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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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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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-