Letters

Trends in asthma mortality

Data on seasonality of deaths due to asthma were omitted from paper but editorial's author did not know

EDITOR—Readers of Ann J Woolcock's editorial about our paper may have wondered at her comments on the seasonality of deaths due to asthma[1] although data on this were not presented in our paper.[2] This was because we had been asked to remove these results to shorten the paper and she had not been told. However, we wish to present them here. The dataset was the same as that referred to in our paper. Briefly, all deaths due to asthma (International Classification of Diseases code 493) in England and Wales from 1 January 1983 to 31 December 1995 were included. Deaths were divided into calendar months, an adjustment being made for the different lengths of the months. Age specific rates were calculated using the mid-period population (1988) and the age groups 0-4, 5-14, 15-44, 45-64, 65-74, 75-84, and ≥85. Analysis was by Poisson regression in STATA 5 (StataCorp, 1997). Seasonality was tested by including both sine and cosine terms with an annual period in the regression. Each age group, except for those who died aged under 5, showed significant seasonality. For those aged 45-64 the significance was marginal ($\chi^2=8.4$, df=2, P=0.015), but for all others the P value was <0.01. The phase of the seasonality was different for each age group (figure). Deaths in the younger age groups peaked in the summer and deaths in the older age groups peaked in the winter. Deaths of people aged 5-14 peaked in August, and in those aged 15-44 the peak was slightly earlier, in July, but the rate in August was also high. Deaths in people aged 45-64 showed an intermediate pattern, with excess mortality from November to March and a separate peak in August. In those aged 65-74 there was a winter excess lasting from November to April but no summer peak.

Age related seasonal trends offer clues to aetiology. It is widely accepted that atopy is the single most prominent risk factor for the development of asthma, and this is supported by strong epidemiological data linking exposure to environmental allergens to the chronic airway inflammation that characterises the disease. The peak in August in young people in England and Wales has been reported before.[3] A possible explanation for the summer peak is exposure to outdoor aeroallergens. The peak pollen season is June and July, but certain fungal spores peak in August. It may take a month or two to build up sensitivity to pollen, but our results suggest that exposure to moulds is a more important factor than exposure to pollens for deaths due to asthma; this has also been suggested previously.[4] Social factors such as compliance or difficulty in getting medical help during the August summer holiday may also play a part. Strong emphasis has been placed on allergens in the home, especially house dust mite, but exposure to these is likely to be year round, and peak exposure to house dust mite allergen is not seasonal.[5] In the group aged ≥45 mortality is greater in the winter months. The most likely explanation is the presence of viral infections and diagnostic transfer from bronchitis (or chronic obstructive pulmonary disease) to asthma. There are strong epidemiological data linking viral infections with exacerbations of asthma, but adequate studies have not yet been carried out in exacerbations of chronic obstructive pulmonary disease.

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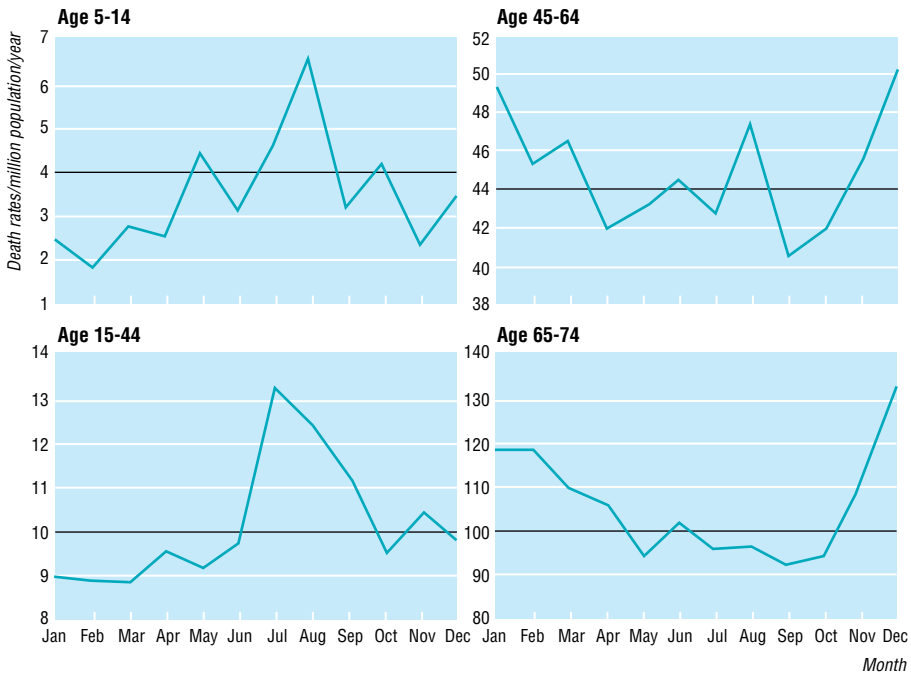

Death rates (per million population per year) by month of death and age group

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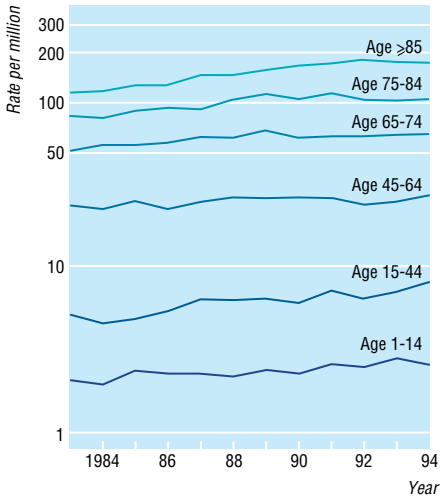
Asthma mortality in United States has risen but is similar to that in England and Wales

EDITOR—M J Campbell and colleagues analyse the age specific trends in asthma mortality in England and Wales from 1983 to 1995.[1] Their analysis indicates that there has been a downward trend in Britain's asthma mortality, especially among younger age groups. The authors suggest that this trend may be due to the increased use of prophylactic treatment.

We analysed mortality files to characterise trends in mortality attributable to asthma in the United States during the same period. The results of this analysis differed from those



Death rates from asthma (per million population) by age group from 1983 to 1994

presented by Campbell and colleagues. During the study period, asthma accounted for 54 455 deaths in the United States and the annual asthma mortality (adjusted for age to the 1980 American population) increased 39% (from 15.3/million to 21.3/million). Age specific death rates increased in every age stratum (figure). By 1994, American age specific mortality from asthma among people younger than 65 was similar to reported rates in England and Wales, whereas among people aged 65 and older they were lower.

Asthma mortality in the United States has risen despite the increased use and sale of prophylactic drugs such as inhaled corticosteroids.[2] Thus our results suggest that other factors, such as changing environmental exposures,[3] recreational drug use,[4] or socioeconomic factors,[5] may be important in trends in asthma mortality over time. On the other hand, at the end of the study period the increasing age specific mortality in the United States was still similar to or lower than the decreasing rates reported for England and Wales, suggesting that there are other differences between these two study groups.

Many gaps exist in our understanding of deaths related to asthma, which are thought to be largely preventable. Both of these studies highlight the need for improved surveillance of and education in asthma and the establishment of comprehensive prevention and intervention programmes.

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1 Campbell MJ, Cogman GR, Holgate ST, Johnston SL. Age specific trends in asthma mortality in England and Wales, 1983-95: results of an observational study. *BMJ* 1997; 314:1439-41. (17 May.)
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Death certification in asthma is inaccurate

Editor—M J Campbell and colleagues analysed age specific trends in asthma mortality between 1983 and 1995, based on statistics derived from the Office of Population Censuses and Surveys.[1] Their encouraging conclusion was that asthma mortality in England and Wales shows a downward trend. This conclusion may well be correct. However, there has been considerable uncertainty over the validity of statistics so derived,[2-4] not because of any defect in the collection and recording of statistics by the Office of Population Censuses and Surveys (now the Office for National Statistics) but, in my view, because of three factors. The first of these is inexperience and uncertainty in the completion of death certificates, often by very junior hospital doctors insufficiently trained in death certification and inadequately guided in individual cases by senior medical staff. The second factor is overuse of the word asthma by both clinicians and patients, combined with failure to differentiate chronic obstructive pulmonary disease from genuine asthma. The final factor is the automated system with which the Office for National Statistics selects out the word asthma from enormously varied death certificates as being the primary cause of death. To test the last of these and gain some insight into the first two, I asked the Office for National Statistics to cooperate with me in looking at a small number of ambivalent death certificates that I had come across in the course of the Northern region's confidential asthma deaths inquiry (roughly 15% of such death certificates examined in which the word "asthma" appeared in part one of the certificate). The table shows details on the certificates, which related mostly to elderly patients. All of these deaths, I am assured, would have been taken into the statistics as deaths due to asthma despite what seems to be an unlikely and often conflicting combination of terms.

From these death certificates, the likelihood that asthma was the primary cause of death seems unconvincing. I suggest, therefore, that one cannot draw conclusions on trends in asthma mortality from simply analysing such information unless individual cases are examined in more detail for accuracy of diagnosis.

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1 Campbell MJ, Cogman GR, Holgate ST, Johnston SL. Age specific trends in asthma mortality in England and Wales, 1983-95: results of an observational study. *BMJ* 1997;314: 1439-41. (17 May.)
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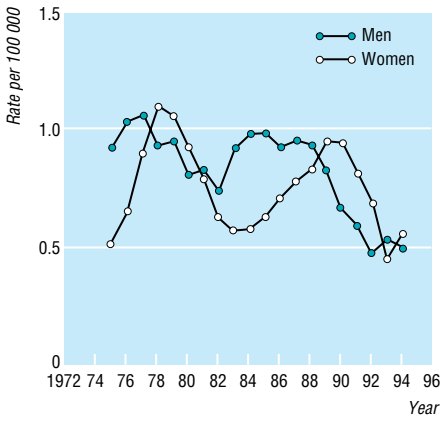
Short term fluctuations may obscure more meaningful, longer term, changes

Editor—The significant downward trend in asthma mortality in England and Wales reported by M J Campbell and colleagues should be viewed from a longer term perspective.[1] There are several precedents for doing so. When considering age specific annual mortality from asthma between 1951 and 1985 Alderson did not find any consistent trend but noted that fluctuations in rates could obscure more meaningful changes, such as the short lived increase in mortality during the 1960s.[2] Burney, in a review of mortality from 1931 to 1985, stated that it "remained fairly constant," although complex age, period, and cohort effects were found.[3] In Scotland, trends in asthma mortality were reported to have been stable between 1970 and 1989.[4] The figure illustrates these data, updated to include the years 1990-5. Mortality is shown as age

Cause of death given on 15 death certificates; all deaths would have been coded as due to asthma

Case No	Cause of death (Part I)			
	Disease or condition directly leading to death (Ia)	Antecedent causes (Ib)	Underlying condition (Ic)	Other significant condition (Part II)
1	Congestive cardiac failure	Asthma	—	—
2	Respiratory failure	Asthma, chronic bronchitis	—	—
3	Asthma	Chronic obstructive airway disease	—	Cerebrovascular disease
4	Acute asthma	—	—	Gastrointestinal haemorrhage, ulcerated duodenal leiomyoma
5	Septicaemia	Bilateral lung transplants	Asthma	—
6	Respiratory failure	Emphysema	Asthma	—
7	Bronchopneumonia	Asthma/immobility	—	—
8	Respiratory failure	Emphysema	Chronic severe asthma	—
9	Heart failure	Severe asthma	Carcinoma of colon	—
10	Bronchopneumonia	Asthma	—	—
11	Chronic obstructive airway disease and asthma	—	—	—
12	Pneumonia	Asthma	—	—
13	Chronic obstructive airway disease	Asthma	Smoking	—
14	Coronary thrombosis	Congestive cardiac failure	Severe asthma	—
15	Pulmonary embolus	Chronic asthma	—	Chronic cholecystitis



Age standardised death rates from asthma per 100 000 population aged 5-44, 1974-95. Data are plotted as three year moving averages

standardised three year moving averages for ages 5-44. This age range was chosen because of the difficulties in diagnosing asthma in small children and because of confusion with partially reversible chronic obstructive airways disease above middle age. Over the entire period these rates fluctuated without any consistent pattern, with relative rates in men and women changing throughout.

One might be tempted to argue that mortality fell among women during the late 1970s and early '80s, or that it increased during the late '80s. Like Campbell and colleagues we also found a decline in mortality in both sexes during the 1990s. In view of previous patterns, however, it is questionable whether this recent decline reflects improved prophylactic treatment of the disease or simply minor changes in a broadly constant secular trend.

We agree with Ann J Woolcock's accompanying editorial, which raised important questions about the meaning of asthma at older ages, the relevance of sex differences, and the need to relate mortality to prevalence, severity, and treatment.[5] We are puzzled, however, by her statement that "there was a big fall in death rates between 1983 and 1984"; this is not apparent in Campbell and colleagues' paper.

If improved prophylactic treatment for asthma were responsible for decreasing mortality, a concomitant fall in hospital admission rates might be expected. Our research in Scotland has confirmed previous reports that hospital admissions for asthma are rising and continue to do so. This continuing steep rise suggests that we cannot yet say that better prophylactic treatment for asthma has shown a clear benefit.

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1 Campbell MJ, Cogman GR, Holgate ST, Johnston SL. Age specific trends in asthma mortality in England and Wales, 1983-95: results of an observational study. *BMJ* 1997; 314:1439-41. (17 May.)
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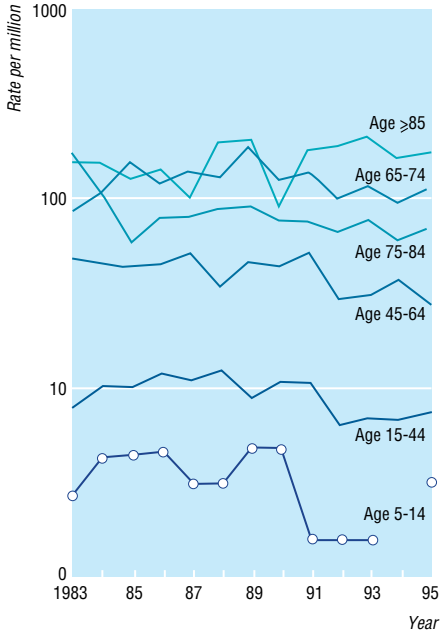
Asthma mortality is falling in most age groups in Scotland

EDITOR—Until 1989, asthma mortality in young adults in England and Wales remained similar to that occurring three decades ago.[1] Ann J Woolcock's editorial, commenting on recent downward trends in asthma mortality in England and Wales,[2] requested comparisons of asthma mortality between populations, preferably in relation to prevalence, severity, treatment, and allergic status.[3]

We can make three contributions. The first is data from Scotland (source: Scottish Office, from the Lung and Asthma Information Agency) for 1890 deaths due to asthma (1983-95), in which Poisson regression analysis suggests that asthma mortality is falling in most age groups (figure): by 7.3% (95% confidence interval 2.9% to 16.5%) a year in 5-14 year olds; 3.2% (0.1% to 6.3%) a year in 15-44 year olds; 3.4% (1.4% to 5.5%) a year in 45-64 year olds; 2.5% (0% to 4.9%) a year in 65-74 year olds; and 2.8% (0.2% to 5.3%) a year in 75-84 year olds. Only in people aged ≥85 is asthma mortality estimated to have increased, by 2.1% (−2.5% to 6.9%) a year. Trends in asthma mortality do not differ significantly ($P > 0.05$) between the sexes, except in 15-44 year olds, in whom the fall is concentrated in men.

The rate of fall in asthma mortality is less pronounced in the 15-64 year age groups in Scotland than in England and Wales, but, with the smaller number of Scottish deaths, the confidence intervals are wider and the rate of fall may be negligible or of the same magnitude. International comparisons of trends in asthma mortality will require larger populations—ones with higher asthma mortality than Scotland's or ones obtained by grouping several years' data. At smaller geographical levels few deaths occur annually and rates will be poorly estimated because of random variation, even if age groups or years are merged.



Death rates from asthma per million by age group in Scotland, 1983-95. No deaths occurred in 5-14 year age group in 1994

Secondly, since deaths due to asthma are relatively uncommon, risk factors may be efficiently studied by case-control comparisons. This would necessarily include validation of certification of deaths due to asthma, which is particularly subject to diagnostic transfer in older age groups, and could be expedited by ongoing confidential inquiries into deaths due to asthma.

Finally, the international study of asthma and allergies in childhood will be producing questionnaire based data on the prevalence and severity of asthma in children in 40 countries.[4] Assessment of treatment and physical measurements remain to be done. The European Community respiratory health survey has already reported on the prevalence and treatment of asthma in adults across Europe.[5] Data are emerging to link prevalence, severity, allergic status, and treatment to outcomes, including death, within international frameworks.

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1 Anderson HR, Strachan DP. Asthma mortality in England and Wales, 1979-89. *Lancet* 1991;337:1357.
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Trend in occurrence of asthma among children and young adults

Reporting of common respiratory and atopic symptoms has increased

EDITOR—Per Magnus and Jouni J K Jaakkola question reports of an increased prevalence of wheezing illness and asthma in recent population studies.[1] In the three Aberdeen surveys the questions relating to asthma, shortness of breath, eczema, and hay fever were identical; the question on wheeze was modified between the first and second studies.[2] The responses to the four identical questions showed significant and continuing rises in the prevalence of attacks

Prevalence of respiratory symptoms and reported diagnoses of atopy in three studies in Aberdeen 1964-94. Figures are numbers (percentages) of children

	1964	1989	1994
No of children studied	2510	3403	4034
Parents were aware of diagnosis of:			
Asthma (diagnosed by doctor)	104 (4.1)	347 (10.2)	789 (19.6)
Eczema	132 (5.3)	409 (12.0)	714 (17.7)
Hay fever	81 (3.2)	405 (11.9)	511 (12.7)
Respiratory symptoms:			
Attacks of shortness of breathness	136 (5.4)	341 (10.1)	753 (18.7)
Wheeze in past 3 years	261 (10.4)	1025 (19.8)	1025 (25.4)
Wheeze only with URTI	167 (6.7)	238 (7.0)	(7.8)
Wheeze plus hay fever	35 (1.4)	177 (5.2)	253 (6.3)
Wheeze plus eczema	45 (1.8)	160 (4.7)	298 (7.4)

URTI=Upper respiratory tract infection.

of shortness of breath and awareness of a diagnosis of asthma or eczema.

Magnus and Jaakkola suggest that future surveys should include objective assessments of asthma, including tests of non-specific bronchial hyperresponsiveness and skin prick tests. Atopy as defined by a positive result of a skin prick test with common inhaled allergens is so common in the general population (up to half of subjects) that this is unlikely to be informative. Although non-specific bronchial hyperresponsiveness is reasonably reproducible when applied to adult asthmatic populations derived from hospital, it is a much less stable marker in population based studies of children.[3]

The authors raise an interesting point concerning the changing labelling of wheezing illness in recent years. Whereas before 1970, certainly in Britain and Australia, recurrent wheeze was categorised as either asthma or wheezy bronchitis, this distinction has not been made in more recent surveys. Consequently, important information on causation and prognosis may have been obscured, as different respiratory tract symptoms such as breathlessness, cough, and wheeze with or without atopic features may underlie different clinical syndromes[4] with different prognostic implications.[5]

We have therefore looked again at the results of the three Aberdeen surveys to examine changes in the prevalence of asthma and of wheezy bronchitis, using the original definition of "wheeze only in the presence of upper respiratory tract infection." Whereas the prevalence of respiratory symptoms and reported diagnoses of atopy, including wheeze associated with other atopic disease, has shown a consistent increase, the prevalence of wheezy bronchitis has remained remarkably stable (table).

Either our population has become extremely sophisticated in translating increased publicity about asthma into increased reporting of common respiratory and atopic symptoms or these symptoms have indeed increased in our community over the past 30 years. The population stability of wheeze associated with upper respiratory tract infection and the increased reporting of atopic symptoms suggest that changing environmental factors are associated with the latter but not with the former.

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1 Magnus P, Jaakkola JJK. Secular trend in the occurrence of asthma among children and young adults: critical appraisal of repeated cross sectional surveys. *BMJ* 1997;314:1795-9. (21 June.)
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Labelling of cough alone as asthma may partially explain increase

EDITOR—Per Magnus and Jouni K Jaakkola give several factors that may contribute to the overreporting of asthma and hence the apparent increase in prevalence.[1] We wish to add two further possible factors. Firstly, in the past decade the symptom of cough alone has increasingly been used to diagnose asthma.[2] There are several problems with this: the repeatability of questions on cough in epidemiological studies is poor, the subjective reporting of cough is unreliable,[2] and cough alone is a poor marker of asthma in both epidemiological and clinical studies.[3] Therefore the labelling of cough alone as asthma may partially explain the increase. Secondly, even studies that included an objective measure of airway hyperresponsiveness in their definition of asthma need to be interpreted with care. Peat et al reported a significant increase in airway hyperresponsiveness to histamine in Australian children.[4] This may, however, be partly explained by a change in the method of delivering histamine.[5] Thus even objective methods can be misleading.

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1 Magnus P, Jaakkola JJK. Secular trend in the occurrence of asthma among children and young adults: critical appraisal of repeated cross sectional surveys. *BMJ* 1997;314:1795-9. (21 June.)
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Helicobacter gastroduodenitis

Routine treatment will lead to extra workload

EDITOR—The editorial by Anthony Axon and David Forman contains many unwarranted assumptions.[1] They suggest that doctors face ethical and perhaps legal difficulties if they fail to diagnose and treat *Helicobacter pylori* gastroduodenitis routinely in dyspeptic patients. This ignores the observation that *H pylori* gastroduodenitis is only slightly more common in people with dyspepsia than healthy volunteers[2] and that only a minority of patients with non-ulcer dyspepsia derive symptomatic benefit from eradication of *H pylori*. For these reasons the British Society of Gastroenterology's dyspepsia management guidelines do not recommend eradication unless there is proved infection in association with erosive gastritis, peptic ulcer, or lymphoma.

Axon and Forman estimate that *H pylori* infection causes 8000 deaths a year in England and Wales, most from gastric cancer. The relation between *H pylori* infection and gastric cancer is more complex than they suggest. Disease patterns are changing: distal tumours (strongly associated with *H pylori* infection) are becoming less common, yet the incidence of carcinomas of the gastric cardia (not clearly associated with helicobacter) is increasing.[3] Strangely, duodenal ulcer (very strongly linked to helicobacter) seems to confer some protection against gastric cancer.[4]

Caution is necessary when discussing an extremely common disorder in a widely read publication. The authors' gross overestimate that 15% of infected people become "seriously ill … at risk of a potentially fatal outcome" will alarm many general practitioners and lead to widespread adoption of treatment based on serology alone, a strategy condemned in a recent *Drugs and Therapeutics Bulletin*.[5] We fear that hospital gastroenterology services will struggle to cope with the extra workload generated by their suggested approach to *H pylori* infection, which is unfortunately based on hypotheses rather than hard facts.

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1 Axon A, Forman D. Helicobacter gastroduodenitis: a serious infectious disease. *BMJ* 1997;314:1430-1. (17 May.)
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Authors' reply

EDITOR—The starting point for our editorial,[1] criticised by Miles C Allison and David Williams, was the fact that increasing numbers of patients with dyspepsia are now being tested for *Helicobacter pylori* infection. We clearly stated that there was no convincing evidence to indicate that treating patients with positive results would improve symptoms in those with non-ulcer dyspepsia. Nevertheless, the testing process of necessity leads to a management decision about whether to treat patients who have now been identified as infected but who do not have an ulcer. Our strong preference, again clearly stated, was that such patients should be entered into appropriate clinical trials to provide evidence of benefit in relation to both immediate symptoms and long term outcomes, such as cancer.

In the absence of trial results, treatment decisions still have to be made and our editorial sought to provide data to assist the decision making process. The vast majority of people infected with *H pylori* will never have any clinical symptoms but a minority will develop substantive disease and a proportion of these will die as a result of their infection. Unfortunately, there are no means of distinguishing the two groups, and currently all those infected have to be regarded as being at increased risk of a fatal illness.

We believe our figures of 1 in 35 men and 1 in 60 women dying before the age of 85 as a consequence of their *H pylori* infection are based on reasonable assumptions. Given current knowledge, it is inevitable that uncertainty will be attached to this type of calculation but the figures provide "order of magnitude" estimates for the most serious consequences of infection. Adjustment for the subsite distribution of gastric cancer does not modify these estimates substantially. Our figure of 15% was an approximation for all those who develop either cancer or peptic ulcer as a result of their infection. It has been estimated that 10-20% of people infected with *H pylori* will develop peptic ulcer disease,[2] and although most of these do not become seriously ill, this is only because effective treatment is available. We do not, therefore, agree with Allison and Williams that 15% is a "gross overestimate."

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1 Axon A, Forman D. Helicobacter gastroduodenitis: a serious infectious disease. *BMJ* 1997; 314:1430-1. (17 May.)
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Adding heat probe treatment to adrenaline injection for spurting haemorrhage of peptic ulcers

Injection of adrenaline and human thrombin is best option

EDITOR—Chung et al reported a randomised study examining the efficacy of giving heat probe treatment after endoscopic adrenaline injection for actively bleeding peptic ulcers.[1] No benefit over adrenaline injection alone was detected. However, a subgroup analysis of patients with spurting haemorrhage showed benefit (reduced surgical rates) in the group given dual treatment. We are concerned about the stratification of patients in this subgroup analysis.

Endoscopic treatment is more likely to fail in patients with posterior duodenal ulcers than in patients with anterior ulcers.[2 3] Although the distribution of ulcer sites did not differ overall between the two treatment groups, it would be relevant to know the distribution of ulcers in the subgroup with spurting haemorrhage. For example, a greater number of posterior ulcers in the group given adrenaline injection alone would favour an apparent beneficial effect of dual treatment. Furthermore, comorbidity influences rebleeding rates.[4] Thus more data on comorbidity should have been provided, with stratification in the subgroup analysis for this variable.

Only two of the four end points used in the study were significant, some of the ulcers in the patients given dual treatment perforated, and the patients were incompletely stratified. These factors cast some doubt on the "advantageous" role of combined adrenaline injection and heat probe treatment in spurting haemorrhage. For the time being, endoscopic injection of adrenaline and human thrombin—an alternative therapeutic option not discussed by Chung et al—may be a more sensible option, since this has been shown to reduce ulcer rebleeding and mortality.[5]

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Authors' reply

EDITOR—Posterior duodenal ulcers are closer to large vessels than anterior duodenal ulcers are and are more likely to rebleed. Whether one can accurately identify such ulcers at endoscopy, however, is doubtful. In a study by Straker et al, true posterior location was identified in only 30% of cases by an experienced endoscopist.[1] In our experience, there is often a considerable discrepancy between the location of duodenal ulcers reported at endoscopy and their true location at laparotomy. We therefore chose not to stratify our patients on the basis of the apparent location of the ulcers.

Several studies have indicated that dual treatment is superior to injection of adrenaline alone. Kubba et al obtained excellent results by combining human thrombin and adrenaline.[2] In their study a single operator did all the endoscopic treatment. Excellent results obtained by enthusiasts need to be validated by other centres before the technique is generally adopted. The possibility that human blood products might transmit disease, however remote, must also be taken into consideration.

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1 Straker RJ, Bienvenu JC, Nord HJ. Endoscopic orientation within the duodenal bulb. *Endoscopy* 1992;24:266-7.
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"Clearing house" is needed to match available junior doctors to unfilled SHO posts

EDITOR—We wish to report a recent unexpected experience in recruitment of senior house officers. A single post as senior house officer in the accident and emergency department of St Thomas's Hospital, a teaching hospital in London, was advertised in the *BMJ* on 28 June 1997, the appointment to start just five weeks later. Within the subsequent 10 days 233 applications were received. Only four other posts as senior house officer in accident and emergency were advertised in the same edition of the journal, but there were 68 vacant senior house officer posts in major specialties that were also due to start at the beginning of August.

The 233 applicants had primary medical degrees from 24 different countries; 78 were from medical schools in the United Kingdom, 54 from other European Union countries, and 101 from non-European Union countries. The applicants were predominantly male (n = 173). Seventy two had five or more years' clinical experience since graduation. Among the 78 United Kingdom graduates, 60 were completing their second house job and had yet to obtain further employment.

We are concerned that so many house officers were still without further employment only five weeks before full registration; this suggests that they had been inadequately prepared for this difficult stage in their career. It was learnt at interview that some of them had been unsuccessfully seeking appointment to a medical or surgical rotation and then found that preference was given to candidates who already had experience in accident and emergency. They had missed by many months the usual recruiting round for posts that began in August, and at least 60 young United Kingdom doctors found themselves in a career hiatus through lack of guidance during their house officer year. Is this where some of the "lost tribe" falls?[1-3]

A possible solution to this disturbing situation would be the introduction of a "clearing house" at national level for all junior doctors approaching the end of their contracts who have yet to obtain further placement. Even if they did not obtain a job in their specialty of greatest interest they would have a better chance of obtaining further employment than by applying individually to various hospitals around the United Kingdom. Such a system could operate in June-July and December-January each year and be similar to the Universities and Colleges Admissions Service's clearing house for university placements. It would surely be a more efficient way of matching available junior doctors to unfilled posts when the time remaining is limited.

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Debate over mentally ill patient's caesarean section was too emotional

EDITOR—We are the clinicians involved in the case of Tameside and Glossop Acute Unit versus CH (a patient) [1996] 1FLR 762. We wish to add light to the debate on this case published in the *BMJ*,[1] which was not helped by the provocative title of the article or by Goldbeck-Wood's inaccurate editorial.[2] It was not our "perceived ethical duty to rescue a threatened fetus"[2] but our clinical responsibility to help our patient obtain the best outcome of her pregnancy: there was no conflict over the mother's wishes but concern over her severely distorted perception of fetal wellbeing in the later stages of pregnancy. We set out not to "override CH's rights" but to clarify them.

Because there were signs of intrauterine growth retardation, it was not the obstetrician's plan to deliver by caesarean section but to induce labour and monitor throughout, in anticipation of a normal delivery. On three occasions, after a long discussion, agreement for induction of labour was obtained, only for it to be refused when an approach to induce was made.

Caesarean section was the alternative to regular sexual assaults to insert prostaglandin pessaries, carry out vaginal examinations, rupture the forewaters, or apply a fetal scalp electrode. Maintaining an intravenous transfusion and monitoring in an uncooperative, potentially violent, patient would be difficult.

It is not unreasonable to assume that, if a patient who is actively psychotic with paranoid delusions experiences a perinatal death, she will blame her attendants and that this will compromise her compliance with future care, as well as producing a grief reaction. Optimal obstetric care would then be part of the treatment of her mental state.

Whitfield's remarks in his commentary focus this crucial debate, which is, in essence, a legal one. We agree that there should be public debate and parliamentary scrutiny of the limits of section 63 of the Mental Health Act 1983. Unfortunately, Bewley's commentary is so emotive that the facts and details of the complexity of management have suffered badly: the implications of the neuroleptic malignant syndrome developing and possible fetal death have been ignored.

An important postscript is that the patient remains under the care of the same psychiatrist (GM), has made a complete symptomatic recovery from her mental illness, and complies fully with her care package. She is also a competent and independent mother, caring for both her children. She still believes that the caesarean section was necessary but still does not accept that she was unwell at the time.

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Insulin dependent diabetes is probably due to environmental effect during childhood

EDITOR—Petersen et al report autoantibodies associated with insulin dependent diabetes mellitus in a set of monozygotic and dizygotic Danish twin pairs in which one or both twins had diabetes.[1] We have similar results from a study of British twins collected over 30 years. We studied antibodies to islet cells, glutamic acid decarboxylase, and the novel antigen protein tyrosine phosphatase.[2] We did not study insulin antibodies, because in insulin treated patients they cannot be designated autoantibodies. Eighteen pairs of monozygotic twins discordant for recently diagnosed insulin dependent diabetes were matched for age, sex, and duration of disease with 18 pairs of dizygotic twins discordant for the disease and followed up prospectively. Antibodies were found in six of the 18 non-diabetic monozygotic twins and in six of the 18 non-diabetic dizygotic twins. In most pairs, antibodies were detected in only one of the twins. Combinations of more than one antibody were detected in both twins in six of the monozygotic but only one of the dizygotic pairs ($P < 0.05$). Antibody combinations in seven of the 36 non-diabetic twins were associated with subsequent progression to insulin dependent diabetes in six of the seven twins ($P < 0.01$). Diabetic twins had more antibodies and more combinations of antibodies than twins who remained non-diabetic.

These studies, taken together, indicate that autoimmunity associated with insulin dependent diabetes is environmentally determined; taken with an American study, they suggest that the prevalence of this shared environmental effect is high.[1-3] Petersen et al speculate whether a continuum of events is required for islet cell autoimmunity in twins to progress to clinical diabetes; such a continuum has already been shown. Numerous twin studies, recently summarised, have shown humoral, cellular, and metabolic changes in twins who do or do not develop insulin dependent diabetes.[4] Twins who develop insulin dependent diabetes differ from those who do not in the extent, quality, and persistence of these changes.

While we have favoured the idea of an early environmental event causing insulin dependent diabetes, we cannot say that either our data or those of Petersen et al support the notion that this event occurs specifically in fetal or early postnatal life.[5] Dizygotic twins share their environment more than other siblings do throughout childhood. Thus the higher frequency of autoantibodies in non-diabetic dizygotic twins compared with siblings of patients with insulin dependent diabetes argues for a common environmental effect operating at some time during childhood, but not necessarily in utero.

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Palliative drugs are not for shortening life

EDITOR—Clare Dyer's report about Annie Lindsell, who has motor neurone disease, suggests that she may still be trying to ensure that her life can be ended at her request when she feels that she cannot face any further distress.[1] She wants her general practitioner to be lawfully able to give drugs to relieve her distress in the terminal stages of the illness, even if this could shorten her life.

Although patients with motor neurone disease are understandably worried about their future and the possibility of a distressing death, with good palliative care throughout the illness, death need not be distressing.[2] Many symptoms, particularly pain, dyspnoea, and drooling, can be alleviated by careful manipulation of drugs and the careful assessment of all the patient's needs—physical, psychosocial, and spiritual. These symptoms can be helped without necessarily shortening life, and morphine has been shown to be effective in the control of pain and dyspnoea. A study at the Wisdom Hospice showed that the mean dose of oral morphine for patients at home was 90 mg/24 h and the mean duration of use was 240 days, confirming that morphine does not necessarily lead to death.[3]

The Motor Neurone Disease Association has developed the "breathing space pack" to help in the management of a crisis in the terminal stages of the disease.[4] The pack is provided by the association, and, after discussion between the patient, family, and primary health care team, drugs are prescribed by the general practitioner to alleviate distress. In this way drugs are readily available and all are aware of the best way of controlling symptoms and the action to take. As with the prescription of any drugs, there is always a need to balance the benefits against the risks. The aim of these drugs is to relieve distress and not to shorten life, although there is a risk of the latter.

Many patients with motor neurone disease fear increasing dependency and disability, and there is little that can be done to alleviate these symptoms. Nevertheless, there may be ways of helping patients cope, and a careful multidisciplinary assessment allows the most to be made of their remaining abilities. Professor Stephen Hawking has shown how communication can continue, even if it is by way of complex computer communication systems.

Palliative care can provide care for people with advancing disease, but without the need to shorten life. Ms Lindsell may need reassurance that her symptoms will be helped appropriately and the necessary drugs will be given. Drugs, however, should always be for the control of symptoms and not for shortening life.

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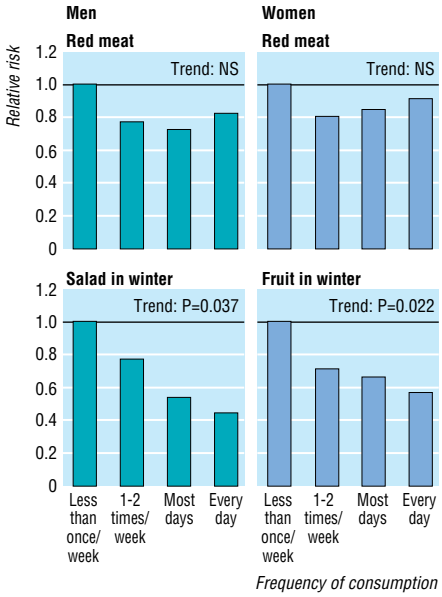
Frequent consumption of red meat is not risk factor for cancer

EDITOR—Headlines such as "Big meat eaters cancer warning" (*Daily Mail*, 13 September) have appeared in advance of the publication of the Department of Health's report on diet and cancer.[1 2] A prospective study, however, analysed data from a nationwide random stratified sample of British adults to determine the relation between diet and cancer and found a protective role for fruit and salads but no evidence that frequent consumption of meat is a risk factor for cancer.

Details of dietary habits (including the usual frequency of consumption of 31 food items), smoking behaviour, and health status were obtained from respondents to the health and lifestyle survey in 1984-5, and details on health status were obtained from the follow up survey in 1991-2.[3] All participants were flagged on the NHS Central Register so that death certificates were received with appropriate coding (International Classification of Diseases, ninth revision). Altogether 1630 men and 2030 women, aged 35-75, did not have cancer in 1984-5 and either were interviewed again in 1991-2 or had died from cancer by then (89 men and 73 women, including 28 who had died of colorectal cancer). Forty four men and 76 women who were interviewed again in 1992 had developed cancer.



Relative risk of developing cancer in people who consumed specific foods several times a week compared with those who consumed them less than once a week, adjusted for age and smoking

Logistic regression analysis was used to examine the relation of the frequency of consumption of red carcase meat and of fruit and salad in winter[4] (reflecting year round consumption) with the development of cancer over seven years. Confounding variables included age (in five year categories) and smoking (in four categories), with the sexes analysed separately. The importance of smoking as a significant risk factor for the development of cancer was confirmed.

There were no indications that, when compared with the reference category of eating red meat less than once a week, more frequent consumption of meat was associated with the development of cancer in men or women (figure). In contrast, there were significant trends for the relation between increasing frequency of consumption of fruit or salads in winter with a decreasing risk of developing cancer, the association being strongest with salads in men and with fruit in women.

Most of the supportive evidence in prospective studies for an association between consumption of meat and colorectal cancer comes from the United States rather than Europe.[5] The way in which the meat is cooked and the relation of meat to fruit and salad vegetables in a balanced diet might explain the inconsistent findings.

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Incidence of early syphilis acquired in former Soviet Union is increasing

EDITOR—Since 1990 there has been a growing epidemic of syphilis in the countries of the former Soviet Union.[1] The Russian health ministry recently reported a 15-fold increase in the incidence of syphilis in adults and a 20-fold increase in children.[2] Most cases are attributed to sexual transmission. Explanations for this phenomenon include the rapid growth of the sex industry, including child prostitution; increasing numbers of homeless people and refugees in Russian cities; poor diagnostic facilities; punitive legislation reducing the likelihood of presentation to treatment services; and limited or inadequate treatments. Effects of this epidemic have already been noted in other countries: in Finland the incidence of syphilis has increased considerably since

the border with Russia was opened in the early 1990s.[3]

We undertook a retrospective review of cases of infectious syphilis to assess the impact of the Russian epidemic on this genitourinary medicine clinic in central London. Eleven cases were treated between January 1995 and December 1996. All patients had signs of primary or secondary syphilis or positive serological findings, or both. Six patients could only have acquired syphilis as a result of sexual contact with a Russian partner, either in Russia or in Britain.

These results show that a substantial proportion of cases of syphilis treated in this clinic can be attributed to the epidemic in Russia. Similar cases have recently been reported from other centres.[4] The Public Health Laboratory Service has recorded that, since routine reference laboratory reporting of syphilis began in April 1994, the disease was acquired abroad in 48 of 87 cases (A Nicoll, personal communication). Of these, the largest proportion (14/87) were acquired in eastern Europe.

We have observed an increasing number of Russian commercial sex workers attending our outreach service in Soho, and this phenomenon has also been reported in the local press.[5] The incidence of syphilis has decreased dramatically in Britain since the early 1980s, and the disease is therefore increasingly easy to overlook. As it may present in various ways, patients may be seen by clinicians in many specialties. Surveillance data from the Public Health Laboratory Service show that 28% of cases of infectious syphilis are reported from sources other than genitourinary medicine clinics. These cases illustrate the need for increased vigilance for early syphilis, especially in people who have had sexual contacts in the former Soviet Union.

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Stroke prevention in atrial fibrillation

Suggested range of international normalised ratio may lead to overanticoagulation

EDITOR—In their editorial on the use of anticoagulants in atrial fibrillation Tim Lancaster and colleagues suggest that a target international normalised ratio of 2.0-4.0 should be sought.[1] We have reservations about this. Any intensity of oral anticoagulant treatment increases the risk of serious bleeding and particularly intracranial haemorrhage, which carries a mortality of 60%. With an international normalised ratio in the suggested range, the risk of intracranial bleeding is increased 7-10-fold and is proportionate to the intensity of anticoagulation.[2] Furthermore, elderly patients, who are at higher risk of ischaemic stroke with atrial fibrillation, are also at the highest risk of bleeding due to oral anticoagulants. Intensity of anticoagulation is very much a balance of risks.

Lancaster and colleagues base their conclusions primarily on two reports. The stroke prevention in atrial fibrillation III randomised trial, which compared adjusted dose warfarin (international normalised ratio 2.0-3.0) with low dose warfarin (ratio 1.2-1.5) plus aspirin, was halted because the low dose warfarin was ineffective in preventing ischaemic strokes in high risk patients with atrial fibrillation.[3] The adjusted dose warfarin group fared better, but patients whose mean international normalised ratio was 1.9-2.4 were at no greater risk of ischaemic stroke than those with mean ratios of ≥ 2.5. Hylek et al concluded that the risk of ischaemic stroke is significantly higher at ratios of < 2.0, but they based this observation on only the ratio on admission in patients presenting with stroke and on a single ratio in control cases.[4] No effort was made to assess the average ratio over a period in either group.

Deciding the dose of oral anticoagulant is an imprecise art. The higher the target international normalised ratio the greater will be the fluctuation in the ratio that is observed and the greater the risk of dangerous overanticoagulation. Many less experienced doctors will adjust the dose only when the ratio falls outside the target range, and a range of 2.0-4.0 or even 2.5-3.5 provides considerable scope for overanticoagulation, especially in older patients. We would argue that, on the basis of the evidence, a target range of 2.0-2.5 (or a target ratio of 2.2 or 2.3 if computerised support systems are used) will provide safer prophylactic anticoagulation against ischaemic stroke in older patients (aged > 70) with atrial fibrillation. It is surely better to err on the side of lower level anticoagulation while aiming for a therapeutic target range than to risk iatrogenic and potentially fatal haemorrhage in a group of patients at high risk of both ischaemic stroke and intracranial bleeding.

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Authors' reply

EDITOR—To prevent the actual international normalised ratio falling below 2.0 or rising above 4.0, we recommended a target range of 2.0-3.0. We do not agree that there is a 7-10-fold increase in the risk of intracranial haemorrhage at this intensity of anticoagulation. The source cited by Frank Booth and Arjun Mehta for these figures was an informal review.[1] It included studies with target ranges as high as 4.8 and patients who were taking warfarin for reasons other than atrial fibrillation. A better estimate of the risk of haemorrhage comes from randomised trials of warfarin in patients with atrial fibrillation. In five primary prevention trials, warfarin was effective for the prevention of stroke in atrial fibrillation with target ranges as low as 2.0-3.0. In a pooled analysis of these studies, patients treated with warfarin had an absolute increase in risk of intracranial haemorrhage of 0.2% a year—a threefold relative increase.[2] Observational data suggest that there is a much steeper increase in risk when the international normalised ratio rises above 4.0.[3]

We set out to highlight new evidence that more clearly defines the effective intensity of anticoagulation for patients with atrial fibrillation. A priority for future research should be to determine the best strategy for keeping the international normalised ratio in the target range.

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Elimination of firearms

All guns should be banned from homes

EDITOR—Paula Baillie-Hamilton questions whether reducing guns in the community will reduce premature deaths.[1] She urges that advocates for a reduction in guns first "look at the size of the problem" but then goes on to consider only the rate of homicide by guns. For every person murdered with a gun in England and Wales there are 4.75 who commit suicide with a gun.[2] The gun lobby argues that reducing access to guns will simply result in method substitution, but the largest multination study concluded that the rate of household ownership of a gun correlated with the rates of homicide and suicide in which guns were used, as well as the overall rates of homicide and suicide.[2] The study found no evidence of a substitution effect in nations with low rates of gun ownership.

Baillie-Hamilton also fails to consider the relative size of the problem of deaths due

to guns in nations with fairly laissez faire gun policies. A comparison of homicide rates found that the rate of homicides not due to guns was 3.7 times higher in the United States than in Britain, while the rate of homicides due to guns was 175 times higher.[3] Forty eight per cent of American households have guns, compared with 4.7% of British households.[2]

Writing from Australia, I find the most curious aspect of the debate over gun control in Britain since the shooting in Dunblane to be why the legislation has concerned only handguns. Thomas Hamilton used a semiautomatic pistol in Dunblane; Martin Bryant used a semiautomatic rifle when he killed people in Tasmania. Britain changed its handgun laws; Australia outlawed semiautomatic rifles and pump action shotguns. Can we presume from this that if the killers' choice of weapons had been reversed, Tony Blair would have effectively stopped pheasant and grouse shooting? A sane policy, surely, would be to ban all guns from homes and require their use to be confined to target shooting ranges under the strictest of licensing and storage conditions. Gun shops are already entrusted to store guns in maximum security. Such a policy would surely gain their support.

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Author gave underestimate of deaths due to firearms

EDITOR—Paula Baillie-Hamilton is right that comparatively few homicides in England and Wales are due to firearms.[1] It is also true that firearms cause relatively few deaths overall in England and Wales compared with countries with more widespread gun ownership. But the impact of firearms on mortality is grossly underestimated by the Home Office figures she quotes for homicides in 1992-4.

Figures for England and Wales compiled by the Office for National Statistics show that there were at least a further 577 deaths due to firearms in 1992-4, including 480 suicides (*International Classification of Diseases* (ninth revision) codes E955.0-4), 58 injuries of undetermined intent (E985.0-4), 28 accidents (E922), and 11 injuries due to legal intervention by people using firearms (E970).[2 3] Statistics from the Office for National Statistics also include 131 deaths certified as due to homicide (E965.0-4) by firearms in this period. This, however, is an underestimate of the true number because the Office for National Statistics does not receive final information on homicides until legal proceedings have been completed.[4] If we use the Home Office figure of 196 homicides, instead, the total number of deaths due to firearms in the three years 1992-4 was 773, of which only a quarter were homicides. Thus the average number of deaths due to firearms each year is 258, about four times the figure given by Baillie-Hamilton.

The number of deaths due to firearms is small compared with the number caused by tobacco smoking or motor vehicle accidents, but it is not negligible, and at inquest for 70% of cases suicide or an open verdict (representing mainly self inflicted injuries in England and Wales) is recorded.[5] Evidence from past natural experiments (the change from domestic coal gas to natural gas and from barbiturates to benzodiazepines) indicates that suicide can be reduced by removing the more effective instruments of self destruction.[5] Seven out of every 10 deaths from firearms in England and Wales are self inflicted. Many of these could probably be prevented by making lethal weapons less accessible.

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Medical advice is available for ships at sea

EDITOR—Paul Bemrose can rest assured that the Italian proposal to offer a medical consulting room to the world[1] most certainly did happen, and continues to this day. The International Radiomedical Centre, in Rome, celebrated its 60th birthday in 1995 and produced a booklet detailing the history of the organisation.[2] Founded by Professor Guido Guida (figure), it has grown to provide medical advice to patients aboard ships, on planes, and on small Italian islands and has cared for more than 36 000 patients.

We have been providing a similar service from this hospital since 1993. Any ship requiring medical advice can telephone, fax, or telex Sydney Radio in Australia, which will transfer the call to us. We cover no preset boundaries of the earth's surface, and, although most of our calls come from merchant shipping in Australia's international waters, we have recently given advice to vessels in the Mediterranean and mid-Atlantic.

Despite the wonders of modern telecommunications it is still the basic things that let us down. Merchant ships fail to stock the recommended drugs, and even if they do the drugs are often out of date when needed. In 1937 the International Radiomedical Centre campaigned for ships' medical chests to be updated; only a few weeks ago I was involved with a case of presumed cerebral malaria, untreatable because the merchant vessel did not stock the quinine recommended in the *International Medical Guide for Ships*.[3] The patient died, despite our efforts to drop supplies by air to the ship, which was in the middle of the Indian Ocean.

Our experience shows that the provision of medical care to the crews of merchant ships is still poor. They receive fragmented and multilingual primary care, and when they need urgent medical attention their extreme isolation is exacerbated by a lack of basic drugs and equipment. There is still much to be done, and we would be interested to hear from organisations providing similar services.

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1 Bemrose P. Medical advice for the world. *BMJ* 1997;314:1735. (14 June.)
2 Centro Internazionale Radio Medico. *The sixty years of the International Radio Medical Centre.* Rome: CIRM, 1995.
3 World Health Organisation. *International medical guide for ships.* Geneva: WHO, 1988.

Professor Guido Guida, who founded the International Radiomedical Centre in Rome

Defence of cardiologist in misconduct case

Union defence should not always apply

EDITOR—After the collapse of the libel action by Peter Nixon against Channel 4,[1] I would like to suggest, as a member of the Medical Defence Union, that a number of issues should be addressed by the organisation. In the Nixon case £2m of members' contributions has been wasted defending a semiretired cardiologist whose views are not accepted by the majority of his colleagues.

The organisation must decide whether it should be involved in funding libel actions in any circumstance. If there are certain situations in which it should fund libel actions, should these be confined to mainstream medicine when an important principle is at stake that could affect the livelihood of other members? Also, should the union be more careful in assessing the scientific merit of the papers that it chooses to defend?

The issues raised by this action had no relevance to other members of the union, and many will feel aggrieved that this action was ever begun. Absolutely no benefit was obtained, and indeed the effects on the reputation of the union can only have been adverse.

Martin Wolfson *General practitioner*
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1 Dyer C. Cardiologist admits research misconduct. *BMJ* 1997;314:1501. (24 May.)

Members of Medical Defence Union are entitled to help with defamation cases

EDITOR—The Medical Defence Union has a reputation for winning cases that it decides to defend or take up on behalf of its members. Although most of these go unreported, they are of great importance to the doctors concerned. The board, council, and cases committee of the union systematically and critically analyse the decisions they make, including the procedures used and the outcomes, with the objective of maintaining and improving the quality of service offered to members.

The Medical Defence Union generally helps members with problems of a medicolegal nature that arise from the clinical practice of medicine. In regard to the issues raised by Martin Wolfson, as the union is a discretionary mutual society, its board of management regularly reviews the benefits of membership offered to members. These benefits include the possibility of help with defamation cases.

Wolfson's question about limiting the benefits of membership to mainstream medicine is an interesting one: how would this be defined? The livelihood of many members is in part dependent on areas of practice that some might consider outside the mainstream.

The union bases its decisions to give help on careful examination of the facts of individual cases and on expert guidance received from clinicians and legal advisers experienced in areas relevant to the case. In the rare event that cases have unexpectedly adverse outcomes they receive particularly close attention from the union, and, as in clinical medicine, these cases often provide the best lessons.

Michael Saunders *Chief executive*
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Delayed diagnosis for breast disease is mostly due to patients

EDITOR—We applaud the Department of Health's initiative to reduce the time taken for a woman with a suspicious breast lesion to have a consultation with an expert.[1] We have been undertaking a project to determine the clinical, social, and psychological factors that influence delay. Delay due to the patient and delay due to the system must be distinguished. Patient delay may be defined as the time between the patient detecting a breast symptom and visiting a healthcare provider to seek evaluation of the symptom. System delay is the time from that evaluation to the start of treatment.

We have calculated the delay in a consecutive sample of 686 women aged 40-75 presenting to King's College Hospital breast clinic with a breast symptom; 83 were later diagnosed as having cancer. Patient delay was determined with a structured interview, corroborated when possible from general practice records. System delay was determined from general practice and hospital records. The mean age of the sample was 53 (SD 9.7).

Patient delay showed a highly skewed distribution, with some women presenting immediately, a large group presenting within one to three weeks, and the remainder delaying for up to several years. Mean patient delay was 123 (659) days (median 13). System delay was also skewed, with most of the women being seen in a specialist clinic within two to four weeks while others were initially reassured or not referred on for other reasons (mean 45 (170) days; median 18). Patients who were found to have malignant disease presented earlier (mean 73 days; median 7) and were generally processed through the system quicker (mean 23 days; median 14).

Preliminary analyses suggest that sociodemographic factors such as age and ethnicity do not exert a significant influence on either type of delay. Patient delay does not seem to correlate with system delay.

Clearly, there is scope to reduce the time a women has to wait until her breast symptom is dealt with by a specialist. Resources and training that will facilitate speedy onward referral from primary care and that enable a rapid response from secondary care need to be provided. However, two points should be borne in mind. Firstly, the proportion of patients in this setting who were found to have malignant disease (as opposed to those identified through screening) was low. Secondly, patient delay contributes the bulk of total delay. Hence, effective health promotion strategies should be weighted towards approaches that encourage all symptomatic women to present early, as well as approaches targeting healthcare professionals.

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Anthony David *Professor of cognitive neuropsychiatry*
Departments of Psychological Medicine, Surgery, King's College School of Medicine and Dentistry

Tim Crayford *Lecturer in public health*
Edna Elias *Breast care specialist nurse*
Jonathan Roberts *Consultant surgeon*
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1 Warden J. Labour acts to cut NHS costs. *BMJ* 1997;314: 1574. (31 May.)

Words matter

EDITOR—I have kidney failure and for three years have had peritoneal dialysis. The medical profession keeps referring to my illness as "end stage kidney failure." Some of my fellow patients, particularly those of a worrying nature, are quite distressed by the words "end stage."

In future, could doctors please use the term "kidney failure" without any qualifying adjective?

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Correction

The future of healthcare systems

We regret greatly that Dr Graham Winyard's name was attached by mistake to the list of authors on a letter published last week (11 October, p 953). The authors copied the letter to Dr Winyard, medical director, NHS Executive, and by mistake his name was attached to the list of authors. Our shame is compounded by our misspelling of his name. We apologise to Dr Winyard.

Advice to authors

We receive more letters than we can publish: we can currently accept only about one third. We prefer short letters that relate to articles published within the past four weeks. We also publish some "out of the blue" letters, which usually relate to matters of public policy.

When deciding which letters to publish we favour originality, assertions supported by data or by citation, and a clear prose style. Letters should have fewer than 400 words (please give a word count) and no more than five references (including one to the BMJ article to which they relate); references should be in the Vancouver style. We welcome pictures.

Letters, whether typed or sent by email, should give each author's current appointment and full address. Letters sent by email should give a telephone and fax number when possible. We encourage you to declare any conflict of interest. Please send a stamped addressed envelope if you would like to know whether your letter has been accepted or rejected.

We may post some letters submitted to us on the world wide web before we decide on publication in the paper version. We will assume that correspondents consent to this unless they specifically say no.

Letters will be edited and may be shortened.