the United Kingdom or Brussels in the same way that the practices of the Joint Committee on Higher Medical Training have done already.

We must stop separating the issues of manpower, training, accreditation, and hours of work. They are all intimately linked. We need wholehearted reform of our practices of work and training, and the Hospital Doctors' Association has called for the royal colleges, the General Medical Council, the Department of Health, and the representative associations of the profession to meet and take up the challenge.

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1 Brearley S. Accreditation after Goldstein. *BMJ* 1992;**304**:518-9. (29 February.)

## Visiting registrars

SIR,—I congratulate Stephen Brearley on his editorial on accreditation.[1] A system riddled with anomalies was bound to have its days numbered. He goes on to say, however, that accreditation is a purely British phenomenon and designed to fulfil a uniquely British need. Does he mean the use of overseas doctors (alias the politically correct "visiting registrar") in district general hospitals, where the bulk of NHS work is done?

He recommends using visiting registrars for unrecognised senior registrar posts. Is this because they lack the necessary intelligence or because they do not deserve better? Once more the visiting registrar becomes the stop gap for unrecognised locum senior registrar posts. Justice is long overdue here too.

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## Renal protective effect of enalapril in diabetic nephropathy

SIR,—We feel that Staffan Björck and colleagues[1] are not justified in concluding that enalapril reduces the rate of decline in kidney function in diabetic nephropathy more than metoprolol, given a similar antihypertensive effect. The accompanying editorial by Carl Erik Mogensen[2] points to some disagreement between their results and those of similar studies.[3,4] Their results therefore need detailed scrutiny.

The question at issue is whether Björck *et al* did in fact achieve an equivalent antihypertensive effect with the enalapril and metoprolol based regimens. They emphasised the similarity in mean arterial pressure but ignored important differences in supine blood pressure. In absolute terms supine diastolic pressure was 6 mm Hg higher ($p<0.005$) with metoprolol, and the antihypertensive effect from baseline values was substantially smaller (11/1 mm Hg metoprolol; 17/12 mm Hg enalapril). One might argue about the relative importance of diastolic, systolic, or mean arterial pressure, but what is clear is that a mean difference in diastolic pressure of 6 mm Hg cannot be dismissed. For example, such a difference is associated with a 40% reduction in the incidence of stroke in intervention and epidemiological studies.[5] It is quite conceivable that the blood pressure differences observed could be responsible for the apparent benefit of enalapril on renal function.

There is no doubt that angiotensin converting enzyme inhibitors reduce proteinuria, a response observed also in non-diabetic nephropathy even when renal function tends to decline more rapidly than with other treatment.[6] However, the association between proteinuria and progression to end stage renal failure hinges predominantly on the relation observed in untreated patients,[7] which may not hold in the treated state. The hypothesis that angiotensin converting enzyme inhibitors are renoprotective is likely to be answered convincingly only by much larger studies than that of Björck *et al*, and preferably by studies measuring real rather than surrogate endpoints.

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1 Björck S, Mulec H, Johnsen SA, Norden G, Aurell M. Renal protective effects of enalapril in diabetic nephropathy. *BMJ* 1992;**304**:339-43. (8 February.)
2 Mogensen CE. Angiotensin converting enzyme inhibitors and diabetic nephropathy. *BMJ* 1992;**304**:327-8. (8 February.)
3 Parving HH, Smidt UM, Mathiesen ER, Hommel E. Ten years' experience with antihypertensive treatment in diabetic nephropathy. *Diabetologia* 1991;**34** (suppl 2):A38.
4 Gronhagen-Riska C, Honkanen E, Metsarinne K, Rosenlof K, Tikkanen I, Fyhrquist F. ACE-inhibtion versus conventional antihypertensive treatment (beta-blockade) in diabetic nephropathy. *Am J Hypertens* 1990;3:67A.
5 Collins R, Peto R, MacMahon S, Herbert P, Fiebach NH, Eberlein KA, *et al*. Blood pressure, stroke, and coronary heart disease. 2. Short-term reductions in blood pressure: overview of randomised drug trials in their epidemiological context. *Lancet* 1990;**335**:827-38.
6 Apperloo AJ, de Zeeuk D, Sluiter HG, de Jong PE. Differential effects of enalapril and atenolol on proteinuria and renal haemodynamics in non-diabetic renal disease. *BMJ* 1991;**303**: 821-4.
7 Williams PS, Fass G, Bone JM. Renal pathology and proteinuria determine progression in untreated mild/moderate chronic renal failure. *Q J Med* 1988;**252**:343-54.

SIR,—Staffan Björck and colleagues claim to have shown a specific beneficial effect, independent of blood pressure, of enalapril on the decline of glomerular filtration rate in diabetic nephropathy.[1] We would like to draw attention to several problems with this study that lead us to question the validity of the conclusions.

In both the interim report of their study and in the final paper Björck *et al* reported significantly lower supine diastolic blood pressure values in the enalapril group than in the metoprolol group.[1,2] Since even small differences in blood pressure above the normotensive range may have a substantial effect on the progression of diabetic nephropathy[3] the effect of enalapril cannot be attributed to an action of the drug independent of blood pressure.

Differences in the decline of glomerular filtration rate between the groups were only significant when the measurements of the rate before randomisation of patients were included in the evaluation; differences were not significantly different during the drug treatment period (table III).[1]

Deterioration of renal function over time was analysed by linear regression. This method of analysis can be used only if the decline of renal function is linear—but this was not the case in the enalapril group. After six months of treatment glomerular filtration rate deteriorated significantly in only the enalapril group, as stated in the interim report and in the final report (fig 3).[1] Since the decline of glomerular filtration rate in the enalapril group was not linear no linear regression analysis can be calculated for this group. A transient drop in glomerular filtration rate when enalapril was started may have artificially "reduced" the slope of the decline of glomerular filtration rate in a linear regression analysis. Unfortunately the authors do not give the final values for the glomerular filtration rate in each group and they do not state whether the rate was different between the groups at the end of the study. Most probably it was not.

Patients receiving drugs whose renal function deteriorated rapidly should have been excluded in both treatment groups (patient No 23) and not only in the enalapril group (patient No 21).

Patients with a follow up of only six months should have been excluded as this follow up period allowed glomerular filtration rate to be measured only once during drug treatment. The lack of further measurements renders any regression analysis of the decline of glomerular filtration rate under the influence of the investigated drugs impossible.

In conclusion, the study does not provide convincing evidence for a specific beneficial effect of enalapril on the decline of glomerular filtration rate in diabetic nephropathy.

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1 Björck S, Mulec H, Johnsen SA, Norden G, Aurell M. Renal protective effect of enalapril in diabetic nephropathy. *BMJ* 1992;**304**:339-42.
2 Björck S, Mulec H, Johnsen SA, Nyberg G, Aurell M. Contrasting effects of enalapril and metoprolol on proteinuria in diabetic nephropathy. *BMJ* 1990;**300**:904-7.
3 Sawicki PT, Didjurgeit U, Mühlhauser I, Heinemann L, Berger M. Near normotension and near normoglycaemia in blind type 1 diabetic patients with overt diabetic nephropathy. *J Diabetic Complications* 1990;**4**:179-83.

AUTHOR'S REPLY,—It was a new finding in our study that the enalapril group tended to have a lower blood pressure than the metoprolol group in the supine position and that the reverse occurred in the standing position. This phenomenon cannot be handled by an increased sample size. The mean of the supine and the standing blood pressure is the only measure that can be used for the evaluation because of this unexpected difference in antihypertensive action. If we had used more aggressive antihypertensive treatment to reduce supine blood pressure more in the metoprolol treated patients the two groups certainly would not have been comparable, with a much lower standing blood pressure in the metoprolol treated patients. The difference of 1 mm Hg that we obtained in mean arterial blood pressure between the two groups is very small.

Even without a control group the result of enalapril treatment can be seen to be very good and is the best reported in this type of patient. After the initial half year the fall in glomerular filtration rate was only 0·4 (6·9) ml/min/year. For the six patients followed for three years the corresponding figure was 0·4 (1·4) ml/min/year, which is remarkable in patients with diabetic nephropathy and a 50% reduction in renal function. The figure is the same as the normal, age dependent fall in glomerular filtration rate.[1]

Another conclusion from our data is that studies of angiotensin converting enzyme inhibitors should focus on the renal effects after renal function has been allowed to stabilise for some months. Therefore, even though ours is one of the larger prospective studies in diabetic nephropathy, new studies comparing other types of antihypertensive