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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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.³ 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.⁴

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>Pseudococina</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

which *Y enterocolitica* was inoculated. The table shows the organisms involved, the age of the components at the time of transfusion, and the outcome of the reaction. The most common contaminants were pseudomonads (nine cases) and yersinia (five). The mortality was considerable. Since its discovery in 1982¹ as a potential hazard in transfusion practice, fewer than 50 transfusion reactions due to yersinia have been reported worldwide.

The survey also highlighted the probability that transfusions are underinvestigated bacteriologically and underreported in Britain, as in other countries. Only four centres routinely investigated bacteriologically all adverse reactions notified. Most centres devolved this responsibility to local hospital blood banks or bacteriology laboratories.

The Advisory Committee on Transfusion Transmitted Diseases is producing guidelines on investigating and reporting such adverse reactions.

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Complementary medicine needs critical evaluation

EDITOR,—Patrick C Pietroni's article on the relation between general practice and complementary medicine omits any evidence justifying the use of alternative treatments.¹ In appealing to interest among general practitioners and popularity among patients to justify complementary treatments he implies that the rationale for using such treatments is fashion rather than objective evidence. At a time when conventional treatments are evaluated with ever increasing scrutiny it seems ironic that alternative medicine—for which the evidence is more elusive—can be accepted uncritically. Surely the economic limitations imposed on general practitioners and their primary health care team require managers to prefer treatments that work rather than appeal to irrational sensibilities.

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- 1 Pietroni PC. Beyond the boundaries: relationship between general practice and complementary medicine. *BMJ* 1992; 305:564-6. (5 September.)

Wernicke's encephalopathy and hyperemesis gravidarum

EDITOR,—The case of Wernicke's encephalopathy in hyperemesis gravidarum reported by P S Bergin and P Harvey¹ outlines an important but forgotten lesson from the past. Indeed, Sheehan indicated a mortality of 35% in his report in 1939.² I report another case that included optic papillitis.

A 31 year old, previously well woman with two successful previous pregnancies presented at 13 weeks with a six week history of worsening hyperemesis. Despite intravenous fluids and rectal emetics she continued with intractable vomiting for a further three weeks. Her α fetoprotein

concentration was raised at 16 weeks; fetal death was confirmed by ultrasonography. She underwent a prostaglandin induced abortion.

Two days later she woke with poor central vision (light only in the left and 6/18 in the right eye) and ataxia. Clinical examination showed bilateral sustained ankle clonus with extensor plantars, horizontal nystagmus, and swelling and haemorrhages in both optic discs. She received intravenous Parentovite and improved dramatically over the following five days with resolution of her visual impairment and ataxia. By six weeks she had no abnormal clinical findings.

Optic papillitis is rarely associated with Wernicke's encephalopathy, having an incidence of less than 5%.³ The usual fundal signs are retinal haemorrhages. Indeed, this poorly known feature has previously delayed the diagnosis.⁴ This sign should not obscure or delay the diagnosis.

I agree that oral thiamine supplements are urgently required by any patient with hyperemesis gravidarum, but in all 10 cases of Wernicke's encephalopathy reported in English in the past decade the vomiting had continued for one month and usually occurred after instigation of intravenous fluids. It is also important to remember that Wernicke's encephalopathy can occur whenever there is poor nutrition, including in anorexia nervosa⁵ and hunger strikes.⁶

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Value of Dundee coronary risk-disk

EDITOR,—Correspondence on the value of the Dundee coronary risk disk has been more concerned with theoretical validity than with usefulness,^{1,2} but, as I wrote a year ago, "validity is a matter for argument, but the acid test is whether the Dundee coronary risk-disk is found useful."³ It is, and over 4000 disks have been dispatched since then. The disk is used not only to motivate patients in general practice and occupational medicine but to audit the performance of cardiovascular risk factor clinics and to meet the local targets concerning risk factors and coronary prevention in the *Health of the Nation* initiative.⁴ At the time of my earlier response⁵ to the Oxford critique of the disk,⁶ results of our survey of users were incomplete; we are now past the two thirds response rate. The answer to the claim that the Dundee score is preferable to the Dundee (bus queue) rank is that users of the rank, or the rank and score together, outnumber users of the score alone, are the heavier users, are more likely to show the result to their patients, are more positive about the motivational value of the disk, and recommend the disk more strongly to other users.

Limiting the disk to three modifiable risk factors was a deliberate compromise that has been justified by the comments we have received. Most of the users were satisfied; of those who were not, some thought that not enough factors were included and others that a scoring system based on three factors was too complicated.

Mortality from coronary heart disease by sex in England and Wales 1990

Allan St J Dixon says that the system is flawed⁷ because it does not include the menopause as a risk factor, and he attacks comments attributed to me at a conference.⁸ Deaths from coronary heart disease multiply with age in both sexes; a constant multiple would produce a straight line on a logarithmic plot. The natural menopause occurs close to age 50, and few women stop menstruating more than a few years either side of this. If the menopause was associated with the withdrawal of a protective factor, as is widely claimed, death rates in women should rebound upwards, showing a concave plot inflecting upwards around 50. In fact, the plot is smooth and convex (figure); it is the curve in men that bends over more with age, or, as I stated in my talk, "it's the men wot wobble, not the women." Similar curves apply to other years and other countries, and it is not just national statistics that challenge the myth regarding the menopause. The nurses' health study, of 121 700 American women, showed no evidence of increased risk with a natural menopause.⁹ The deterioration in some coronary risk factors at the menopause, which is alleged to drive the presumptive acceleration in coronary risk, must therefore be compensated by improvements in others, and many prevailing assumptions are too simplistic.¹⁰

None of this is to deny a potential benefit from hormone replacement therapy, but again the situation is not as simple as Dixon suggests. If his verdict of unequivocal benefit represented the consensus the National Institutes of Health in the United States would not be mounting the women's health initiative clinical trial/observational study (costing \$50 million a year for 14 years), one arm of which is a comparison of oestrogen, oestrogen plus progestogen, and placebo in postmenopausal women.

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