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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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

negative for *H pylori* admitted to flare ups of pain.² The fact that such symptoms probably resulted from coexistent reflux or functional bowel disease rather than recurrence of ulcer is not important in this context.

Deferring retesting of patients' *H pylori* status until symptomatic relapse has a superficial economic appeal, as the urea breath test is relatively expensive. The savings made through not retesting patients who remain asymptomatic will, however, be partly offset by the extra costs of treating patients who suffer a relapse. Even simple relapse will incur added costs (drugs, consultations, loss of work) before repeat testing is arranged, and the costs of just one complication would finance many breath tests. In a 12 month follow up study, among 66 patients with ulcer who remained positive for *H pylori* after eradication treatment two bled from an ulcer and two were admitted to hospital with abdominal pain.³

Excluding patients with a history of complicated ulcer and advising patients to reconsult if symptoms recur will not remove the possibility of patients presenting with severe symptoms or complications. Sonnenberg and Townsend estimated the costs of treating duodenal ulcer with alternative management strategies, including treatment to eradicate *H pylori* both with and without subsequent testing for *H pylori*.⁴ When use of a post-treatment test costing up to \$400 was assumed, routine verification of eradication seemed less expensive than awaiting symptomatic recurrence and resulted in patients spending less time with active ulceration.

Evidence is accumulating to support a change from Schwartz's dictum of "no acid, no ulcer" to "no *H pylori*, no ulcer." But what about "no pain, no *H pylori*?" We urge caution in the implementation of a symptom based assessment of *H pylori* status after treatment, doubting both its reliability and its cost effectiveness. It seems harsh to require some patients to suffer a recurrence of symptoms before establishing whether the treatment has been effective. The wider provision of *H pylori* testing services should be a priority; patients' wellbeing should not be risked for marginal cost savings.

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Routine retesting is necessary

EDITOR,—The decision whether to retest for *Helicobacter pylori* after a course of eradication treatment in peptic ulcer disease depends on the likely outcome. If one expects that the organism will nearly always be killed by a course of such treatment and that there will be few other dyspeptic symptoms not due to ulcer, then arguing against routine retesting makes sense. Perminder S Phull and colleagues adopt just such an argument on the basis of finding a 2.5% prevalence of symptoms of reflux and no other dyspepsia in their patients from whom *H pylori* had been eradicated.¹ This low figure for continuing symptoms is, however, at odds with figures reported elsewhere and suggests that the study population may have been preselected on

the basis of having "pure" duodenal ulcer disease. We found that in 140 patients with peptic ulcer whose infection was successfully treated 39% reported heartburn, 25% reported symptoms of the irritable bowel syndrome, and 22% had a further consultation with the general practitioner during a median follow up of 249 days.² Powell et al found that 12-18% of patients with peptic ulcer used H₂ receptor antagonists in each three month period after successful eradication of *H pylori*.³

In practice, regimens to eradicate *H pylori* achieve a success rate of 85% at most. The 15-20% of patients in whom the treatment fails are highly likely to experience recurrent symptoms and to present again, and our figures suggest that up to a third of patients in whom eradication is successful will eventually present again. In other words, around a third of all patients given eradication treatment for peptic ulcer disease can be expected to visit their doctor again with dyspepsia. Routine retesting after eradication treatment enables the clinician to provide reassurance for those in whom it has been successful if they have recurrent dyspepsia and to prescribe repeat eradication treatment in advance of clinical relapse in those in whom it has failed; in addition, routine retesting may of itself reduce reconsultation rates. Routine retesting remains our practice.

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Screening for diabetic retinopathy

EDITOR,—J M Mason and colleagues state that the performance of high street optometrists in the Department of Health's study of screening for diabetic retinopathy was poorer than that of general practitioners and that this is surprising.¹ A tabulation that I have done of the results from that and other studies shows that the rate of detection of sight threatening diabetic retinopathy by high street optometrists and general practitioners when they use direct ophthalmoscopy alone is similar: the rate for general practitioners was 52% in one study and 55% in another, and that for optometrists 48%—that is, both groups miss about half of the cases.² As other studies in the tabulation show that even ophthalmologists, when allowed only direct ophthalmoscopy, have detection rates of only 64% and 65%, the main problem is shown to be not with the screeners but with the method used—direct ophthalmoscopy.

Mason and colleagues refer to recent work showing that specialist optometrists detect 71% of cases of sight threatening diabetic retinopathy with ophthalmoscopy, with this figure rising to 100% when photography is added. They erroneously reference a paper by Gatling et al as the source of these data. In fact, the data were collected in my department.³ The optometrist, who had a (relatively good) detection rate when using ophthalmoscopy of 71%, was highly experienced, specialised in diabetic retinopathy, and had been screening large numbers of diabetic patients in

the hospital diabetic clinic for many years. This cannot be extrapolated to the mass of high street optometrists using ophthalmoscopy alone.

Mason and colleagues are also concerned about the cost of adding photography, but is it that great? Once the patient is in front of the screener and has had his or her visual acuity measured and pupils dilated, taking photographs results in a minimal additional cost. In my department we estimate that our camera has undertaken of the order of 10 000 eye screenings in the past five years, and it is still going strong. The cost of the camera is well under £1 per patient and falling all the time. We use medical photographers of medical technical officer grade 2 at a cost of less than £1 per patient screened, but this cost is obviated if the ophthalmologist does the photography.⁴ Polaroid photographs are about £1 an eye, and the instant digitised images that will probably characterise the photography of the future not only seem to provide higher detection rates⁵ but remove the cost of the Polaroid photographs.

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The melanoma epidemic

Excess exposure to ultraviolet light is established as major risk factor

EDITOR,—Jonathan L Rees confuses histological nomenclature of early malignant melanoma and the relation between the risk of melanoma and exposure to the sun.¹ We need to separate the steadily increasing incidence of melanoma in all countries over the past 40 years from reported short term dramatic increases in localised areas. The short term increases are usually associated with increased awareness resulting in attention being drawn to melanomas that may have started to develop 10-20 years previously. The long term increase, however, is not an artefact and is causally related to exposure to the sun.²

The fact that pathologists now discuss the exact criteria for in situ melanoma, the radial growth phase, and early invasion is good news, since 10 years ago they were diagnosing thick tumours with a poor prognosis. What cannot be known is the natural course of early melanomas or those in the radial growth phase had they not been excised. A proportion would probably have progressed to the vertical growth phase with full capacity for metastatic spread.

Rees's arguments against exposure to the sun being a factor in the aetiology of melanoma are not original. It is well recognised that primary melanoma may occur on a covered site and that a high total lifetime exposure to the sun does not equate with an increased risk of melanoma. One of us and a colleague, however, have shown clearly that, per unit area of epidermis, the male ear (a site that has considerable exposure to the sun) has the highest incidence of melanoma of any part of the body.³ In addition, patients with melanoma have a significant excess of solar elas-