patients who have undergone splenectomy.[1] People who receive this advice may ask about their baseline risk of dangerous sepsis, how this will be reduced by taking medicine every day, and other options for prevention.

The risk of serious infection after splenectomy varies according to indication, age, and underlying disease; sepsis occurs despite prophylaxis, and compliance is often poor.[2 3] How does the committee recommend we interpret these facts for our patients? The committee quotes work by Cummins *et al*[4]; our understanding of this work is that it focuses exclusively on children with sickle cell disease and concludes that less than half of them took prophylaxis. The committee also quotes literature stating that the incidence of severe sepsis late after splenectomy is so rare as to make the efficacy of prophylactic antibiotics impossible to evaluate statistically.[2] Furthermore, the committee offers no evidence to contradict an established recommendation that for lower risk groups (such as adults who have lost their spleens through trauma) the preferred (and less burdensome) management is early recognition and treatment with a ready supply of amoxycillin.[2 3]

In this age of evidence based medicine and interest in individualised relative risks, experts who interpret research and set guidelines should focus on what is likely to be most useful to the clinicians and patients who are expected to implement the guidelines.[5] Is the committee aware of good evidence that the balance of risk is better in patients who take antibiotics daily than in those in whom an approach involving early recognition of infection and early treatment is adopted? If this recommendation is based mainly on expert opinion, is there any way of establishing the relative risks for each approach, given the small numbers and rare events?

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1 Working Party of the British Committee for Standards in Haematology Clinical Haematology Task Force. Guidelines for the prevention and treatment of infection in patients with an absent or dysfunctional spleen. *BMJ* 1996;312:430-4. (17 February.)
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Guidelines do not discuss resistance to antibiotics among pneumococci

EDITOR,—The guidelines on preventing and treating infection in patients with an absent or dysfunctional spleen do not discuss the impact of current changes in patterns of resistance to antibiotics among pneumococci.[1] The few and perfunctory phrases mentioning the problem are supported by a seven year old reference. Since then, resistance to penicillin and other antibiotics has steadily increased, with large variations among countries. The most recent figures for England and Wales (for 1994) from the Communicable Disease Surveillance Centre show that 2.5% of strains of pneumococci are resistant to penicillin (about half of these are highly resistant) and 11.2% are resistant to erythromycin.[2] A recent review shows the widespread nature of the problem, with rates of resistance to penicillin as high as 36%.[3]

The association between a high rate of resistance to penicillin and failure of treatment is established only for meningitis and otitis media, but whether penicillin can be regarded as suitable treatment for life threatening pneumococcal septicaemia caused by highly resistant strains remains uncertain. I would be reluctant to accept penicillin as the agent of first choice in such a situation. The question is given greater urgency because patients who have previously received penicillin, such as those receiving long term prophylaxis, are more likely to carry a resistant strain.[3]

The link between use and resistance is even more closely established for erythromycin, and resistant strains of pneumococci are commonly found in populations exposed to this antibiotic. For this reason I question the wisdom of long term prophylaxis with erythromycin in patients who are allergic to penicillin. If, however, such a policy were to be followed the suggestion that the dose of erythromycin should be increased in the event of febrile illness is dangerous, since these patients are the ones most likely to carry a resistant strain. Pneumococcal septicaemia associated with asplenia can take a devastatingly rapid course, and there are no second chances in the choice of antibiotic.

The working party that drew up the guidelines makes a particular point of its review of the literature and methods of developing the guidelines. An additional, more basic approach, such as telephoning a few people familiar with these problems (infectious diseases physicians, paediatricians, and the relevant department at the Communicable Disease Surveillance Centre), would have prevented such egregious errors and omissions.

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Working party's reply

EDITOR,—As M R Workman and colleagues state, the high susceptibility to salmonella infection among patients with sickle cell disease remains unexplained. Because there is no evidence to link this with hyposplenism the guidelines do not refer to the problem.

Our guidelines are part of the drive to increase the education of health professionals, and A A Palejwala and colleagues' study indicates that this need is greatest in general practice. Palejwala and colleagues and Helen Howie and Ann F Bisset point out the advantages of computerised data systems. We are aware of several regional registers and acknowledge the usefulness of data linkage.

G C Kassianos requests information on the level of antibody that provides protection against pneumococcal infection. This level is not known. The need for reimmunisation and the timing of the next dose may be determined by measuring antibodies to pneumococcus before and one month after immunisation and then at three and five years, especially in patients at highest risk. Reactions to the vaccine can be minimised by avoiding reimmunisation of patients with high circulating levels of antibody. Patients with poor or absent antibody responses should be targeted for (lifelong) antibiotic prophylaxis.

Chris Butler and Paul Kinnersley question the recommendation that prophylactic antibiotics (oral phenoxymethylpenicillin or an alternative) should be offered life long. We agree that most of the published evidence suggesting a protective effect of phenoxymethylpenicillin refers to children with sickle cell disease.[1] Although overwhelming infection in otherwise healthy asplenic adults is rare, *Streptococcus pneumoniae* is the most likely pathogen, and in Britain it remains highly sensitive to penicillin. We recognise that many adults may not accept lifelong antibiotic prophylaxis. Our additional suggestion that patients should keep amoxycillin or a macrolide antibiotic at home to be used at the first sign of possible infection could well apply to such patients. To establish the relative risks and benefits of each approach it might be possible to recruit patients into a study, matching for age, sex, and reasons for and time since splenectomy. A national study is more likely to provide meaningful results but would depend on the creation of regional and national registers.

H P Lambert takes us to task for dealing superficially with the problem of resistance to antibiotics. Because of the small number of patients taking prophylaxis the impact of these recommendations on patterns of resistance is likely to be low. Resistance to penicillin and erythromycin is certainly a cause for concern. Because of this risk the guidelines recommend cefotaxime or ceftriaxone as empirical treatment for patients taking antibiotic prophylaxis. Patients who are allergic to both penicillin and cephalosporins may be given chloramphenicol.

Lambert should note that the working party included a medical microbiologist and a paediatrician, and advice was sought from the Communicable Disease Surveillance Centre and an infectious disease physician. The guidelines took over two years to prepare because of the extensive consultation that took place and the need to negotiate the *BMJ*'s review system.

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Testing to check success of treatment to eradicate *H pylori*

Patients' wellbeing should not be risked for marginal cost savings

EDITOR,—Perminder S Phull and colleagues suggest that dyspeptic symptoms are good at predicting a successful outcome of treatment to eradicate *Helicobacter pylori* in patients with duodenal ulcer.[1] Other published data, however, have shown that recurrent dyspepsia in patients with duodenal ulcer who are cured of *H pylori* infection is not as rare as the authors' results suggest.[2 3] In one study, in which 207 patients with ulcer were followed up for a median of 250 days after treatment, 31% of those who were