less critically manuscripts which are on the right side of the fence on the issue of passive smoking.)

Consideration of the first of these two biases led to a reduction in the estimated relative risk from 1·35 to 1·30 for the paper of Professor Wald and his colleagues but from 1·34 to 1·15 in the National Research Council report. This source of bias cannot fully account for the excess over unity of the relative risk, albeit the National Research Council report suggests that statistical significance would no longer obtain. And the possibility of other biases is noted.

The two survey studies make differing adjustments for exposure to passive smoking away from home. While Professor Wald and his colleagues make an upward adjustment of 18%, from a relative risk of 1·30 to 1·53, the National Research Council report makes an upward adjustment of only 8%, from 1·15 to 1·24.

For assessing statistical significance, this last adjustment is not relevant. It presupposes that passive smoking does increase risk; for if it did not the adjustment would not be needed. But relevance would attach if one wished to estimate the toll in lung cancer attributable to passive smoking.

The National Research Council report notes a study by Jarvis *et al* on biochemical markers of smoke absorption.[3] From that work one would have to judge that the claim of being a non-smoker was more frequently false than has been allowed for in the bias adjustments that have been made. Also, the data on cotinine concentrations in the plasma, saliva, and urine reported by Jarvis *et al* suggest that the relative risk associated with passive smoking would be quite limited, say of the order of 1·05. Passive smokers had, on average, cotinine values 0·5% of the way between the level for those not exposed to passive smoking and the level for active smokers. Assuming active smoking to have a relative risk of 10, added risk of 900%, the predicted relative risk for passive smoking would be 1·045.

It is interesting that the National Research Council report shows a predicted relative risk of 1·14 based on dosimetric considerations. The underlying assumption was that passive smoking had only 1% of the effect of active smoking. That 1% effect was then coupled with a relative risk of 15, added risk of 1400%, for active smoking.

In the event, whether the true relative risk is 1·05 or 1·14, it is unlikely that any epidemiological study has been, or can be, conducted which could permit establishing that the risk of lung cancer has been raised by passive smoking. Whether or not the risk is raised remains to be taken as a matter of faith according to one's choice.

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1 National Research Council Committee on Passive Smoking, Board on Environmental Studies and Toxicology. *Environmental tobacco smoke: measuring exposures and assessing health effects*. Washington, DC: National Academy Press, 1986.
2 Blot JB, Fraumeni JF Jr. Guest editorial. Passive smoking and lung cancer. *Journal of the National Cancer Institute* 1986;77: 993-1000.
3 Jarvis M, Tunstall-Pedoe H, Feyerabend C, Vesey C, Salloojee Y. Biochemical markers of smoke absorption and self reported exposure to passive smoking. *J Epidemiol Community Health* 1984;38:335-9.

Autologous blood transfusion

SIR,—In the wake of the recent blunder by the BMA it is doubly unfortunate that your leading article on autologous blood transfusion should contain factual errors which could fuel the fears of patients and doctors about transfusion quite unnecessarily.

Dr L A Kay states that non-A non-B hepatitis "often causes chronic active hepatitis or cirrhosis and develops in up to 10% of blood recipients in the United States." This is misleading, in that it implies that up to 10% of transfusion recipients will develop serious liver disease. Only one study of the long term sequelae of post-transfusion non-A non-B hepatitis has been reported.[1] Of the 50% of cases which became chronic, as evidenced by raised transaminase activities persisting for more than six months, 10-15% may be expected to show evidence of clinically important liver disease. The National Heart, Lung, and Blood Institute in the USA is so concerned about the lack of clinical data on this subject that it has just issued a request for research proposals to investigate the clinical course of post-transfusion non-A non-B hepatitis.

These figures are almost certainly an overestimate of the problem as they make no allowance for the proportion of recipients who die of their original disease (over 50% in most retrospective studies). Furthermore, the incidence of post-transfusion non-A non-B hepatitis is probably much lower in the UK than in the USA, having been found to be 2·4% in coronary bypass patients in the only recent study.[2] The true figure may well be even lower as groups at high risk of HIV infection have been excluded from donation.

Selective IgA deficiency occurs in around one in 700 of the population, not 7% as stated by your reviewer. Antibodies to IgA occur in up to 40% of these, but anaphylactic transfusion reactions due to IgE directed against IgA are very uncommon.[3]

No one in the blood transfusion service would wish to minimise the risks of transfusion, but it is important that decisions about alternative, and possibly expensive, strategies are based on accurate information. Autologous blood transfusion is an important option to evaluate, though there is evidence from practical experience elsewhere that it is likely to be applicable only to a small proportion of patients.[4] It will have no impact on the care of those patients who make the greatest demands on the transfusion service, such as those with marrow failure or major haemorrhage.

The Scottish National Blood Transfusion Service is currently developing a pilot programme to assess the effectiveness, applicability, and cost of such procedures.

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1 Alter HJ. Post-transfusion hepatitis: clinical features, risk and donor testing. In: Barker LF, Dodd RY, eds. *Infection immunity and blood transfusion*. New York: Alan R Liss Inc, 1985:47-61.
2 Collins JD, Bassendine MF, Codd AA, Collins A, Ferrer RE, James OFW. Prospective study of post-transfusion hepatitis after cardiac surgery in a British centre. *Br Med J* 1983;287: 1422-4.
3 Burks AW, Steele RW. Selective IgA deficiency. *Ann Allergy* 1986;57:3-8.
4 Kruskall MS, Glazer EE, Leonard SS, *et al*. Utilization and effectiveness of a hospital autologous pre-operative blood donor program. *Transfusion* 1986;26:335-40.

AUTHOR'S REPLY—It is most encouraging that the Scottish National Blood Transfusion Service recognises the importance of evaluating autologous blood transfusion. Its pessimism on the number of patients who will be eligible for the procedure is based on the experience of Kruskall,[1] which, as I mentioned in the article, is out of line with that of most workers.

I doubt whether most readers will misinterpret my statement on the long term sequelae of non-A non-B hepatitis. Drs Gillon and McClelland rightly point out that few largescale prospective studies of non-A non-B hepatitis have been carried out in Britain. They cite the low incidence of post-transfusion non-A non-B hepatitis after cardiac surgery in a single recent British study.[2] Unfortunately, of the 248 patients studied only 44 were regularly examined for their transaminase activities; the rest were tested only during their stay in hospital and at six months, so long incubation non-A non-B infection, which my be associated with intermittent raised transaminase values,[3] would have been missed. The authors themselves remark on their low incidence of non-A non-B hepatitis compared with similar studies in Europe using volunteer blood, which showed an incidence of 18-19%.[4,5] In fact it is no more than the 2·2% rate in hospital patients who have not received a transfusion.[3,4]

If careful prospective studies were done in Britain we should probably find a sharp geographical variation in the incidence of post-transfusion non-A non-B hepatitis, depending on the socioeconomic state of the community, as the incidence varies in the United States of America from 4% to 17%.[6,7]

Until further long term studies are done we cannot be sure how many patients with non-A non-B hepatitis develop chronic liver disease, but up to 10% is the usually quoted estimate. Drs Gillon and McClelland assert that even this need cause little concern, since half of all transfused patients die of their original disease. My concern is for healthy patients undergoing elective surgery, who are highly unlikely to die before chronic complications of hepatitis infection occur. The number of people at risk because of IgA deficiency is indeed 1 in 700, not 7 in 100, and I apologise for missing the error.

The blood transfusion service developed out of the need to treat battlefield casualties during the second world war, and even today the injured and those with marrow failure must rely on donor blood. But why should those with healthy bone marrows accept any additional risks from blood transfusion when they undergo elective surgery?

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1 Kruskall MS, Glazer EE, Leonard SS, *et al*. Utilisation and effectiveness of a hospital autologous pre-operative blood donor program. *Transfusion* 1986;26:335-40.
2 Collins JD, Bassendine MF, Codd AA, *et al*. Prospective study of post transfusion hepatitis after cardiac surgery in a British centre. *Br Med J* 1983;287:1422-4.
3 Aach RD, Kahn RA. Post-transfusion hepatitis: current perspectives. *Ann Intern Med* 1980;92:539-46.
4 Tremolada F, Chiappetta F, Noventa F, *et al*. Prospective study of post transfusion in cardiac surgery patients. *Vox Sang* 1983;44:25-30.
5 Grillner L, Bergdahl S, Jyrala A. Non A, non B hepatitis after open heart surgery in Sweden. *Scand J Infect Dis* 1982;14: 171-5.
6 Aach RD, Lander JJ, Sherman LA, *et al*. Transfusion-transmitted viruses: interim analysis of hepatitis among transfused and nontransfused patients. In: Vyas GH, Cohen SN, Schmid R, eds. *Viral hepatitis*. Philadelphia: Franklin Institute, 1978: 383-96.
7 Aach RD, Szmuness W, Mosley JW, *et al*. Serum alanine aminotransferase of donors in relation to risk of non A, non B hepatitis in recipients. *N Engl J Med* 1981;304:989-94.

SIR,—Dr L A Kay's foray into the arena of autologous transfusion (17 January, p 137) has stirred up a cloud of dust that is likely to obscure recognition of the salient facts. A decision to advocate autologous transfusion in place of the use of voluntary donor blood should be made on the basis of the established levels of risk from routine transfusion and not as a result of fears exaggerated by the media.

Although there are several reasons for supporting the use of autologous blood, the threat of the acquired immune deficiency syndrome (AIDS) is uppermost in people's minds. The risk of