enterostomy; most may therefore still need to be considered for transplantation.

In Europe 272 patients with biliary atresia underwent liver transplantation to December 1988 with a three year actuarial survival of 66%, most deaths occurring in the first 90 days (European Transplant Registry). The single most important prognostic factor in terms of outcome is the age and weight of the recipient. Children who weigh under 10 kg have a higher rate of technical failure often associated with hypoplastic vessels. Even so, Esquivel *et al* reported the Pittsburgh experience of 20 patients under the age of 1 year given transplants after failed Kasai procedures, of whom 12 (60%) were alive between 1 and 56 months.[5] In our own experience 14 children aged under 38 months have undergone liver replacement after failed portoenterostomy, with a current one year actuarial survival of 57%. Ten of these 14 children had a segmental reduction of an adult liver. The reduced or segmental liver allograft now offers the infant with failed portoenterostomy a reasonable prospect of survival with excellent quality of rehabilitation and to an extent overcomes the severe shortage of suitable paediatric donors.

While agreeing with Dr Nelson that the primary treatment of choice must be portoenterostomy at less than eight weeks from birth, we believe that at present most children in Europe with biliary atresia will not achieve satisfactory long term results and therefore transplantation may be an option which should be considered. It seems important in these children to avoid re-exploration and reintervention unless the prospect of re-establishing bile flow is good—namely, when bile flow has suddenly stopped in an otherwise successfully established portoenterostomy. Repeated explorations increase the technical difficulties and potential problems with blood loss, thus diminishing the child's ultimate chance of a successful liver transplant. In the current climate of reduced immunosuppression and with the prospect of not using steroids in the long run, liver transplantation offers the only realistic option for full rehabilitation.

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SIR,—We agree that infants with persistent jaundice need early referral to diagnose and treat biliary atresia.[1] We would suggest, however, that referral should be considered at 2 weeks of age if only to estimate a direct bilirubin concentration and consider organic liver disease early enough.

We would also like to comment on Dr Nelson's views of the success of Kasai portoenterostomy in managing biliary atresia, and the results of liver transplantation. Although many children achieve satisfactory bile drainage if operated on soon enough,[2] the results vary from centre to centre and 40-50% of children with "successful" Kasai operations will develop cirrhosis and portal hypertension and will require liver transplantation in time.[3] Survival after liver transplantation has improved since the introduction of cyclosporin and one year survival rates are now 75-80%,[4] with a further 13% dying one to five years after transplantation.[5]

Though we would agree that liver transplantation will not replace portoenterostomy as the initial operation for biliary atresia, any procedure which prolongs life increases the likelihood of a suitable donor being obtained. Multiple revisions of the portoenterostomy, however, undoubtedly increase the operative difficulty at transplantation and should be avoided.

There is not a liver transplant programme in Japan because of cultural reasons and not simply because of the success of the Kasai operation.

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Hypophosphataemic rickets after ifosfamide treatment

SIR,—Dr R Skinner and colleagues reported hypophosphataemic rickets after treatment of childhood malignancy with high dose ifosfamide.[1] A previous study also reported nephrotoxicity related to use of this drug in children: Smeitink *et al* in 1988 wrote of tubular toxicity leading to Fanconi's syndrome in three children and glomerular toxicity in one.[2]

Our own experience includes a case of ifosfamide induced Fanconi's syndrome leading to a pathological fracture and poor bone healing.[3] Hypomagnesaemia was also present even though the patient had not received cisplatin. We have been able to control the toxicity with supplements, but long term follow up is necessary in this and other cases to determine the degree of reversibility. While oral phosphate supplementation may be adequate, intravenous treatment was required in this case because of the difficulty in tolerating the high doses of oral phosphate required.

The other three of our children who received ifosfamide had persistently low serum phosphate values up to one year after finishing treatment. In one child difficulty walking and an abnormal gait might have been attributable to this.

We therefore support the conclusion that phosphate concentrations should be monitored closely in children receiving this drug, to prevent renal damage and its sequelae.

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General practitioners' attitudes towards AIDS

SIR,—In his study of general practitioners' attitudes towards AIDS Dr J Shapiro reported that 17% of those studied would test for HIV without informed consent and a further 7% were undecided.[1] I found similar responses amongst a random sample of 1000 general practitioners in New Zealand: 22% reported that informed consent for testing was not at all important and 4% were undecided. I, too, found that years since graduation was a significant variable as was the number of patients requesting an HIV test. Younger doctors and those with the most requests for tests believed more strongly in informed consent.

In addition I examined attitudes to anonymous testing and to confidentiality. Anonymous testing, although recommended and widely practised in New Zealand, was disagreed with by 28% of the sample, including 7% who disagreed strongly. In fact, only 41% agreed with it, the remainder being neutral. Attitudes to confidentiality were shown by the doctors' responses to questions on sharing information about patients with AIDS. Sixteen per cent of the doctors would give such information to reception staff, and the same proportion would give it to colleagues outside the practice. Women doctors were less likely to do this than men.

It is worrying that the findings of this study and those of Dr Shapiro show some indifference to major issues regarding patients' rights such as informed consent and anonymous testing. This is particularly sad given the crucial role that general practitioners will have in the future in caring for those with AIDS or HIV infection and their families. Such indifference will inevitably lead to mistrust on the part of patients and to a reluctance to seek help from general practitioners when it is needed. Future efforts in educating general practitioners about HIV must address these matters.

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Passive smoking and middle ear effusion in children

SIR,—Dr D Strachan and colleagues presented an interesting report on the possible association of passive smoking with otitis media with effusion,[1] but there are three points that need addressing before their conclusions can be reached.

Eustachian tube dysfunction is extremely common in children and gives rise to negative middle ear pressure and middle ear effusion. The prevalence of middle ear effusion varies inversely with age, but there is also a marked seasonal variation, possibly related to a similar variation in upper respiratory tract infections.[2] The authors have quite rightly confined themselves to a single age group, but they carried out their tests over a period from January to June. Those tested earlier should have a higher rate of abnormal results, but this is not taken into account in the analysis.

Perhaps more importantly they fail to indicate whether the children had already had ear, nose, and throat operations (which at the age of 7 must be a considerable percentage) or whether there were other important factors such as cleft palate or Down's syndrome.

As otolaryngologists we deplore the use of tympanometry alone in the diagnosis of middle ear disease. It is a useful screening test, but in the presence of an abnormal finding we believe otoscopy must be performed. Frequently an obvious cause for the flat tympanogram such as wax, perforation, or even a grommet will be found. Dr Strachan and colleagues unfortunately do not seem to have checked their findings with otoscopy.

A possible indicator of this fault can be found in