requesting permission for HIV antibody testing some life assurance companies rightly ask proposers to nominate their general practitioner or another named doctor who should be informed in total confidence in the event of a positive result. This doctor need not be the one who obtained the sample, who may be less well known to the proposer and not necessarily in a position to offer long term support. This makes the procedure more personalised and acceptable to the proposer.

My life assurance society does not at present use saliva tests for HIV antibody testing so I have not had to delete references to false positive results in policy holders' documents. In the less likely event of a false positive result of a blood test I would insist that the subsequent negative result was given due prominence. These documents remain strictly confidential to the underwriting team.

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- 1 Morris DF. False positive salivary HIV test. *BMJ* 1992;305:834. (3 October.)
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EDITOR,—We endorse Delia F Morris's letter about salivary testing for antibody to HIV.¹ It would be interesting to know if the laboratory concerned was in the private sector since the quality control measures mentioned would already have been implemented in NHS laboratories.

It has also been brought to our attention that salivary testing is being suggested for use in screening people in custodial care (prisons and young offenders' institutions). It has been emphasised that, at the moment, salivary tests are suitable for anonymous surveys of seroprevalence in various populations but should not be considered to be an alternative to testing serum in named patients. We believe that it would be unfortunate if salivary testing received adverse publicity since it is potentially very useful; a foolproof method of obtaining a reliable sample for testing has yet to be identified, described, and assessed.

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Cycle helmets

EDITOR,—The most surprising aspect of the recent debate on cycle helmets is how widely the crucial points were missed.¹⁻³ There is no logical argument against wearing a helmet approved by the American National Standards Institute; it will do no harm and may do some good.

Doctors are whistling in the wind if they believe that public health strategies, schemes, and programmes will change the public's attitude. The considerable increase in helmet wearing over the past five years is due simply to changed perception of its fashion status. For most people the real reason that they do not wear a helmet is that they think that they look "uncool" or stupid (depending on peer group) riding around with a lump of

polystyrene and acrylic on their head. Those of us who are cyclists and doctors and believe in wearing a helmet as one of several self protective techniques must be grateful that manufacturers and retailers clearly understand the group and individual psychology of the disparate population of child, commuter, and recreational and sports cyclists. That they understand their market better than we understand a potential patient population should be a salutary lesson.

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- 2 Illingworth C. Do cycle helmets prevent serious head injury? The argument for helmets. *BMJ* 1992;305:882-3. (10 October.)
- 3 Trippie HR. Helmets for pedal cyclists. *BMJ* 1992;305:843-4. (10 October.)

EDITOR,—It is interesting how personal experience modifies one's views. I refer to use of cycle helmets.¹⁻³ A cyclist for well over 50 years, I used to share the view expressed by Mark McCarthy.²

Some years ago I was unconscious for a short time, with amnesia, after falling from a cycle and sustained head trauma requiring a fair amount of suturing. Although I quickly recovered from the injuries, I was left with a postural vertigo that lasted for over two years. Cynthia Illingworth states that "head injuries which at first seem to be minor may later prove more serious or lead to sequelae." My experience makes me wonder just how much hidden morbidity there is after head injuries incurred during cycling. In advocating the use of helmets doctors should emphasise their protection against morbidity as much as the life-saving aspects.

Modern helmets are light, well ventilated, and easy to wear. After a week or two of regular use they are on a par with car seat belts because you soon forget that you are wearing one. More use of helmets by adults would have a favourable influence on the more vulnerable young. I am not sure that legislation would be the answer but believe that doctors should recommend use of helmets as part of advice on lifestyle.

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- 2 McCarthy M. Do helmets prevent serious head injury? *BMJ* 1992;305:881-2.
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Eradicating *Helicobacter pylori* in patients with duodenal ulcer

EDITOR,—We should like to comment on Shorland W Hosking and colleagues' paper reporting short term treatment for *Helicobacter pylori*.¹

The prevalence of metronidazole resistant *H pylori* before treatment and patients' compliance are major determinants of the eradication rate of treatments including metronidazole.^{2,3} Although we initially reported an overall eradication rate of 87%, subsequent subgroup analysis disclosed a 93% eradication rate for strains sensitive to metronidazole but only 19% for resistant strains.¹ Therefore it would be valuable to see a similar analysis with respect to Hosking and colleagues' data, because only then can the true effectiveness of their regimen be assessed. The importance of metronidazole resistance in determining the outcome of treatment and the emergence of multi-resistant strains of *H pylori* when treatments fail should not be underestimated; this problem makes pretreatment culture and in vitro antibiotic

sensitivity testing important for clinical trials and routine management.

Assessment of compliance is mentioned by the authors, but unfortunately no details of how this was done are given. Informal inquiry at the end of treatment is not adequate.

The usual definition of eradication (confusingly referred to as clearance in table II of their paper) requires all tests to be negative at least one month after the end of treatment. In studies relying solely on culture (for which five days on non-selective media is not sufficient) false negative results may have occurred.⁴ It is also not clear how patients with false positive results on urease tests and the one patient who subsequently regrew *H pylori* were classified. Thus it is possible, on an intention to treat analysis, that the eradication rate was only 64/78 (82%) and not 70/74 (95%).

The inherent problems of biopsy based methods for detecting *H pylori* are compounded in patients given omeprazole (incidentally, the dose used by Hosking and colleagues in their study is not stated) if biopsy specimens from the gastric corpus are not analysed.^{5,6} These problems could have been avoided by using a urea breath test to detect eradication. The breath test accurately detects the low levels of recolonisation often present after failed treatments and also provides the clinical "gold standard" alluded to by the authors in their discussion.^{5,6}

Finally, the design of the study may suggest that rates of duodenal ulcer healing and recurrence were additional end points. Graham *et al* have already shown, however, that ranitidine and a similar regimen against *H pylori* significantly increases the healing rate of duodenal ulcers,⁷ and we and others have shown that short term anti-helicobacter treatment without acid inhibitors is effective primary ulcer healing treatment for bleeding or non-bleeding ulcers.^{8,9}

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- 1 Hosking SW, Ling TKW, Yung MY, Cheng A, Chung SCS, Leung JWC, *et al*. Randomised controlled trial of short term treatment to eradicate *Helicobacter pylori* in patients with duodenal ulcer. *BMJ* 1992;305:502-4. (29 August.)
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AUTHORS' REPLY,—Resistance of *Helicobacter pylori* to metronidazole was not measured in our trial. Logan *et al*'s data showed that 19% of resistant strains were eradicated by their regimen

containing metronidazole.¹ This indicates that metronidazole resistance on its own is of limited value in evaluating the outcome of treatment.

Compliance was measured by counting the number of tablets remaining at the time of the follow up endoscopy.

The definition of "eradication" of *H pylori* varies from paper to paper. We followed the Sydney Working Party's report on *H pylori*, which defines eradication as failure to culture the organism at least one month after treatment by using a previously validated technique.² Our cultures were performed with media and techniques described in the same report, except that we omitted antibiotics in the culture medium. This was because our own validation studies showed no difference in results when such selective media were used. A urease test was included to evaluate its usefulness in measuring eradication against our "gold standard," which was culture. Thus our eradication rate is as reported—95%.

We agree with Logan *et al* that the modified urea breath test has certain advantages, but the necessary equipment is not available in many hospitals. Our data suggest that omeprazole alone is ineffective in eradicating *H pylori*. Previous reports on redistribution of *H pylori* within the stomach after omeprazole are difficult to interpret as biopsy specimens were taken immediately on stopping omeprazole rather than four weeks later.³

Healing ulcers by using anti-helicobacter treatment alone without acid suppression is an interesting idea. A randomised trial studying this possibility is currently in progress in our hospital.

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- 1 Logan RPH, Gummert PA, Misiewicz JJ, Karim QN, Walker MM, Baron JH. A one week eradication regime for Helicobacter pylori. *Lancet* 1991;338:1249-52.
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Postneonatal mortality in England, Wales, and Sweden

EDITOR,—Publication of D A Leon and colleagues' paper¹ gives me the opportunity to update the figures for the postneonatal cot death rate for England and Wales to include those for 1991.² The table shows that since 1988 the rate has continued to fall both nationally and regionally—nationally by 35% in three years and regionally by from 18% to 68%, with one region recording no change. The

Cot death rates (ICD 780-798, sole cause)/1000 live births in England and Wales, 1988-91

	1988	1989	1990	1991	% Fall 1988-91
England and Wales	2.0	1.7	1.5	1.3	35
Region:					
Northern	1.7	1.8	1.8	1.4	18
Yorkshire	2.1	2.4	1.8	1.6	24
Trent	1.6	1.3	1.1	1.3	19
East Anglia	1.6	1.4	1.9	1.2	25
North West Thames	1.8	1.8	1.2	0.8	55
North East Thames	1.7	1.3	1.1	0.6	65
South East Thames	2.5	1.5	1.6	1.4	44
South West Thames	2.2	1.7	1.3	0.7	68
Wessex	2.5	2.3	1.7	1.3	48
Oxford	2.0	1.3	1.3	1.1	45
South Western	2.8	2.2	1.6	1.6	43
West Midlands	1.8	1.7	1.6	1.2	33
Mersey	1.3	1.6	1.3	1.3	0
North Western	2.5	1.8	1.8	1.9	24
Wales	1.7	1.6	1.4	1.2	29

rate in three of the Thames regions fell by 55%, 65%, and 68% over the three years; this was accompanied by a fall in total postneonatal mortality to 1.8, 2.0, and 2.3/1000 live births respectively. These figures are around the Swedish level. These reductions occurred before the Department of Health officially advised the public at the end of 1991 that babies should sleep supine and not be allowed to overheat.¹ We will have to wait another year before assessing the results of this.

Swedish (and Japanese) total postneonatal mortality for many years has been half that for England and Wales (about 2.0-4.0/1000 live births). The difference is made up by the British rate for cot death—2.0—compared with the Swedish rate of almost nil. Abolition of cot deaths would equalise the figures. England and Wales now seem to be on the way to this objective. If three regions can have a cot death rate under 1.0/1000 arguably others—and perhaps all—can do the same. And if it can be accomplished in the south of England it should in time also be accomplished in the north.¹

Presumably, however, even after this equality has been achieved there will still be social inequality, as has been shown in Sweden. Perhaps it is after all part of the human estate rather than something created by people.

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Transfusing *Yersinia enterocolitica*

EDITOR,—Concern for the safety of stored blood in relation to infection with *Yersinia enterocolitica* has recently been discussed.¹ We have recently encountered a problem with bacterial contamination of bone marrow harvested from a patient with non-Hodgkin's lymphoma. Samples of bone marrow are routinely sent to the bacteriology laboratory after inoculation into standard blood culture bottles. It is not unusual for these blood cultures to yield Gram positive bacteria after incubation, but the significance of such growth is questionable and is often considered to represent contamination with skin flora during collection. In this case, however, four blood culture bottles seeded with bone marrow all yielded a heavy pure growth of *Staphylococcus epidermidis* within 24 hours of incubation. The same bacterial strain was also recovered from the tip of a Hickman line removed from the patient at the time of the bone marrow harvest and was the presumed source of infection. Bone marrow is normally stored in liquid nitrogen after addition of the cryopreservative dimethylsulphoxide (DMSO, 10% vol/vol) and is then thawed out at the bedside before reinfusion. We have recommended that if this particular bone marrow is required then antibiotic cover must be given.

This recommendation stems from the results of our in vitro experiments to determine the survival of bacteria in the presence of DMSO. Five strains each of seven staphylococcal species, including *S epidermidis* and *S aureus*, were inoculated into nutrient broth containing 10% DMSO. All 35 strains grew after incubation aerobically at 37°C for 18 hours. Five *S epidermidis*, one *S aureus*, one *Escherichia coli*, and one *Pseudomonas aeruginosa*

were also seeded into pooled human serum containing 10% DMSO. All bacterial strains survived, with no reduction in viable counts, during incubation in air with 5% carbon dioxide at 37°C for 48 hours. Furthermore, one strain of each species survived freezing at -20°C in serum with 10% DMSO for two weeks, although there was a threefold logarithmic reduction in the numbers of viable Gram negative bacteria.

The addition of DMSO to harvested bone marrow is likely therefore to protect not only mammalian cells but also any bacteria present during storage. Indeed, others have used this cryoprotectant during the storage of bacterial strains.² As harvested bone marrow is thawed only immediately before reinfusion (to minimise the detrimental effects of freeze-thawing), there is no way of determining whether any viable bacteria are also being transfused. The relatively large bacterial load present in the bone marrow in this case could potentially produce serious sepsis after reinfusion. We recommend that, where there is evidence of contamination of bone marrow, antibiotics should be given to prevent infection when the marrow is returned, as bacteria are likely to survive during storage.

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EDITOR,—We agree with Michael Prentice's conclusions regarding transfusion of contaminated blood.¹ Until recently few post-transfusion incidents due to bacterial contamination had been reported in Britain.² Notification of two cases associated with *Yersinia enterocolitica*³ (J Smillie and F Ala, annual meeting of British Blood Transfusion Society, Nottingham, 1991) prompted the Advisory Committee on Transfusion Transmitted Diseases to instigate a survey of all regional transfusion centres to investigate the true nature of the problem. Questionnaires were sent out to cover 1986-90, and replies were received from 18 centres.

Twelve confirmed reactions due to bacterial contamination were notified, of which nine were associated with the issue of 11 million red cell components and three with 2.8 million platelet concentrates; in addition, one unit of red cells was discarded before being issued and was found to be contaminated. These figures suggest a maximum risk factor of about 1:1 000 000. A further three incidents were notified in 1991, in

Confirmed bacterial reactions to transfusions and other contaminations notified

Organism	Component	Age of component (days)	Patient died
<i>Yersinia enterocolitica</i>	Platelets	4	No
	Red cells	25	No
	Red cells	29	Yes
	Red cells	16	Yes
	Red cells	28	Yes
<i>Pseudomonas fluorescens</i>	Whole blood	32	Yes
	Red cells	26	Yes
	Red cells	22	No
	Red cells	25	Component not transfused
<i>Pseudocina</i>	Red cells	<10	Yes
	Whole blood	20	Yes
	Red cells	18	No
<i>Achromobacter xylosoxidans</i>	Red cells	29	Yes
<i>Erwinia herbicola</i>	Platelets	3	No
<i>Staphylococcus</i>	Red cells	4	No
<i>Serratia marcescens</i>	Platelets	3-5	Yes