who had developed photosensitivity insisted on continuing treatment rather than sacrifice pain relief or mobility.

The 61 deaths reported to the Committee on Safety of Medicines may underrepresent the mortality attributable to benoxaprofen but, if the widely quoted estimate that more than half a million patients have received it is correct, it is likely that at least 10 000 patients died from natural causes while taking benoxaprofen. The deaths attributed to benoxaprofen probably account for only a very small fraction of the total mortality in this predominantly elderly population.

We had already commenced a new and larger study of benoxaprofen designed to test whether or not the new instructions about the treatment of elderly patients had been effective. Two anti-arthritic agents not previously included in prescription-event monitoring had been selected as controls. The suspension of the licence for benoxaprofen halted this study, but, fortunately, we had already identified a large number of patients who started treatment last year. Less than 10% of them had been included in the pilot study, and we hope soon to be able to test various hypotheses in a large population.

To avoid disturbing the progress of 10 other drugs currently in the prescription-event monitoring pipeline we propose to introduce the expanded benoxaprofen study in one region at a time. This is not merely an academic exercise. It is as important to determine the characteristics (for example, age or diagnosis) of patients for whom the benefits may outweigh the risks as it is to confirm the extent of the risk in vulnerable groups.

I do hope that we can count on your help in this extension of the benoxaprofen study and in other prescription-event monitoring studies. Once again I would like to thank several thousand colleagues who have already helped in the pilot study.

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Chest radiography as a marker of alcoholism

SIR,—Further to the findings of Dr D R M Lindsell and others (28 August-4 September, p 597) I would like to furnish the following data. I have described the medical morbidity of 235 consecutive admissions to a detoxification centre,¹ and part of that study involved a chest x-ray examination and serum liver function tests. The detoxification centre at the University Hospital of South Manchester was opened in October 1977. A man or woman found by the police to be "drunk and incapable" or "drunk and disorderly" and known to have had similar convictions in the past could be taken by a police officer directly to the detoxification centre. Once there, all criminal charges were dropped, and the patient was immediately examined by a doctor. Of the 235 chest radiographs taken, 24 showed rib fractures and four showed clavicular fractures, giving a total of 28 (or 12%). Only one patient had any disturbance of liver function tests.

These findings may indicate that in a population of "binge" drinkers the periods of sobriety enforced either by imprisonment or by poverty serve as a protective mechanism against the development of liver damage, which often occurs in the "chronic imbibers." These results also confirm that fractures on a chest radiograph are still more common in any population of problem drinkers than in the normal population, even in the absence of liver disease.

The drinking pattern of the group I studied was loss of control "binge" drinking, which inevitably led to drunkenness. It was felt that the fractures occurred due to injuries sustained while drinking, and this was supported by discussions with the individuals.

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¹ Redmond AD. *The medical morbidity of 235 individuals admitted to Britain's first hospital based, purpose-built, detoxification centre.* Manchester: Victoria University, 1979, MD Thesis.

Diabetic complications: retinopathy

SIR,—While I wholeheartedly agree with Dr Peter Watkins (7 August, p 425) when he says that diabetic retinopathy needs to be actively sought if it is to be detected early enough to prevent blindness, I would take issue with his further statement that diabetic maculopathy should be treated when visual acuity begins to decline—that is, a decrease of two lines on the Snellen chart. This seems to me to be rather closing the stable door after the horse has bolted.

The British multicentre photocoagulation trial showed that the patients who do best from treatment with photocoagulation in relation to maculopathy are those with exudates encroaching on the macula but in whom the vision remains at 6/5 or 6/6. These patients had one eye treated and the other eye was left untreated and there were four further lines of deterioration in the untreated eye compared with the treated eye over five years. If one waits until these exudates have reduced the vision to 6/12 or 6/18 then the difference in deterioration between the two eyes is reduced to the order of two lines over five years. This evidence therefore suggests that exudative maculopathy should certainly be treated in the premaculopathy phase. In addition, I have noticed as a complication of treatment that hard exudates can enlarge immediately after treatment. If one waits until the hard exudates are poised on the edge of the macula there is therefore a greater risk of morbidity from treatment—another indication for earlier treatment.

It is essential that a situation is created in which the retinas of people suffering from diabetes mellitus are examined at regular intervals, especially once they are into the danger zone for diabetic retinopathy chronologically, so that their retinal lesions may be treated before they become symptomatic. It is often too late to treat disc new vessels once the symptoms of vitreous haemorrhage have begun to develop or maculopathy once the symptoms of reduced central visual acuity have begun to develop. This is because a patient may have the most severe form of proliferative retinopathy or the most devastating hard exudates poised on the edge of the fovea without having any symptoms or being aware of any serious problem.

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ABC of Diabetes: diabetic emergencies

SIR,—I refer to the letter of Dr O M P Jolobe (14 August, p 509) and his suggested programme for managing diabetic emergencies.

The point that insulin should be withheld until the potassium result is known is well made, but he loses my support with his "counsel of safety."

My point is that, although probably merely an oversight, it is not specified in the letter which diabetic emergency one is dealing with. Should the stated "counsel of safety" happen to be followed in a patient in a hyperosmolar, non-ketotic state the resulting hypernatraemia might be disastrous—rather more disastrous and irreversible than the hypokalaemia he fears. Especially so if one is prepared to follow Dr Watkins' regimen for the stated two hours while waiting for the electrolyte results to be phoned through, by which time two to three litres of normal saline may have been infused.

The diagnosis of the hyperosmolar aketotic state is not difficult—once it has been thought of. With dehydration making urine unavailable for testing and plasma ketostix testing by no means universal, the diagnosis might well be overlooked by the house physician.

None of the above, of course, is earth-shattering information, but I would not want a newly qualified colleague to come away from Dr Jolobe's letter with the idea that a fast saline infusion may be given with impunity to any patient with "diabetic precoma."

In practice it is unusual for the biochemist to take two hours to give the results of blood sugar, electrolytes, or even blood gas estimations. Since it seems to me that this length of time would be unlikely to be critical in the outcome, if one is to wait for two hours a safer counsel of safety might be to do absolutely nothing in the meantime.

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SIR,—Discussing with colleagues here the timing of insulin and potassium in diabetic ketoacidosis (14 August, p 509), I agree that two hours is too long to wait for a potassium result before commencing insulin. Because the vast majority of diabetics in ketoacidosis will have levels of serum potassium verging on hyperkalaemia, it is safer to commence insulin without waiting for the potassium result if one expects laboratory delays.

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The arms race and health care

SIR,—Dr D J Holdstock (7 August, p 421) has indicated the scale of current diversion of resources into armaments. A proportion of that expenditure is devoted to weapons which if used are likely to cause very extensive civilian mortality and morbidity. The BMA Board of Science will provide next year an assessment of these possible effects, but it has not been asked to comment on the ethical aspects of supporting use of weapons with mass destructive capability developed as a result of technological progress in the last few decades. In view of the nature of the destructive effects of such weapons, these ethical issues require adequate evaluation by the profession.

Although wars in the past often had adverse and at times devastating effects on civilians, in general it could have been argued that maintaining the integrity of a nation state and safeguarding its resources by armed conflict served the interests

of the majority of the population and hence the people for whom doctors had medical responsibility. When military policies involve deliberate and premeditated mass destruction of an enemy population, however, and when both sides share such capability the issues are radically altered. Any doctor who advocates or supports such use of weapons must assume some responsibility not only for the possible death and suffering of many civilians in other nation states but also for the destruction of his own patients and their resources. It should also perhaps be taken into account that the forms of death and morbidity for which he is potentially responsible are probably as appalling as it is possible to imagine, and that many of those on whom such suffering is to be inflicted, particularly children, cannot be held in any way responsible for the course of events.

Those who accept the arguments for deterrence clearly indicate their willingness to use the weapons involved: if this were not the case the deterrent effect would automatically be invalidated. Such support for use of weapons of mass destruction is by itself worthy of ethical evaluation but must be balanced by a proper consideration of the deterrence position. Since they are unlikely to wish to be responsible for destructive effects of weapons far exceeding any in history, those who support deterrence must rely on its effectiveness. If it were guaranteed to be entirely effective in preventing war, deterrent use of weapons would be of major value to mankind and probably worthy of medical support, provided that adverse effects due to the production and deployment of the weapons could be avoided.

In the event that a doctor believes that there is a significant risk that deterrence by implied mass destruction will cease to be effective, however, his continued support for such a policy directly contravenes the principles of preservation of life and prevention of suffering represented in the Hippocratic Oath and more recent statements of medical ethics.¹

Those who believe that deterrence will be entirely effective are almost undoubtedly in a minority at the present time, and may have failed to note both the alternative judgments of numerous informed opinions and the possible implications of some recent military developments. Since reliable data on which to base an adequate risk analysis concerning the course of future events are unavailable, a significant likelihood that use of weapons of mass destruction will occur cannot be excluded. It would therefore appear to be unethical to support deterrent use of such weapons.

A further contingency is that, as argued recently by Zuckermann,^{2,3} deterrence between the major powers with nuclear weapons was implemented early in the decade 1960-70, and that some military developments since that time may have increased the likelihood that deterrence will break down.

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¹ *The Handbook of Medical Ethics*. London: BMA, 1981.

² Zuckermann S. *Proc Am Phil Soc* 1980;124:1-14.

³ Zuckermann S. *Nuclear Illusion and Reality*. London: Collins, 1982.

Cigarette consumption and biochemical measures of smoke intake

SIR,—In criticising (26 June, p 1951) the statistical studies by Mr C J Vesey and others (22 May, p 1516) on carboxyhaemoglobin and plasma thiocyanate in smokers, I was not arguing that studies of blood levels of tobacco or dietary components are of little predictive value for epidemiologists. Indeed, I have for some time advocated the wider use of biochemical measurements to help predict the future onset rate of whatever disease is of interest, and some of the work in which I am

currently engaged is based entirely on this. But, to be of good predictive value, biochemical measures must be reasonably reproducible. To pursue the example of blood cholesterol and other lipids introduced by Dr Michael Russell in the replies from him and from his colleagues (14 August, p 507) to my criticisms, preliminary analyses of a few dozen individuals whose blood was drawn both in 1972 and in 1976 have shown that plasma cholesterol was reasonably reproducible (people who were high in 1972 tending to be high in 1976 as well), but that the plasma free fatty-acid profile was not (indeed, the 1972 values were scarcely correlated at all with the 1976 values), presumably because the recent intake of fatty acids is so variable from day to day.

This suggests that although a single plasma cholesterol can usefully characterise an individual, a single measurement of an individual's plasma free fatty acid profile cannot. Thus, although dietary fatty acid intake directly affects plasma fatty acid profile, correlation of the results of a 1972 survey of such profiles with subsequent disease onset rates is unlikely to be of much help to epidemiologists who want to study the health effects of fatty acid intake. This difficulty cannot be circumvented by discovering that in the 1972 survey one fatty acid is correlated with another: what chiefly matters for prediction of chronic disease is whether the 1972 values characterise an individual, and the critical test of this is the extent to which the 1972 and the 1976 values are correlated with each other.

In the light of such considerations, I objected to the claim, made without any proper discussion of long-term reproducibility, that carboxyhaemoglobin and thiocyanate had been shown to be: "Superior to self-reported cigarette consumption as an index of exposure to tobacco smoke," and I cannot agree with the authors' replies that: "The issue of stability over time is a secondary one," nor that their statistical analysis is "valid," if by "valid" they mean that it demonstrates their stated conclusions.

I hope their conclusions are true, of course, because I too would greatly like some measures of tobacco exposure to be established as being as or more reliable than self-reported consumption, and I would strongly support their final suggestion that biochemical and questionnaire information could be complementary. Such complementarity may, for example, be especially important for lung cancer, since this is a disease that originates chiefly from the cells of the main airways, and the determinants of exactly what is deposited in them as the cigarette smoke rushes past are so complicated that, among heavy smokers, those who describe themselves as "inhalers" may even be at lesser risk than smokers of similar amounts who because they inhale more slowly do not describe themselves as "inhalers."¹

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¹ Doll R, Peto R. *Br Med J* 1976;ii:1525-6.

Nicotine chewing-gum

SIR,—The excellent results of the double-blind trial of nicotine chewing-gum of Mr M J Jarvis and colleagues (21 August, p 537) should lead to its more widespread use especially for

intervention in "at risk" smokers by general practitioners. My ongoing study shows the rewards of a stop-smoking clinic in general practice and reaffirms that patients should be encouraged to use the gum over a longer period of time.

Following earlier suggestions made by the London group, attention was paid to careful instruction on chewing technique, completion of smoking diaries, the offer of a substitute for hand inactivity, and, especially, regular follow up. A hundred patients, whose cigarette consumption exceeded 15/day and with either a pre-existing or a predisposition to a smoking-related disease were given a supply of Nicorette provided they expressed a sincere desire to give up smoking. After two, four, six, 12, and 26 weeks the number abstinent were 37, 51, 38, 29, and 28 respectively.

A full six months free of all cigarettes was verified by expired-air carbon monoxide measurement in 28. Of these long-term successes, 24 had used Nicorette for a minimum of three months. Preliminary findings also suggest that a six-day gum-acclimatisation programme before complete smoking withdrawal improves the acceptability and efficacy of the product. Sixty-two per cent of patients who were successful received this approach. A control group were advised to substitute all cigarettes with gum from the outset.

Assessment is continuing up to 12 months and will be reported in due course.

R F HYDE

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SIR,—In concluding that nicotine chewing-gum is the first treatment for smokers that "has a specific effect over and above that attributable to an attention-placebo response," Mr M J Jarvis and his colleagues (21 August, p 537) showed an extraordinary neglect of the recent publications on psychological treatment. When several behavioural treatment methods are combined into "packages," abstinence rates of 33-76% have been reported at six months' follow-up¹⁻⁹ and 32-46%^{10,11} at one year. These results are clearly superior to the frequently cited attention-placebo effects. There is more than one effective way of helping people to stop smoking.

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¹ Best JA, Bass F, Owen LE. *Can J Public Health* 1977;68:469-73.

² Chapman RF, Smith JW, Layden TA. *Behav Res Ther* 1971;9:255-64.

³ Delahunt J, Curran JP. *J Consult Clin Psychol* 1976;44:1002-7.

⁴ Elliott CH, Denney DR. *J Consult Clin Psychol* 1978;46:1330-9.

⁵ Hamilton SB, Bornstein PH. *J Consult Clin Psychol* 1979;47:598-600.

⁶ Lando HA. *J Consult Clin Psychol* 1977;45:361-6.

⁷ Lando HA, McCullough JA. *J Consult Clin Psychol* 1978;46:1583-5.

⁸ Paxton R. *Behav Res Ther* 1980;18:45-50.

⁹ Paxton R. *Behav Res Ther* 1981;19:117-23.

¹⁰ Lando HA. *Addict Behav* 1981;6:123-33.

¹¹ Pomerleau O, Adkins D, Pertschuk M. *Addict Behav* 1978;3:65-70.

SIR,—The report by Mr M J Jarvis and others (21 August, p 537) of a carefully planned trial at the Maudsley Hospital might lead practitioners to conclude that the most effective help they can give to patients wishing to stop smoking is to prescribe nicotine chewing gum. While any adjunct which leads to an abstinence