are) are the only measure available. Moreover, croup scores are useful in hospital clinical practice. As most croup presents outside normal working hours, croup scores are useful to junior paediatricians as a reminder of assessment criteria and as a measure of severity.

M Cronin and R Diedericks express concern that I did not refer to nebulised adrenaline. As the title of my editorial made clear, my primary aim was to draw attention to the role of corticosteroids in croup. The one trial comparing nebulised adrenaline with nebulised corticosteroids showed broadly similar effects, although, as it is reported only in abstract form, interpretation must be limited (D Fitzgerald *et al*, meeting of Australian Thoracic Society, March 1995). Although nebulised adrenaline clearly has a role in the management of croup, I believe that it has no role in mild to moderate croup and (with rare exceptions) should not be used in the community. The role of nebulised adrenaline is to avert or delay the need for intubation, and consequently it should be used only under controlled conditions. Relapse after the use of nebulised adrenaline is well described—indeed, in the study that Cronin and Kiedericks cite, by Kristjansson *et al*, over a third of the children receiving adrenaline relapsed, and the authors recommended that children receiving adrenaline should be observed for two hours. I believe that children who require nebulised adrenaline should be observed where facilities are available for urgent intubation should the need arise.

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Deaths of children in house fires

EDITOR,—Ian Roberts's editorial on the deaths of children in house fires omits to mention what is overwhelmingly the most important factor in fatal residential fires—namely, cigarettes.¹ "Smokers' materials" are the source of ignition in nearly half of the house fires in Britain that result in a death. Matches are not included in the government's definition of this term, and in fatal residential fires the term essentially refers to cigarettes. Any discussion of deaths among children because of house fires ought therefore to mention cigarette smoking by members of the victims' households.

In 1994, at a national conference on fatal house fires in Scotland, Professor Timothy Squires noted that "over 40% to almost 50%" of such fires were caused by cigarettes. Now, against the background of an overall decrease in the rate of deaths in fires in the two ensuing years, that percentage has risen to 54% in Scotland.

Recently a scientist who used to be the vice president in charge of research and development for a major cigarette manufacturer in the United States went on public record as saying that the tobacco industry has known "for at least 30 years" how to produce what are popularly called "fire safe cigarettes" (ordinary cigarettes with a drastically reduced propensity to result in a fire).²

Because of requests from the Association for Nonsmokers' Rights, in July 1994 the government released data on the number of children (aged 16 and under) killed in fires caused by smokers' materials in the United Kingdom in 1988-92. The statistical tables (provided by the Home Office's fire statistics section) showed that, on average, 24 children a year died in house fires caused by smokers' materials. Over 90% of such deaths could be avoided if fire safe cigarettes were sold.³

There are many other reasons, too, why cigarette smoking should be taken into consideration when

physical harm to children is being discussed. The chewing and swallowing of cigarettes by toddlers accounts for about 15% of unintentional poisonings of children in Greece. In two thirds of the cases studied both parents were smokers, and in 93% of cases at least one parent was a smoker.⁴ Add to this the effects of smoking during pregnancy and of passive smoking by children⁵ (as well as the effect of smoking on the budgetary constraints of families) and it becomes clear that adults' cigarette smoking should be one of the first factors to be considered by scientists discussing the physical harm done to children in our society.

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High levels of polyunsaturated fat may inhibit cancer growth

EDITOR,—Mahmoud Zureik and colleagues report a striking reduction in cancers of all types in association with high levels of polyunsaturated fat in blood cholesterol esters.¹ They reject a causal hypothesis, partly because the inverse association of cancer with polyunsaturated fat is not site specific.

This rejection of a causal hypothesis may be premature. In 1956 Sinclair suggested that high levels of polyunsaturated fat might protect against a range of cancers.² In the past decade investigators from many laboratories have reported that concentrations of polyunsaturated fatty acids that do not harm normal cells inhibit the growth of, or kill, over 40 human cancer cell lines.^{3,4} Thus a plausible biological basis exists for an inhibitory effect of high levels of polyunsaturates on cancer growth.

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Acoustic neuroma may impair vision

EDITOR,—A Wright and R Bradford's review of the management of acoustic neuroma omits the importance of corneal health and the visual consequences of ignoring early signs of corneal disease.¹ Patients with acoustic neuroma may have three factors leading to corneal compromise: decreased

corneal sensation preoperatively, postoperative facial palsy with associated poor lid closure, and decreased vasomotor tone to the lacrimal gland after seventh nerve palsy. Thus they often have an unprotected, dry eye devoid of sensation.

We have recently reviewed the records of patients who have had conventional surgery (excluding stereotactic radiosurgery) for acoustic neuroma in Leeds and Sheffield. Of 132 patients, 62 had documented involvement of the fifth nerve preoperatively. Eighty five had facial palsy in the postoperative period. Fifty three patients required definitive lid surgery (lateral tarsorrhaphy, central tarsorrhaphy, or medial canthoplasty). Thirteen patients developed corneal vascularisation, stromal opacities, or frank ulceration. A further three had visual impairment as a result of optic atrophy.

The anterior segment disease can be prevented by several prophylactic measures. Firstly, air may be injected into the subcutaneous tissues of the upper lid to produce a mechanical ptosis, which will last for a few days until it is clear whether facial nerve function is preserved. Secondly, preoperative tarsorrhaphy with histoacryl glue (superglue) will provide 10-14 days' cover. This should not be done by inexperienced doctors since glue can seep backwards into the interpalpebral fissure, form a plaque, and abrade the cornea. Superglue can also be reapplied postoperatively if there is a transient facial paresis that seems to be resolving. Thirdly, botulinum toxin can be used to produce a ptosis that will usually last for about three months. This is not risk free since the Bell's phenomenon may be abolished and permanent vertical diplopia may occur.

We recommend early (even preoperative) referral to an ophthalmologist and the use of air or superglue in the immediate perioperative period until the patient is fit enough for full slit lamp examination. Ocular lubricants should be used freely and frequently. Patients should be told preoperatively that surgery for an acoustic neuroma can potentially impair vision in the ipsilateral eye.

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Plasma cholesterol response to dietary saturated fat

Conclusions are based on circular arguments

EDITOR,—Charlotte Cox and colleagues report a double crossover trial looking at individual variation in the response of plasma cholesterol concentrations to dietary saturated fat.¹ Responsiveness to diet was calculated for each subject as the difference between total cholesterol concentration during two types of diet. Consistent responders were then defined as those in whom the difference between the two differences was within one standard deviation of the mean for all participants; the remainder were defined as variable responders. Not surprisingly, the authors found subjects who fulfilled their definition, which led them to conclude that, indeed, a group of consistent responders exists. Their tautological and circular arguments then continue.

Firstly, the authors note a "striking" correlation in the changes between the first and second crossover periods. We would have expected this: it merely confirms the definition of consistency in response. The authors then postulate the existence

of two subgroups within the group of consistent responders: hyperresponders and minimal responders. Again, a significant difference in total cholesterol between the subgroups (table III in their paper) only confirms the definition.

Secondly, a significant difference in baseline characteristics such as cholesterol ester transfer activity and total cholesterol (table IV in the paper) could be due entirely to regression to the mean.^{2,3} Here, a hyperresponse or a minimal response (whether consistent or not) would be determined by the initial cholesterol values.

Thirdly, the same baseline characteristics show a significant correlation with the mean change in total cholesterol for consistent responders (table V in the paper). This can be explained by the difference between hyperresponders and minimal responders. These variables, therefore, are not predictors of response but determinants of the subgroups. With this step the argument comes full circle. It remains unclear whether any biological evidence can be derived from this circulation.

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Authors' reply

EDITOR,—Susanne Doepfner and Irene Guggenmoos-Holzmann criticise our approach, which included reporting correlations between changes in cholesterol concentrations during the two diets in the subjects defined as consistent responders. They do not mention, however, that we also found a significant correlation between changes in cholesterol when data for the whole population were considered. Given that the study was conducted among free living subjects, we consider that the correlations provide reasonable evidence for an overall consistency in response.

Doepfner and Guggenmoos-Holzmann further believe that regression to the mean is likely to explain our observations that certain variables measured at baseline differ in hyperresponders and minimal responders and that these variables may partly predict response. They seem to have overlooked our finding that variable responders showed smaller changes in plasma cholesterol during both diets than did hyperresponders (table III in our paper) while mean cholesterol concentrations were similar in the two groups, suggesting a similar degree of regression to the mean. Furthermore, regression to the mean is likely to have been reduced by the fact that one measurement of cholesterol was made at the time of screening to select subjects for the study and a second measurement served as the baseline value for the study.¹ In addition, we measured plasma cholesterol at four and six weeks during each diet. Values were similar and were therefore averaged. Thus we believe that there is little to suggest that some of our main findings "could be due entirely to regression to the mean."

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Effect of detection of depression in general practice

Authors do not give enough information

EDITOR,—Christopher Dowrick and Iain Buchan¹ have shown what Hoepfer *et al* showed some years ago²: that telling a general practitioner that a patient has a high score on a screening questionnaire does the patient no good. It is difficult to see why it should. I find it strange that the authors do not think it necessary to show what the doctors did for the patients in the three groups (those with conspicuous depression, detected depression, and undetected depression), although in any other branch of medicine the course of a patient's illness might be expected to bear some relation to interventions. It is hardly surprising that patients whom the doctors intended to treat did less well than those whom the doctors did not intend to treat, since they might be expected to have more severe depressive illnesses.

We are not told how good these particular doctors were at detecting depressive illnesses: possibly most of the severe depressions were being treated. We are told (and it is not that reassuring) that 24% of the subjects did not respond to the initial questionnaire and that a further 32% did not respond to follow up, but we are not told whether the default rates were equal between the groups. Most worrying of all, the depressions included in the study were prevalent rather than incident depressions, yet a treatment effect would be expected to be maximal in the latter.

The issue that Dowrick and Buchan deal with is important, and it is therefore important that experiments are carefully designed and that careful note is made of how patients in whom a depressive illness was detected were treated. A study needs to contain enough patients receiving treatment for depression to show a treatment effect.

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Patients may be receiving inadequate treatment

EDITOR,—Christopher Dowrick and Iain Buchan suggest that neither diagnosis nor disclosure of depression in general practice has an appreciable impact on outcome and that detection should be seen simply as a marker of severity.¹ It is well recognised that there is appreciable unidentified psychiatric morbidity in primary care,² and Dowrick and Buchan's paper reinforces this point. Two other findings that might have had an impact on outcome, however, are not highlighted. These are, firstly, adequacy of treatment and, secondly, non-attendance at follow up appointments.

It is a matter of some concern that the authors' data suggest that even when morbidity is conspicuous and an intention to treat exists, inadequate treatment is offered. This is particularly important if conspicuous morbidity has a worse prognosis in the long term.¹

Unsurprisingly, a proportion of the subjects in the study subsequently failed to attend. It is interesting that this proportion varied across the three groups studied: the proportion who completed all three questionnaires was 71% in those whose depression was undiagnosed and undisclosed, 63% in those with undiagnosed depression that was subsequently disclosed, and 56% in those with diagnosed depression. Thus the group who do worst are the group least likely to attend subsequently. Possibly this is another

reflection of severity of illness. A second possibility is that these drop out rates are themselves a function of inadequate treatment. It has been suggested that low attrition rates may be associated with effective care.³ Might the higher attrition rates and poorer outcome in this group be associated with ineffective care?

This valuable survey has potentially important implications for the treatment of depression in primary care, especially if inadequate and ineffective treatment is being offered to those who subsequently fail to attend. The paper offers useful information to those campaigning to defeat depression.

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Study had methodological limitations

EDITOR,—Christopher Dowrick and Iain Buchan report a trial to assess whether the outcome of depression is influenced by diagnosis by the general practitioner or by disclosure of the results of screening in cases of undetected depression.¹ They conclude that disclosure did not improve prognosis at six and 12 months and that treatment of depression in primary care has little influence on the "course of a condition whose major determinants may lie outside the reach of the medical profession." We believe that this is an unnecessarily gloomy conclusion and reinforces a nihilistic attitude towards the treatment of depression and other psychiatric disorders that is commonly seen in primary care.

The authors did not consider important methodological limitations. The sample was small, so the possibility of a type II error was high. The analysis was not based on intention to treat, whereby all randomised subjects are included in the analysis and if some are lost to follow up then missing values are replaced by the last available figure. The worse outcome among those patients for whom general practitioners declared an intention to treat was confounded by the severity of depressive symptoms found in the non-randomised part of the study. Follow up assessments took place at only six and 12 months; disclosure may have a maximal effect at six weeks.² The authors do not distinguish what they call intention to treat between the experimental and control groups. Such a comparison might provide information on whether general practitioners were more inclined to do something for those patients whose unrecognised condition was disclosed. Simple disclosure of a depression score may be unhelpful for many general practitioners. More detailed feedback, with a profile of symptoms and diagnostic information, seems to affect the outcome of depression at six weeks in primary care.²

The authors note that even when general practitioners prescribed antidepressants the treatment did not always accord with the guidelines in the *British National Formulary* even though the general practitioners professed knowledge about these. The provision of guidelines for treatment together with the disclosure of a clinical diagnosis might be a more effective way to influence the outcome of unrecognised depression. When compared with patients with depression identified by general practitioners, patients with unrecognised depression have less severe symptoms and impairments in social functioning and are less likely to have