For the avoidance of halothane to be recommended in children requiring repeat anaesthesia another volatile anaesthetic agent must be shown to be associated with a lower overall morbidity and mortality. Avoiding halothane might eliminate the risk of unexplained hepatitis after administration of halothane, but a different complication with a higher overall mortality might replace this.

I will continue to use halothane repeatedly in children as it is superior to all currently available volatile anaesthetic agents. The only contraindication to its use is if a child has previously developed unexplained hepatitis after receiving halothane.⁵ Previous postoperative fevers and the appearance of a rash are of no consequence.

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- 1 Wark HJ. Postoperative jaundice in children. The influence of halothane. *Anaesthesia* 1983;38:237-42.
- 2 Warner LO, Beach TP, Garwin JP, Warner EJ. Halothane and children. The first quarter century. *Anesth Analg* 1984;63:838-40.
- 3 Lunn JN, Mushin WW. Mortality associated with anaesthesia. London: Nuffield Provincial Hospitals Trust, 1982.
- 4 Dienstag JL. Halothane hepatitis. Allergy or idiosyncrasy? *N Engl J Med* 1980;303:102-4.
- 5 Strunin JL. Hepatitis and halothane. *Br J Anaesth* 1976;48:1035-6.

AUTHORS' REPLY.—The main purpose of our paper was to provide objective evidence that halothane hepatitis can occur in children, and we are pleased that all your correspondents concede that this is the case. The prevalence of this complication may be learnt only if clinicians accept that this exists. The only way to determine the incidence of the problem is in a prospective study with long term follow up of children receiving halothane, as in many instances they will be discharged from hospital before the onset of the hepatitis. Our own experience suggests that halothane hepatitis in adults is underreported to the Committee on Safety of Medicines.¹

The *in vitro* techniques that we describe will confirm the diagnosis in 70% of cases. The antibody has been detected only in patients with halothane hepatitis.² In children, as in adults, we have been unable to detect halothane antibodies in those with fulminant hepatic failure from causes unrelated to halothane, in those who have had multiple exposures to halothane but have normal liver function values, or in those with other causes of liver disease. In addition, we have been unable to relate the titre of antibody to the severity of liver disease.²

Whether halothane hepatitis is indeed an antibody mediated disorder is currently being investigated by other centres.^{2,6} The position has advanced considerably since 1980, when the editorial to which Dr Wark refers was published. The specificity of the antibody to sensitised rabbit hepatocytes (not guinea pig, as stated by Dr Wark) has been confirmed by different techniques.² While we accept that the value of halothane antibodies in confirming halothane hepatitis has not been confirmed by other centres, it has not been refuted, and other authors have shown sensitisation to halothane related antigens in patients with halothane hepatitis.^{4,5}

Clearly, the risk of halothane hepatitis in a child or adult being considered for anaesthesia must be assessed against the risks of the clinical setting in which it is used and the risks of alternative procedures. All current evidence suggests that the risk of halothane hepatitis is increased by repeated exposure to halothane. We would support the

Committee on Safety of Medicines' recommendation that "A history of unexplained jaundice or pyrexia after exposure to halothane is an absolute contraindication to its future use in that patient."⁷

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- 1 Kenna JG, Neuberger J, Williams R. Specific antibodies to halothane induced liver antigens in halothane hepatitis. *Br J Anaesth* (in press).
- 2 Neuberger J, Kenna JG. Halothane hepatitis. *Clin Sci* 1987;72:263-70.
- 3 Callis AH, Brooks SD, Vaters SJ, et al. Evidence for a role of the immune system in the pathogenesis of halothane hepatitis. In: Roth SH, Miller KW, eds. *Molecular and cellular mechanisms of anaesthesia*. New York: Plenum Publishing Corporation, 1986: 443-53.
- 4 Nunez-Gornes SF, Marx JS, Motzsko C. Leucocyte migration inhibition in halothane induced hepatitis. *Clin Immunol Immunopathol* 1979;14:30-4.
- 5 Saito K, Kumagai K. Postoperative hepatitis induced by halothane exposures. *Tohoku J Exp Med* 1980;130:291-6.
- 6 Satoh H, Fukuda Y, Anderson DK, et al. Immunological studies on the mechanism of halothane induced hepatotoxicity. *J Pharmacol Exp Ther* 1985;233:857-62.
- 7 Committee on Safety of Medicines. *Current problems No 18*. London: Committee on Safety of Medicines, 1986.

ABC of AIDS: Neurological manifestations

SIR.—An extensive review of toxoplasmosis associated with the acquired immune deficiency syndrome (AIDS) has been prepared in our laboratory for future publication. Dr C A Carne (30 May, p 1399) mentions the difficulty of interpreting toxoplasma serology results derived from AIDS sufferers. It should be noted that current methods applied to the diagnosis of toxoplasma infection in association with AIDS are problematic, and there is a need for improved techniques.

Computed tomography findings are not specific to toxoplasma infection, while the use of brain biopsy remains contentious.^{1,2} Empirical trials of antitoxoplasma treatment may be complicated by severe drug reactions.³ Active cerebral toxoplasmosis in association with AIDS dictates long or even lifelong treatment.⁴ In AIDS victims cerebral toxoplasmosis usually results from an exacerbation of a chronic, previously latent infection. The Sabin-Feldman dye test result is normally positive but not considerably increased, and rising titres are rarely shown. Toxoplasma specific IgM is not detected in most cases of toxoplasmosis in patients with AIDS, reflecting reactivation rather than primary infection.⁴ There is evidence, however, that the use of a toxoplasma agglutination test may be of value in patients with AIDS, particularly when results are expressed as a ratio to the dye test titre.⁵ We are currently evaluating Western blot analysis of toxoplasma antigens recognised by human antibody, and studies using nucleic acid probes are in hand. Consequently, we would be grateful to receive clinical information and samples from patients with AIDS with suspected toxoplasmosis to help in developing improved diagnostic methods.

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- 1 Wong B, Gold JW, Brown AE, et al. Central nervous system toxoplasmosis in homosexual men and parenteral drug abusers. *Ann Intern Med* 1984;100:36-42.
- 2 Anonymous. Toxoplasmosis diagnosis and immunodeficiency. *Lancet* 1984;i:605-6.
- 3 Delaney P, Neeley S, Schwartz R. Fatal CNS toxoplasmosis in a homosexual man. *Neurology (NY)* 1983;33:926-7.

- 4 Navia BA, Petito CK, Gold JW, Cho ES, Jordan BD, Price RW. Cerebral toxoplasmosis complicating the acquired immune deficiency syndrome. *Ann Neurol* 1986;19:224-38.
- 5 Luft BJ, Brooks RG, Conley FK, McCabe RE, Remington JS. Toxoplasmic encephalitis in patients with acquired immune deficiency syndrome. *JAMA* 1984;257:913-7.

Not all travellers need immunoglobulin for hepatitis A

SIR.—We agree with the policy of selective screening before immunising for hepatitis A recommended by Drs Jonathan H Cossar and Daniel Reid (13 June, p 1503). Drs Cossar and Reid cite our figures for prevalence of antibodies to hepatitis A from 1980 in support of this policy.¹ In a survey in 1985 we found that the prevalence of hepatitis A antibody (HAVAB, Abbott Laboratories, Chicago) was still high, with 78% of those over 40 years of age showing evidence of past infection. The antibody prevalence increases steadily with age (table), and we therefore suggest that screening before immunisation may be cost effective in those individuals over 30 years of age. The younger person, on the other hand, would not benefit from screening, particularly if going abroad for a short stay, when a single dose of immunoglobulin (costing approximately £3.00) is indicated.

Prevalence of antibodies to hepatitis A in blood donors from west of Scotland

Age (years):	18-25	26-30	31-35	36-40	≥41
% Positive:	30	41	55	58	78

The incidence of cases of hepatitis A associated with travel has also increased. In 1986, 22 out of 76 (29%) cases of hepatitis A diagnosed at our laboratory were related to travel. Seventeen of these cases were in children or adolescents, all of whom were of Asian origin and had visited their home country. These cases included five family outbreaks. In two of the episodes serum samples were available from the parents, who were immune in both instances. All the adult patients were white. Our study highlights the fact that the children of immigrant families who have been born in this country but go back to their home country for a holiday are a particular group at risk, and we are worried that these children are not perceived to be at risk by their parents, who are invariably immune to hepatitis A. This should be a special target group for education and immunisation for hepatitis A before travel.

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- 1 Follett EAC, Barr A, Crawford RJ, Mitchell R. Viral hepatitis markers in blood donors and patients with a history of jaundice. *Lancet* 1980;ii:246-9.

Infantile apnoea and home monitoring

SIR.—Professor Hamish Simpson's statement (30 May, p 1367) that apnoea has never been substantiated as the major cause of the sudden infant death syndrome is hardly surprising as apnoea is a symptom, not a disease. As has been emphasised repeatedly,^{1,2} various pathological processes are probably implicated in the syndrome. Despite continuing uncertainties about which infants are most vulnerable and which provoking factors are operative at some point before death there will be cessation of airflow (apnoea)—central,