mittees to have lists of those undergoing higher training; this will facilitate later accreditation as a specialist.

An outline of the arrangements for both of these procedures is given in the Second Report of the JCHMT recently published and referred to previously (29 November, p 532); the report is obtainable (price £1.50 including postage) at this address. However, in addition we have thought it worth while, through your columns, to inform all senior registrars (or equivalent) in the relevant specialties that they should soon seek enrolment by applying on the prescribed form, obtainable at the same address (see below for details). In the case of haematology application has also to be made to the Royal College of Pathologists, 2 Carlton House Terrace, London SW1Y 5AF. Even if a senior registrar post has not yet been visited and approved the holder should proceed to enrolment. Even if, after one of the current visits, the senior registrar post has *not* been approved, this does not affect the present holder, who should apply for enrolment.

It is expected that (except in the specialties of community medicine, dermatology, haematology, neurology, paediatrics, and venereology) most senior registrars in adult medicine will also seek enrolment in general (internal) medicine, though this is not mandatory. On application for enrolment they will therefore be sent two forms, one for general (internal) medicine and one for their chosen specialty. In the case of haematology two forms will be sent, one for the JCHMT and one for the Royal College of Pathologists. In the case of paediatric subspecialties it will be expected that enrolment will usually be sought also in general paediatrics; two forms will therefore be sent.

Those who are in the last six months of the training period recommended in the Second Report should, after enrolment, later apply to the JCHMT for the prescribed forms for accreditation. Again, where relevant, two forms will be sent for those who have been enrolled both in general (internal) medicine and in another specialty and for those who have been enrolled both in general paediatrics and in a paediatric subspecialty. Those who have been enrolled in haematology will receive two forms, one of which should be submitted to the Royal College of Pathologists.

For the present no consultant already in a post before 1 January 1977 need seek accreditation unless, in the future, he requires this in applying for a post in another country of the European Economic Community. After January 1977 any trainee who is appointed to a consultant post before he has achieved accreditation, which is entirely possible (see p 12 of the report), should apply to the JCHMT for accreditation, which would normally be granted.

The question of some sort of specialist registration after a duration of training less than that recommended in the Second Report but conforming to the minimal standards laid down by the Commission of the European Economic Community is still under discussion and no action is contemplated at present.

It will be appreciated that the publication of the Second Report, and this letter, may result in a very large mass of correspondence for the hard-pressed staff of the JCHMT, which is still receiving quite inadequate governmental financial support. We hope that trainees will bear with us if there are delays in correspondence. To simplify administration we suggest that those applying for *enrolment* should do so in the following months:

Names beginning with	Month in 1976 for applications
A-C	February
D-G	March
H-K	April
L-O	May
P-T	June
U-Z	July

Any exceptional case, if there is a reason for real urgency, may be dealt with outside this timetable.

Similarly we suggest that those seeking *accreditation*, who will mainly be those in the last six months of their training, should spread their applications as follows:

Names beginning with	Month in 1976 for applications
A-C	April
D-G	May
H-K	June
L-O	July
P-T	September
U-Z	October

Everyone will appreciate that this is a major administrative exercise which may take some time to be accomplished. We will do our best to deal with difficulties but appeal in advance for tolerance.

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Vesicoureteral reflux and its familial distribution

SIR,—Your leading article (27 December, p 726) admirably draws attention to a group of patients in whom vesicoureteral reflux (VUR) appears to be familial. It is important to differentiate clearly between the possibly inherited problem of VUR and the resulting renal damage (chronic pyelonephritis, reflux nephropathy). In order to document the familial problem fully it will be necessary to determine the hereditary pattern of VUR and the incidence of renal scarring within this group and compare them with a similar control population—an almost impossible task to perform with any degree of accuracy.

You suggest that as full radiological screening of all members of a family is impracticable and unjustified a compromise may be the zealous testing of the urine in siblings who are unwell for any reason and urography and/or cystography in families with more than one affected member or in whom renal scarring is evident. This is unsatisfactory, for, although the results would be of interest, they would probably not be of benefit to the patient. The discovery of VUR in an infant *after* its first infection may be too late, as scar formation in susceptible areas of the kidneys may already have been initiated.[1] It is well known that most children with chronic pyelonephritis have demonstrable scars when first seen, and the development of further areas of scarring while under medical supervision is very rare.[2]

A different compromise of greater potential benefit would be to concentrate on families in which the affected child is the eldest (but still under, say, 5 years) and preferably the only child. All subsequent siblings could then be investigated by cystography shortly after birth and those with VUR identified at a very early stage. At present there is no method of selecting from this group of infant refluxers all those in whom pyelonephritic scarring is likely to develop, but the presence of gross reflux appears to carry the greatest risk.[3] These children could be treated prophylactically by reimplantation of the ureters or continuous chemotherapy. The demonstration of a reduction in the incidence of renal scarring in children so treated would be of the greatest benefit and help to determine the policy for the remainder of the population of refluxing children.

The results of surgery in this age group are not known at present and perhaps chemotherapy should remain the initial treatment of choice until the results of a trial of surgery versus conservative treatment for infants with VUR become available. Such a trial is at present in progress jointly between the Institute of Urology and the Hospital for Sick Children, Great Ormond Street, London.

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1 *Lancet*, 1974, **2**, 1120.
2 Smellie, J M, *British Journal of Hospital Medicine*, 1974, **12**, 485.
3 Rolleston, G L, Shannon, F T, and Utley, W L, *British Medical Journal*, 1970, **1**, 460.

Carrier solutions for low-level intravenous insulin infusion

SIR,—Recently there has been growing interest in the use of low-dose continuous infusions of insulin in the management of diabetic crisis. A major advantage of this technique is that the patient's response is predictable in a way that was previously not possible. This was to be anticipated, since, in contrast to previous techniques, the infusion of insulin produces a sustained steady-state concentration of insulin in the plasma, the magnitude of which is directly proportional to the infusion rate.[1] Because of the very short half life of the hormone, plasma insulin concentration can be changed more or less instantaneously by simply altering the rate of infusion.

The technique was popularised following our studies on insulin metabolism.[1,2] In these and all other studies which involved the use of polypeptide hormones we have been careful to use albumin as a carrier protein since insulin and other polypeptide hormones are particularly prone to non-specific adsorption to "active surfaces." It seemed only logical therefore to add carrier protein to insulin infusions used therapeutically. Albumin is not easily available in convenient amounts and for this reason some have infused insulin without added carrier protein and, because they have so far encountered no problems, have encouraged others to adopt this practice.

The article by Dr E W Kragen and his colleagues (23 August, p 464) and the subsequent letter from Dr P F Semple and others (25 October, p 228) again highlight the fact that insulin losses from simple salt solutions are substantial and unpredictable[3] and advocate the use of carrier proteins—a view that I wish to endorse.

The use of low-dose insulin infusions in the management of diabetic coma has been included in the most recent edition of an already well-established textbook on the management of diabetes, with the statement that "insulin, in the concentration we use, does not adhere to the plastic of syringe or tubing so it is not necessary, as had been thought, to add human albumin to prevent adsorption."[4] This seems potentially dangerous since all adequately controlled studies that I am aware of are in agreement with the results obtained by Dr Semple and his colleagues, indicating that insulin losses are significant even at concentrations four times