an accident. We advocate that all emergency services have procedures in place so that prompt and appropriate action can be taken to keep to a minimum infection from any agent, including blood-borne viruses, to people exposed in an emergency situation. It is vital that such an emergency service does not replace occupational health services which should be available to all emergency services.

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Non-sedating antihistamines and cardiac arrhythmias

SIR—Since Lindquist and colleagues[1] wrote about the risks of non-sedating antihistamines, several comments have appeared in *The Lancet* about the use of these compounds in the context of cardiac arrhythmias.

With respect to cetirizine, we believe that it is safe from a cardiac perspective. Sale and co-workers[2] showed no lengthening of the QTc interval during 7 days' treatment with up to six times the recommended daily dosage of cetirizine. Furthermore no clinically relevant changes with coadministration of ketoconazole (data on file), erythromycin,[3] or azithromycin[4] have been recorded. These data are clearly consistent with the low hepatic metabolism of cetirizine and its low volume of distribution, which might account for the minimum intracellular penetration of the drug.

After 10 years of experience and the use of over four billion standard daily doses, no case of torsades de pointes cardiac arrhythmia attributable to cetirizine has been reported even when there has been an accidental overdose. Similarly, analysis of the two cases of sudden death mentioned by Lindquist et al[1] does not point to any relation between intake of the drug and the occurrence of these events, which are liable to arise spontaneously in a sufficiently large population. We therefore do not consider that there is a link between cetirizine and cardiac and sudden deaths, as Andrew Rankin (Oct 18, p 1115)[5] incorrectly states, and we are confident that all the available data do not show an increased risk of torsades de pointes cardiac arrhythmia after intake of cetirizine.

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1 Lindquist M, Edward IR. Risks of non-sedating antihistamines. *Lancet* 1997; **349:** 1322.
2 Sale ME, Barbey JT, Woosley RL, et al. The electrocardiographic effects of cetirizine in normal subjects. *Clin Pharmacol Ther* 1994; **56:** 295–301.
3 Sale ME, Woosley RL, Thakker K, Phillips K, Caridi F, Chung M. Effects of cetirizine and erythromycin, alone and in combination on QT interval and pharmacokinetics in healthy subjects. *Ann Allergy Asthma Immunol* 1996; **76:** 93.
4 Sale M, Woosley R, Thakker K, Phillips K, Cardidi F, Chung M. A randomized, placebo-controlled, multiple-dose study to evaluate the electrocardiographic and pharmacokinetic interactions of azithromycin and cetirizine. *Ann Allergy Asthma Immunol* 1996; **76:** 93.
5 Rankin AC. Non-sedating antihistamines and cardiac arrhythmia. *Lancet* 1997; **350:** 1115–16.

Tobacco sponsorship of Formula One motor racing

SIR—Anne Charlton and colleagues (Nov 15, p 1474)[1] claim to show that boys who like motor racing were more likely to start smoking, with the implication that the one caused the other (rather than just showing a coincidence of attitudes). One valid fact is that if either parents or friends smoke, a boy is more likely to start smoking.

In respect of their conclusion about motor racing, a review of the data shows that of the 1063 boys questioned, 125 liked motor racing. Of these, 16 started smoking. This base is so small that if only one of these had answered either question differently, the results would have lost their statistical significance—which is hardly a strong base on which to demand changes in public policy.

I find it disturbing that such weak research and ill-founded conclusions are not only published in *The Lancet*, but also treated as authoritative in an editorial, and are used to generate much media interest. I note that the research was completed in 1995, but was not published then. It would seem that its publication now is intended to embarrass the UK government, following its recent decision on allowing tobacco sponsorship to continue for Formula One motor racing.

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1 Charlton A, White D, Kelly S. Boys' smoking and cigarette-brand-sponsored motor racing. *Lancet* 1997; **350:** 1474.

SIR—Your Nov 15 editorial[1] endorsement of Anne Charlton and colleagues'[2] study of boys' smoking and cigarette-brand-sponsored motor racing, like the study itself, says more about your shaky knowledge of epidemiology, logic, and the Smee report,[3] not to say the nature of sponsorship, than it does about the connection between tobacco sponsorship and adolescent uptake of smoking.

It is now widely accepted that there are multiple risk factors for adolescent smoking. Conrad and co-workers[4] report on over 400 of these, whereas the Goddard report[5] examines seven. Clearly if smoking is to be described as it increasingly is as a paediatric disease, it is a multifactoral disease. This means that a study, such as Charlton's, which fails to control for the multiple risk factors for adolescent smoking, can tell us nothing about the causes of any adolescent's beginning to smoke. A study of this type can in principle tell us only about the association of certain alleged predictors and adolescent smoking: it can never legitimately use the language of cause and effect nor provide evidence of causality.

Thus, Charlton and colleagues' logic that: "72 764 non-smokers might be expected to watch motor racing"; and "of these boys, 9314 would take up smoking", leads to "any ban on tobacco advertising must include all sponsored sport", is specious. Even allowing that the results of the study can be generalised, the study provides neither logic nor evidence to support the claim that tobacco-sponsored motor racing has caused anyone to take up smoking. Indeed, it is just as reasonable to conclude, from the data presented, that both adolescent interest in motor car racing and smoking are caused by some other factor as it is to conclude that adolescent smoking is caused by tobacco sponsorship of motor-car racing.

Indeed, had Charlton et al been more attentive to the Smee report they would have discovered this before undertaking a study that by definition can demonstrate nothing. For instance, in reply to the comment that "these boys were significantly more likely than the others to name Marlboro . . . and Camel", Smee[3] notes that "awareness of advertising is at most a necessary condition for coming under its influence. It is not reliable evidence that advertising increases consumption". Again Smee notes that although adolescents may well recognise tobacco advertisements it does not follow that such advertisements initiate smoking since "it is also possible that those children who react most positively to advertising are already disposed to smoke".

Finally, it is true that the "Smee report made it clear that cigarette advertising is associated with tobacco consumption", but not quite in the way that Charlton and co-workers claim. As to the effect of advertising on consumption in the UK, Smee noted that "advertising does not have a statistically significant effect in any form". Moreover, Smee carried out a detailed statistical analysis of whether tobacco advertising is "likely to influence the smoking prevalence among teenagers". A separate model was developed for both teenage men and women and "in neither model was advertising a significant factor".[3]

J C Luik

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1 Editorial. Labour government's tobacco spin spins them off track. *Lancet* 1997; **350:** 1411.
2 Charlton A, White D, Kelly S. Boys' smoking and cigarette-brand-sponsored motor racing. *Lancet* 1997; **350:** 1474.
3 Smee C. Effect of tobacco advertising on tobacco consumption: a discussion document reviewing the evidence. London: HM Stationery Office, 1992.
4 Conrad L, et al. Why children start smoking cigarettes: predictors of onset. *Br J Addiction* 1992; **87:** 1711–24.
5 Goddard E. Why children start smoking. London: HM Stationery Office, 1990.

Authors' reply

SIR—P A Sadler's first point concerns the causal nature of cigarette sponsorship on Formula One motor racing and boys' smoking. Our research was a cohort study that followed the same children over 5 years from the ages of 9 and 10 years to 13 and 14 years. Thus the study was longitudinal and not cross-sectional, and it was possible, as we said, to observe that the watching of motor racing preceded the onset of regular smoking by up to a year. We also examined the boys' self-perception in six domains[1] and in no respect were those who watched motor racing significantly different from other boys.

The links between children's smoking and their parents' and best friend's smoking are well-known, as Sadler points out.[2] We were well aware of this when we designed the study and planned the analysis, which is why these factors were included. The fact that multiple influences operate on children's uptake of smoking does not negate or reduce the strength of sponsored motor racing as an independent significant factor.

With respect to the small numbers involved, we emphasise that if the scenario that he suggests is projected to the estimated population of 606 365 non-smoking boys in this age group the odds ratio would be 1·81 (85% CI 1·77–1·86). In the study as reported, the findings with regard to the onset of regular smoking in relation to watching Formula One motor racing were still significant. The boys in question did more than just experiment with smoking, which would have represented a much larger group: they became regular, soon to be addicted,[3] smokers.

As previously stated, the research spanned 5 years. The findings were not held back or specially produced to embarrass the UK government, following its recent decision, as Sadler suggests. The analysis of 5 years' complex data takes time and we recently covered the topics of advertising and sponsorship spanning the whole period. We were so struck by the link between watching motor racing and the onset of regular smoking in boys at the age of 13 years—the age the cohort reached in 1995—that we decided to publish it as a separate item. The age of 13 years is known to be a critical one in the establishment of regular smoking.[4] In fact, the letter reached *The Lancet* on Oct 15, 1997, and was accepted for publication well before the government's announcement.

In reply to J C Luik, we make the point that cigarette-brand sponsorship in connection with Formula One motor racing is a specific type of advertising. The product is strongly associated with fast cars, risk taking, danger, the macho image, and fame, and therefore cannot be equated with other advertising strategies.

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1 Harter S. Manual for the self-perception profile of children. Denver: University of Denver, Colorado, 1985.
2 Royal College of Physicians. Smoking and the young. London: Royal College of Physicians, 1992.
3 McNeill A. The development of dependence on smoking in children. *Br J Addiction* 1991; **86:** 589–92.
4 Jarvis L. Smoking among secondary school children in 1996: England. London: Office for National Statistics, 1997.

SIR—You rightly criticise the donation of £1 million to the Labour Party by the Chief of Formula One Constructors Association (Car Racing) and the exemption of Formula One motor racing from tobacco advertising ban in your Nov 15 editorial.[1] In Goa, this news brings to mind the tactics used by the tobacco industry to delay, scuttle, and dilute the provisions of the The Goa Prohibition of Smoking and Spitting Bill, 1997.

This bill was supported by about 20 medical and other voluntary organisations under the auspices of National Organisation for Tobacco Eradication. The Bill bans smoking and spitting in public places and vehicles, advertisements of all types, direct or indirect, sale of tobacco products to individuals younger than 21 years of age (age for consent for marriage in India), and to anyone within 100 m of an educational establishment or a place of worship. Violation of the clauses on advertisement and sale to minors will lead to imprisonment for up to 3 months; other offences will result in various levels of fines. Non-governmental organisations will have some powers in the execution of these provisions.

The Goa Government first tried to delete the advertisement-ban clause, but later, on the recommendation of the select committee, the bill was passed unanimously by the Legislative Assembly. Sadly, the Governor's consent, which usually is a mere formality, was declined. The bill has now been referred to the President of India and it will become law only with his assent. However, there is a precedent of a similar but less stringent bill in New Delhi that was passed by the President and this is encouraging.

The influence of the tobacco industry extends from local government to Union government. Since 1992, the Indian Tobacco Company (ITC), formerly Imperial Tobacco Company (a subsidiary of British American Tobacco), has made contributions to the Prime Minister's and Chief Minister's Relief Fund of up to 27 lakhs (2·7 million Rupees, £50 000). The ITC has also contributed generously to the Rajiv Gandhi Foundation. These are some of the ways that the industry ensures inaction and silence and buys respectability and a good corporate image for the promotion of tobacco products. Do we want our health and our children's health to be sold in this way? Nevertheless, we hope that the UK government that can so easily ban the sale of beef because of the death of 23 people since 1995,[2] can lead the world in banning the sale of tobacco, which kills 120 000 people in the UK every year.[3]

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1 Editorial. Labour Government's tobacco spin spins them off track. *Lancet* 1997; **350:** 1411.
2 Department of Health web page. http://www.coi.gov.uk/depts/GDH/coi529od.uk
3 Health Education Authority press release. Dec 10, 1997.

UK ban on beef on the bone

SIR—The advice from its scientific advisers that led the UK Government to ban the sale of beef on the bone was probably based on evidence that vertebral bone injected intracerebrally induces encephalitis in some recipients. The hazardous material was probably thought to reside in the neural tissue in the intervertebral foramina.

I wonder whether the investigators are aware of the results of experiments conducted about 60 years ago, in which a similar encephalitis was finally attributed to the presence of eosinophils in the inoculum, whatever its source?[1] Since material derived from vertebral bone may well contain bone marrow with a variable proportion of eosinophils, it seems possible that the prohibition of meat on the bone is based on experiments whose results may well be interpreted as entirely innocuous.

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1 Edward DG. Observations on the cellular basis of the Gordon test for lymphadenoma. *Lancet* 1938; i: 936–38.

Assessing the odds—the women were right

SIR—Most of the 1000 women aged between 45–64 years interviewed by the National Council on Ageing, mentioned in your Nov 29 editorial,[1] can expect to live beyond 80 years. In these circumstances, the anxieties which they expressed were surely concerned with premature death.

Examination of the published causes of death of women in England and Wales shows that 56% of the deaths among women aged 45–54 years and 48% of those aged 55–64 years are due to cancer. With increasing age, the proportion of deaths from cancer falls steeply, whereas cardiac and respiratory disease dominate as the cause of death in old age (table).

The women were, again, correct in expressing greatest concern about breast cancer since it is the most common cause of cancer death in middle age, accounting for 33% of deaths from cancer in women aged 45–54 years and for 23% of those among women aged 55–64.

For women, cancer is the most important cause of premature death and the interest of the media and the effort made in cancer research is well justified. The women were right to be concerned but they can be reassured that few women die in middle age and that many of the deaths can be avoided. Women should be encouraged to translate their anxiety to prevention, early diagnosis, and obtaining effective treatment.

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1 Editorial. Assessing the odds. *Lancet* 1997; **350:** 1563.

SIR—In your Nov 29 editorial,[1] you report that two surveys show that US women "feared breast cancer far out of proportion to the threat it poses". The blame can be traced not to the media you claim, but to the American Cancer Society (ACS). Nearly 30 years ago, the ACS, a private charity dominated by surgeons and radiologists, began to promote breast-cancer screening for all women older than 35 years.[2] The ACS was also the driving force behind the 1973 National Breast Cancer Detection Demonstration Project, which was designed to introduce the concept of mammography for symptom-free women.[3] To encourage compliance, it was necessary to raise awareness of breast cancer which is synonymous with raising fear. As any successful health charity knows, fear must be combined with hope; and early detection of breast cancer provides that hope.

In the mid-1980s, improvements in mammography prompted the ACS and the American College of Radiology to mount a more aggressive campaign to encourage mammography screening. Women, especially those under 50, responded accordingly.[4] Ironically, increased mammography acceptance inflated the incidence of breast cancer and, in turn, inflated women's fears. The much-quoted "one in nine" statistic went a long way toward frightening women into compliance. Contrary to risk communication specialist Vincent T Covello, whom you quote, the source of this statistic is the ACS,[5] not the media. The media can be faulted, however, for uncritically reporting these risk estimates. The rare exception is *The New York Times*[5] which explained that the risk accumulates late in life and that the average woman in the USA lives to the age of 79 years, though the ACS calculation assumes a longevity of 85 years.

Age group (years)	Number all deaths	Number (%) due to cancer	Number (%) due to disease of circulatory system	Number (%) due to disease of respiratory system
45–54	8840	4980 (56)	1680 (19)	430 (5)
55–64	18 310	8910 (48)	5190 (28)	1580 (9)
65–74	50 460	18 020 (36)	19 670 (39)	6420 (13)
75–84	97 320	20 570 (21)	46 590 (48)	15 400 (16)
≥85	109 350	11 240 (10)	50 360 (46)	24 430 (22)

Data from Office for National Statistics.

Cause of death in women aged 45 or older in England and Wales in 1996

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1 Editorial. Assessing the odds. *Lancet* 1997; **350;** 1563.
2 Brody JE, Holleb AI. You can fight cancer and win. New York: Quadrangle/New York Times Book Co, Inc, 1977: 65–69.
3 Greenberg DS. X-ray mammography–background to a decision. *N Engl J Med* 1976; **295:** 739–40.
4 Smigel K. Survey shows: American women favor mammography. *J Natl Cancer Inst* 1993; **85:** 439.
5 Blakeslee S. Faulty math heightens fears of breast cancer. *New York Times,* March 15, 1992.

General Practice Research Database for prescribing analysis

SIR—T Walley and A Mantgani (Oct 11, p 1097)[1] note that current prescribing analysis in the UK is limited to prescribing volumes and propose greater use of the General Practice Research Database (GPRD) in epidemiological studies. Although the GPRD contains prescribing data linked to patients' characteristics and diagnoses, Hershel Jick (Oct 11, p 1045)[2] observes that it has primarily been used to investigate drug safety issues. The major impediments to more sophisticated analyses are the size and complexity of the GPRD and the lack of specialised researchers in the UK. We are currently using the West Midlands GPRD to investigate prescribing of proton-pump inhibitors and bronchodilators. We have investigated the potential for using the GPRD in pharmacoepidemiological analyses and the challenges in exploiting the dataset.

Because of the increasing volume and cost of the prescribing of proton-pump inhibitors since 1991, we used the GPRD to investigate patients' characteristics in terms of duration and continuity of proton-pump-inhibitor