figures hold true at least 400 staff a year may experience post-traumatic stress disorder.

Some health care settings have taken steps to reduce problems in staff after incidents,[4,5] but in Britain the best example of an attempt at a co-ordinated programme may be that of the Scottish prison service. It has produced a structured response to assaults on staff, which is supported by a programme of management education.

Immediate care includes the opportunity for privacy followed by a one to one interview with a person in a senior management or counselling position to assess the effects of the assault and allow an opportunity for the victim to talk about it. This is followed by a psychological debriefing. The victim is provided with information on possible psychological effects of the assault and on how to seek help if symptoms persist. Longer term care, if needed, is provided by an experienced clinical psychologist. Care is supplemented by the involvement of management, with flexible allocation of duties. This type of care has gained widespread acceptance even in the previously "macho" culture of the prison service.

The NHS must do what it can to prevent its staff—its most important resource—experiencing similar problems to those experienced by Bende and Philpott's patient. This can best be done by acknowledging the frequency of violence and by learning from the example of other organisations and providing structured support for staff victims.

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1 Bende BC, Philpott RM. Persistent post-traumatic stress disorder. *BMJ* 1994;309:526-8. (20-27 August.)
2 Caldwell MF. Incidence of PTSD among staff victims of patient violence. *Hosp Community Psychiatry* 1992;43:838-9.
3 Scottish Health Management Efficiency Group. *Accidents in the NHS in Scotland.* Edinburgh: SHMEG, 1993. (Scotmeg project report on accidents SP54.)
4 Frema E, March S. Helping the employee victim of violence in hospitals. *Hosp Community Psychiatry* 1986;37:159-62.
5 Flannery RB, Fulton P, Tausch J, DeLoffi AY. A program to help staff cope with psychological sequelae of assaults by patients. *Hosp Community Psychiatry* 1991;42:935-8.

Generic inhalers for asthma

EDITOR,—A major drive towards generic prescribing is occurring in the NHS on the premise that generic products deliver equally effective results while yielding an appreciable saving on costs. We have reservations about the use of generic inhalers for asthma on both counts. In November last year the NHS Supplies Authority arranged a contract for generic inhalers for asthma and at the same time wrote to the chief executives of all NHS trusts, urging them to ignore approaches from their clinicians on the grounds that they may have been influenced by the manufacturers of branded products. The letter included the statement, "there is no doubt after stringent tests by our [quality control] that the generic product is equally effective with the Allen and Hanburys products." This statement must be called into question since adequate data on therapeutic equivalence are not available to clinicians.

When the Medicines Control Agency first licensed generic salbutamol and beclomethasone inhalers it required only in vitro testing of the new products and no in vivo testing. In vitro equivalence of two products does not, however, guarantee therapeutic equivalence. Accordingly, the Food and Drug Administration in the United States recommends that both the safety and efficacy of generic salbutamol inhalers should be evaluated clinically to show equivalence to the branded products. While new regulations in Britain also ask for such in vivo data, these requirements are not retrospective. Moreover, the principal in vitro test on which generic inhalers have been licensed—the twin impinger technique—simply divides the aerosol cloud into "respirable" and "non-respirable" fractions and cannot apparently detect differences in the quantity of the very fine particles that penetrate to peripheral airways.[1]

Clinical data comparing branded and generic salbutamol products are few; we know of five published studies, none of which support bioequivalence.[2,3] Similarly, there are good data on branded inhaled steroids but none on the bioequivalence of the generic products. Furthermore, anecdotal reports indicate confusion and concern on the part of patients when they receive a different inhaler simply because they have gone to a different chemist. This again frustrates good management: compliance is a major problem with patients with asthma, and anything that disturbs a patient's confidence will exacerbate this.

Generic substitution of calcium antagonists and long acting theophyllines has been stopped because of concern about dose equivalence, yet generic substitution of inhalers for asthma is being encouraged even though the appropriate studies have not been performed. Is this safe or desirable for patients? We are not against generic products but need to see adequate clinical data on exactly how the generic products compare with the branded products, with a placebo, and with each other before we are prepared to join the headlong rush that is the generic crusade.

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1 Newman SP, Kenyon CJ. Asthma products bioequivalence. *Pharm J* 1994;253:42.
2 Saarelainen P, Sovijarvi ARA. Comparison of acute bronchodilatation effects of two preparations of salbutamol aerosol in asthma. *Current Therapeutic Research* 1991;50:224-30.
3 Ruffin RE, Latimer KM, Crockett AJ, Blight MM. A comparative bronchodilator study of salbutamol and salbutamol sulphate that were administered by metered-dose inhalers. *Med J Aust* 1989;150:274-7.

Mike Pearson has given lectures at meetings sponsored by pharmaceutical companies including Allen and Hanburys, Astra, Boehringer, and Fisons and has accepted sponsorship for travel to meetings from Astra and 3M; his unit has done pharmaceutical studies for Allen and Hanburys, Astra, Fisons, Lilly, Boehringer Ingelheim, and 3M, for which all fees are paid into a charitable chest research fund.

Richard Lewis and Mike Pearson organised a meeting that Allen and Hanburys funded. Richard Lewis has received sponsorship from Allen and Hanburys to attend meetings, and the patent rights for a collapsible inhaler device that he developed were bought by Glaxo.

John Watson's research post in partially funded by Allen and Hanburys.

Jon Ayres has no conflict of interest.

Geoff Ibbotson has accepted fees for giving lectures at meetings sponsored by pharmaceutical companies.

Dermot Ryan has no conflict of interest.

David Flynn has done work for Allen and Hanburys and Glaxo and has accepted sponsorship from Astra, Serono, Ciba, and Glaxo to attend international meetings, and donations to his research fund have been made by Astra, Serono, and Glaxo; an associate has shares in Glaxo.

Jeff Williams has received payments for lecturing on respiratory medicine and has done clinical research that has been sponsored by several pharmaceutical companies, and his asthma specialist nurse is funded jointly by Allen and Hanburys and Astra.

Triglyceride concentration and coronary heart disease

EDITOR,—Despite the intuitive appeal of lowering the triglyceride concentration along with the cholesterol concentration when both are raised there is no evidence that this strategy is more effective than targeting treatment at cholesterol alone. Even treatments that raise triglyceride concentrations diminish the risk of coronary heart disease in hypercholesterolaemic subjects. In the Lipid Research Clinics coronary primary prevention trial, treatment with diet and cholestyramine resulted in a 19% reduction in the risk of coronary heart disease, despite an increase in serum triglyceride concentrations averaging 2·5%.[1] Oestrogen treatment is also associated with increased triglyceride concentrations and a reduced risk of coronary heart disease.[2] Clinical trials have not established which subgroups of hypercholesterolaemic patients, if any, benefit from a reduction in triglyceride concentrations.

A M Cruikshank notes that the rare patient with severe hypertriglyeridaemia is at risk of pancreatitis[3]; this association, however, is insufficient justification for universal screening of triglyceride concentrations. Certainly, screening could detect some hypertriglyceridaemic subjects who would otherwise be overlooked. But severe hypertriglyceridaemia in the range associated with pancreatitis is usually obvious from its physical manifestations and from visual inspection of a lipaemic blood specimen. Seldom will the result of a screening test be the sole clue to an extreme increase in triglyceride concentration.

The varied results in the literature make it possible to select studies that show an independent association between hypertriglyceridaemia and coronary heart disease, like the three that Peter H Winocour cites.[3] But many studies support the opposite conclusion.[4-6] Triglyceride concentrations may have a role in predicting risk when well standardised measurements of high density lipoprotein cholesterol concentration are not available, as Winocour suggests. The negative correlation between the two measurements means that the triglyceride concentration can serve as a surrogate for the high density lipoprotein cholesterol concentration, albeit a relatively imprecise one. For any lipoprotein test, and especially for one measuring triglycerides, its accuracy in predicting risk depends on the quality of the measurement in the specific laboratory, so clinicians should learn about the accuracy of lipid tests available in the laboratory they use.

Whether to measure the triglyceride concentration is largely a moot issue once hyper-

cholesterolaemia is found and the decision is made to measure the low density lipoprotein cholesterol concentration. Many laboratories use an indirect measurement of this which requires knowledge of the triglyceride concentration. Consistent evidence that triglycerides have an important independent role in the prevention of coronary heart disease, however, remains elusive; calls for triglyceride testing must appeal to faith rather than established fact.

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1 Lipid Research Clinics Program. The Lipid Research Clinics coronary primary prevention trial results. II. The relationship of reduction in incidence of coronary heart disease of cholesterol lowering. *JAMA* 1984;251:365-74.
2 Grady D, Rubin SM, Pettiti DB, Fox CS, Black D, Ettinger B, *et al.* Hormone therapy to prevent disease and prolong life in postmenopausal women. *Ann Intern Med* 1992;117:1016-37.
3 Triglyceride concentration and coronary heart disease [correspondence]. *BMJ* 1994;309:668. (10 September.)
4 Avins AL, Haber RJ, Hulley SB. The status of hypertriglyceridemia as a risk factor for coronary heart disease. *Clin Lab Med* 1989;9:153-68.
5 Pocock SJ, Shaper AG, Phillips AN. Concentrations of high density lipoprotein cholesterol, triglycerides, and total cholesterol in ischaemic heart disease. *BMJ* 1989;298: 998-1002.
6 Criqui H, Heiss G, Cohn R, Cowan LD, Suchindran CM, Bangdiwala S, *et al.* Plasma triglyceride level and mortality from coronary heart disease. *N Engl J Med* 1993;328:1220-5.

Excessive expenditure of income on treatments in developing countries

EDITOR,—R J Hay and colleagues draw attention to an important issue for many developing countries—namely, excessive expenditure of limited disposable income on ineffective treatments.[1] Whereas in Mexico pharmacists, private doctors, and traditional healers seem to be most to blame,[1] in China the problem permeates the entire health care system.

The introduction of a market based economy into the health care system in China since the early 1980s has meant that health professionals and hospitals have to generate most of their income, including the salaries of staff in many cases. Central directives ensure that the cost of basic medical care (for example, consultation fees and bed occupancy) are kept low while profits are made almost entirely from charging for drugs and for the use of technology.

Drugs can be charged at a mark up of 15%, which leads to massive overprescription and in particular to excessive use of injections and infusions. This results in two extremes: the 15% of the population who have some health cover (mostly state employees) and rich people are showered with often useless medicines, while many poor people are afraid to seek health care because of inability to pay for the drugs that will be prescribed. In the middle are the majority, who waste limited resources on ineffective and sometimes dangerous treatments.

Our survey of village health clinics in Zhejiang province, eastern China, in 1993 showed that for upper respiratory tract infections in children an average of four drugs were prescribed at every visit. The drugs were usually a mixture of traditional Chinese and Western treatments, the Western treatments usually being antibiotics. In township hospitals an intravenous infusion is a standard treatment for upper respiratory tract infection and fever in children. The average annual per capita income in that part of China is about 750 yuan (13 yuan = £1). We estimate the drug cost per visit to be around 15-20 yuan; this increases if injectables or infusions are used.

The current funding mechanisms mean that health practitioners and hospitals cannot survive without this source of income. The problem will become intractable unless radical steps are taken to develop alternative strategies for funding health care.

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Protection afforded by cycle helmets

EDITOR,—Frank McDermott and John Lane defy engineering evidence in stating that cycle helmets reduce the risk of serious head injury in accidents involving motor vehicles.[1] Their study, however, looked only at people who had contacted health services after being injured and included no facts on the relative risk of injury with and without a helmet. Their data do not form a valid basis for their assertion.[2] They fail to mention the work of Spaite *et al*, who studied cyclists attending a university trauma centre after being hit by cars. Both head and non-head injuries of people who had voluntarily been wearing helmets were less severe than those of people who had not been wearing helmets. Presumably people who voluntarily wore helmets behaved more cautiously in general, perhaps riding more carefully or being more likely to attend hospital after an accident.[3] This sort of confounding means that studies of voluntary helmet wearing cannot test the hypothesis of protection conferred by helmets.

McDermott and Lane argue that the results of mandatory use of helmets in Victoria, Australia, support the suggestion that helmets have a protective effect against impact from cars. In the first year of compulsory use of helmets, however, cycle use decreased by about 40% while overall deaths of non-cyclists on the roads decreased by 25%.[4] Both head and non-head injuries to cyclists decreased. We suggest that the information from Victoria is not adequate to indicate what, if any, effect compulsory use of helmets is likely to have on injury rates. Human behaviour is too confusing and complex for valid analysis in the face of insufficient numbers, inadequate information, inconstant underlying trends, and a poor scientific approach to data that are selectively quoted and potentially biased from the point of collection.[5] The published work on Victoria displays all of these problems.

Many people find cycle helmets uncomfortable and expensive. Such helmets were never designed to give adequate protection against impact with a motor vehicle. Evidence suggests that compulsory use of cycle helmets would harm health by stopping people from cycling without affecting injury rates. Cycle helmets should remain a matter for free individual choice. Roads safe for everyone are the only real solution.

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1 McDermott F, Lane J. Protection afforded by cycle helmets. *BMJ* 1994;309:877. (1 October.)
2 Keatinge R. Cycle helmets deter people from cycling. *BMJ* 1994;309:541. (20-27 August.)
3 Spaite DW, Murphy M, Criss EA, Valenzuela TD, Meislin HW. A prospective analysis of injury severity among helmeted and nonhelmeted bicyclists involved in collisions with motor vehicles. *J Trauma* 1991;31:1510-6.
4 Davis R. *Death on the streets.* Yorkshire: Leading Edge Press, 1993.
5 Noseworthy JH, Ebers GC, Vandervoort MK, Farquhar RE, Yetisir E, Roberts R. The impact of blinding on the results of a randomized placebo-controlled multiple sclerosis clinical trial. *Neurology* 1994;44:16-20.

Effects of health publicity on prevalence of smoking

EDITOR,—Joy Townsend and colleagues' study of the effects of price and health publicity on cigarette smoking used a broad definition of publicity, which may lead to misunderstandings.[1] The definition was, in effect, a measure of "everything else except price"—which, as the authors acknowledge, comprises a much wider range of influences than publicity alone.

The authors found that health publicity, as they defined it, had had relatively less influence on the smoking habits of more disadvantaged socioeconomic groups, so adding to inequality. The effect of mass communications—that is, health publicity as it is usually defined—seems, however, to depend on the medium used. For example, studies of rates of stopping smoking between 1950 and 1980 in the United States suggest that health scares in the 1950s, which were largely carried by the print media, had relatively little influence on the prevalence of smoking in more deprived groups relative to the population as a whole. These groups were much more responsive, however, to later publicity in the electronic media—especially the "fairness doctrine" antismoking campaign on television between 1967 and 1970.[2] Smokers in all social classes responded equally to Sydney's "quit for life" campaign on television in 1983.[3]

Television, which is generally watched more by members of social classes C, D, and E, is therefore a potentially class free medium for health promotion in comparison with the print media, though these probably have a correspondingly greater influence on decision makers.

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1 Townsend J, Roderick P, Cooper J. Cigarette smoking by socioeconomic group, sex, and age: effects of price, income, and health publicity. *BMJ* 1994;309:923-7. (8 October.)
2 Shopland DR, Burns DM, Samet JM, Gritz ER, eds. *Strategies to control tobacco use in the US: a blueprint for public health action in the 1990s.* Bethesda, MD: National Cancer Institute, 1991. (NIH publication No 92-3316.)
3 Macaskill P, Pierce JP, Simpson JM, Lyle DM. Mass media-led anti-smoking campaign can remove the education gap in quitting behavior. *Am J Public Health* 1992;82:96-8.

Vitamin K for neonates

EDITOR,—Mary Newburn and Rosemary Dodds discuss administration of vitamin K in relation to breast feeding.[1] Von Kries and Göbel have raised concerns about the efficacy of prophylaxis with oral vitamin K.[2] Oral administration is recommended because of the potential carcinogenic risk of parenteral administration in neonates. Surveillance data on late haemorrhagic disease of the newborn in Germany, however, suggested that the incidence of the disease increased after a switch from parenteral to oral prophylaxis. This is in sharp contrast to our data.

In the Netherlands it is recommended that all babies should be given 1 mg vitamin K orally or intramuscularly after birth and that breastfed