that the death rate from ischaemic heart disease increases with age but claim that the rate of increase does not accelerate after the menopause and argue therefrom that there is strong reason to reject the idea that any protection is lost.

I have plotted semilogarithmically the death rates from ischaemic heart disease for both sexes¹ and for comparison have plotted the rates for pneumonia. The curves of the death rates for ischaemic heart disease show, as has been clearly shown before, a greater rate of increase in women, so that their death rate steadily approaches that of men. In contrast, the death rates for pneumonia have curves of identical shape throughout the whole adult lifespan, with no tendency for the ratio between the death rates of the two sexes to change with age.

Your argument that the absence of any marked irregularity in the curve of female death rates from ischaemic heart disease indicates the absence of any additional risk after the menopause is weak. Of course there is no sudden dramatic metabolic change coinciding with the menopause, which is merely the most obvious event in the gradual change of the climacteric. Moreover, the effects of that gradual process are spread out over an even larger number of age groups by the wide range of ages at which the menopause occurs. Attempts to analyse symptoms of the climacteric in terms of chronological age give a confusing picture which, however, becomes clear when patients are grouped according to their biological age—that is, by using the onset of irregular periods and the menopause as datum points.² Again, death from ischaemic heart disease does not occur at a fixed time after the onset of the disease or correlate closely with its severity and must therefore be a somewhat inaccurate indicator of the general status of the coronary arteries.

An equally serious criticism of your article is that it refers to "strong evidence that taking hormones in the form of oral contraceptives increases the risk of developing coronary heart disease" and goes on to say that it is surely "most unlikely that the same hormones could both protect from and predispose to the same disease." I have previously drawn attention in your own pages³ to the fallacious nature of such simplistic arguments, which are based on the assumption that oestrogens are, for all practical purposes, indistinguishable. It is strange that many doctors who would at once appreciate the absurdity of making sweeping generalisations about the side effects of all antibiotics unquestioningly accept such generalisations about all oestrogens in the face of abundant evidence that oestrogens are as diverse in their pharmacological profiles as are other classes of drugs. By far the major part of postmenopausal oestrogen replacement therapy makes use of oestrogens more closely resembling the ovaries' own products than the synthetic oestrogens used in oral contraception, and epidemiological evidence derived from oral contraception should not be used to infer the consequences of postmenopausal oestrogen replacement therapy.

Finally, although we are not, in fact, talking about the same hormones, there would be nothing paradoxical, as you think, in the idea that the same hormones could both protect from and predispose to the same disease. The mechanism by which oral contraceptives are alleged to increase the incidence of thrombosis is an elevation of blood coagulation factors, whereas the beneficial effect of oestrogens is

ascribed to the lowering of plasma lipids and consequent protection against atherosclerosis.

PATRICK BYE
Senior Medical Adviser,
Pharmaceutical Division,
Schering Chemicals Ltd

Burgess Hill, W Sussex

¹ *Mortality Statistics for England and Wales 1974*, Series DH2, No 1. London, HMSO, 1977.

² Jaszmann, L, et al, *Medical Gynaecology, Andrology and Sociology*, 1969, 4, 268.

³ Bye, P, *British Medical Journal*, 1973, 4, 354.

Useless warning

SIR,—The so-called strengthening of the health warnings on cigarette packets agreed by Mr Ennals with the tobacco industry has done nothing to alter the fact that these warnings are likely to have the opposite effect from that intended by the Government.

Notices on packets of cigarettes will now read "Government health warning: smoking can seriously damage your health." This is a half-truth because smoking is invariably damaging to health—witness the fact that top athletes, with very few exceptions, do not smoke. But it is in the nature of man to assume that when he takes a calculated risk he is not going to be one of those who will suffer. Therefore to imply that there is only a risk of damaging health is unlikely to have any deterrent effect. It is no more effective than a street notice declaring "Crossing this road can result in serious injury." Even if the Minister strengthens the warnings to the point of threatening death it will be of no avail so long as there is seen to be an element of risk rather than certainty.

Even more counterproductive is the gratuitous statement that the warning is *Government-inspired*. It is another basic instinct of man to distrust those who govern him. So to describe the warning as coming from the Government is calculated to debase the warning in the mind of the average man. In any event it invites the challenge, "What does the Government know about health? What authority has a bunch of politicians to advise on health matters?" None, of course, so why is it not described as a "Doctors' health warning" or just "Health warning?"

One can only speculate as to who suggested the wording of the warnings. The tobacco industry's sponsorship of sport and the fresh-air image of many cigarette advertisements suggest that it was the tobacco barons, armed with an elementary knowledge of human nature, who induced the Government to agree to warnings which at best could have little or no effect and at worst could actually encourage smoking.

A W FOWLER

Bridgend General Hospital,
Bridgend, Mid Glamorgan

Breath, alcohol, and the law

SIR,—Dr T C Beard's suggestion (16 April, p 1033) that we should adopt the resolution of the 7th International Conference on Alcohol, Drugs, and Traffic Safety and express alcohol concentrations in breath units is surely somewhat illogical. The paper by Dr T A A Alobaidi and others (18 December 1977, p 1479) that he refers to concludes that "breath analysis is not an acceptable method for

accurately determining blood alcohol concentrations" and that "a reasonable guess" [at the breath: blood ratio] should not be used to obtain a conviction in a court of law." To conclude from this that, since the two cannot be got to agree, we should abandon blood in favour of breath involves the assumption that, where they differ, breath is at least as likely to be right.

The paper by Dr Alobaidi and his colleagues is an unfortunate starting point for this discussion because not only are their results much worse than those obtained in recent years by a number of reputable observers, but they give a picture of the present understanding of the subject that is very out of date. The remark they quote from Enticknap and Wright¹ that a breath: blood ratio of 1:2370 "seems a reasonable guess" was not only made 10 years ago but was taken out of context because we went on to say that "the only proper conclusion, however, based on the available evidence, is that the breath: blood ratio is not known within $\pm 10\%$ and can only be determined empirically under operational conditions," and this is still true 10 years later.

Since then the findings of a detailed study at the University of Wales Institute of Science and Technology (UWIST)² have shown that the "guess" of 2370:1 was very close to the mark and that using a 2300:1 ratio under carefully controlled laboratory conditions, with very precise analytical methods for both breath and blood and fully co-operative subjects, the blood alcohol concentration (BAC) can be predicted by breath analysis with standard deviation (SD) of 3.4 mg. An equally careful study by Dubowski³ recently came to a similar conclusion.

Valuable as this basic information is, what really matters is what happens under operational conditions—that is, when breath samples are taken by policemen in police stations and compared with blood samples taken at the same time, and this has not yet been done. The Blennerhassett Committee's report⁴ called for "accuracy within $\pm 10\%$ at the 95% confidence level"—that is, an SD of 5 mg/100 ml at 100 mg/100 ml. It is inconceivable that an SD as low as this could be attained under operational conditions. A level of 10 mg/100 ml is possible, but a figure of 15 mg/100 ml is more likely. If a conviction is to be based on one breath sample and the same criteria are to be used as for blood—that is, an allowance of 3 SDs—the 80 mg/100 ml limit, which is now, in practice, 86 mg/100 ml, would therefore become 110 mg/100 ml or more, depending on the magnitude of the variance that was found. It is true that the BACs of most arrested persons are so much higher than this⁵ that it will make no difference to them, but all the arguments about the accuracy of alcohol tests are not about these people but about those close to the limit, whose lives may be ruined by a wrongful conviction. In addition, Parliament fixed a limit of 80 mg/100 ml after a great deal of research and discussion and to raise it to 110 mg/100 ml or more is a step not to be taken without careful consideration.

Before, therefore, we consider making the change proposed by Dr Beard we need to determine the real precision and accuracy of breath analysis when compared with blood under operational conditions and to investigate the source of the variations in the breath: blood ratio and eliminate them as far as possible.

The first can be carried out very simply

by following the Blennerhassett recommendation that we should adopt the procedure, used in Northern Ireland for the past nine years, of screening with a reasonably accurate breath device and allowing the accused to plead guilty to the result if he wished or to have a blood test if he did not. Provided a breath test was taken at the same time as the blood, the real operational reliability of breath tests would very soon be established. Judging by the experience in Northern Ireland⁶ this procedure would save 90% of the time and labour at present devoted to blood analysis in forensic laboratories, so there should be plenty of time and labour available to investigate the source of variation between breath and blood. The UWIST studies have already indicated that the main source lies in the fact that, owing to its high solubility and low concentration, there is a large "physiological" dead space for alcohol in the upper respiratory tract and that it is variations in this dead space that are mainly responsible for variations in the breath: blood ratio. What is now needed is a study of the factors influencing the properties of the dead space.

If, after due consideration, it is decided to accept the errors of breath analysis I hope that its potentialities will be fully exploited. A number of not very accurate breath tests may well give a better picture of the tissue alcohol level than an analysis, however accurate, of a single blood sample. But to replace one blood sample with one breath sample, which seems to be the practice in Australia and other countries where blood has been abandoned, however convenient for lawyers and police, is much worse than substituting an SD of 10 mg/100 ml for one of 2 mg/100 ml. The precision of blood analysis is known and ensured because the analysis is duplicated, but the precision of a single breath sample cannot be determined and no statistician would be content with data based on such an observation.

B M WRIGHT

Clinical Research Centre
Harrow, Middx

¹ Enticknap, J E, and Wright, B M, *Proceedings of the 4th International Conference on Alcohol and Traffic Safety*, p 161. Bloomington, Indiana, Indiana University, 1966.

² Jones, A W, Wright, B M, and Jones, T P, *Proceedings of the 6th International Conference on Alcohol and Traffic Safety*, p 509. Toronto, Addiction Research Foundation, 1975.

³ Dubowski, K M, *Clinical Chemistry*, 1974, 20, 294.

⁴ Department of the Environment, *Drinking and Driving, Report of the Departmental Committee*. London, HMSO, 1976.

⁵ Klein, D, *Human Factors*, 1976, 18, 211.

⁶ Howard, A L, and Morgan, W H D, *Proceedings of the 5th International Conference on Alcohol and Traffic Safety*, p 53. Freiburg. Schulz Verlag, 1969.

Night visiting by general practitioners

SIR,—The article by Mr M J Buxton and others (26 March, p 827) raises some interesting questions. As the section "Implications for policy" demonstrates, this paper has a political intent. Before attempting to influence policy it is important to examine the quality of the evidence presented.

The authors have tended to dismiss much published evidence on the grounds that it may be "misleading, atypical information based on the experience of a handful of practices." Instead they have attempted to infer the rate of night visits from data on the number of fees claimed. How valid are their assumptions? In Table I they infer that there

has been an increase of approximately 135% in the night-visiting rate per 1000 patients between 1967-8 and 1975-6. This figure depends on their assumption that there are as many visits between 2300 and 2400 as there are in any other hourly period during the night. This is a surprising assumption, as the authors quote in their paper and in the source document¹ setting out their reasoning more fully the work of Brotherston *et al*² and Crowe *et al*.³ Both these papers show a higher number of visits between 2300 and 2400 than in hourly periods in the early morning. Indeed, in their source document Mr Buxton and his colleagues present the implied night visiting rates per 1000 patients assuming that the findings of Crowe *et al* on the timing of calls are valid across the country. Using these assumptions, the implied night-visiting rates increased between 1967-8 and 1975-6 by 60%. One can only speculate why the authors chose to present the figures they did in the article as they provide no reasons for their preference in the article or in their source document. As the authors acknowledge in their paper, interpretation of this apparent increase requires caution. We do not know if night-visiting rates are increasing or if a greater percentage of claims are now being submitted.

Having gathered data one must be careful in their interpretation, as assumptions at this stage may be equally invalid. In the concluding paragraphs, Mr Buxton and his colleagues write: "If the rate of night visiting is seen as a measure of the care provided to the patient then the continuation of deputising services would improve performance in terms of this indicator." This may be so. An alternative explanation could be that any increase in night-visiting rates could be accounted for by patients summoning an "emergency doctor" to deal with a problem which has existed for some time because for one reason or another it has not been dealt with earlier, perhaps because the patient was unable to make an appointment with his own doctor. In this case an increase in night-visiting rates would indicate a decreased performance of medical services.

The authors have provided much food for thought and for this they deserve congratulations. Their article underlines the necessity for further studies on this fascinating topic, which one hopes will be better designed and validated. If and when this is done there will be a firmer basis for discussions on policy.

IAIN GILCHRIST

Bishop's Stortford, Herts

¹ Buxton, M J, and Sayers, J, *An Analysis of GP Night Visiting Rates*. London, Centre for Studies in Social Policy, 1976.

² Brotherston, J H F, *et al*, *British Medical Journal*, 1959, 2, 1169.

³ Crowe, M G F, Hurwood, D S, and Taylor, R W, *British Medical Journal*, 1976, 1, 1582.

*.*Dr Gilchrist sent a copy of this letter to Mr Buxton and his colleagues, whose reply is printed below.—ED, *BMJ*.

SIR,—Dr Gilchrist's comments rightly emphasise that the change over time in the implied night-visiting rates depends on a variety of assumptions. It is precisely because we agree with him that our paper stressed the upward trend rather than a particular figure; there is an upward trend whatever the assumptions made. Indeed, given that—as we stressed in the paper—nothing is known about changes in

the propensity to claim, it would have been extremely foolish to make any point that relied on a particular percentage increase.

Permutating the various assumptions involved in calculating the implied visiting rates produces estimated increases, as our background paper shows, between approximately 60% and 135%. The difference between these two figures reflects alternative assumptions not only about the correction for the extension of eligible hours but also about the effects of delays in making payments. The figures given in the published table, from which Dr. Gilchrist has derived the 135%, were chosen for two reasons. Firstly, the assumption about shorter delays in payments yielded a relatively smooth upward curve, rather than an implausible hiccup in the first year. Secondly, the assumption about the pro-rata effect of the extension of eligible hours seemed reasonable to us, given that changing the hours may well have altered the pattern of visiting. We would indeed welcome more evidence as to the validity of these assumptions, but it must be stressed that no adjustments were necessary for the 1973-4 figures used in the main analysis of geographical variations.

With respect to Dr Gilchrist's interpretation of the evidence, he may well be right in his suggestion that patients now find it easier to get a home visit from an "emergency doctor" than from their general practitioner. But if so, then surely the deputising services are performing a useful role; from the patient's point of view, better a night visit than no visit at all.

M J BUXTON
R E KLEIN

Centre for Studies in Social Policy,
London WC1

New strategies for drug monitoring

SIR,—Your leading article on this subject (2 April, p 861), highlights some problems associated with recent proposals, in particular the choice of suitable controls to compare with index patients on new drugs and difficulties in interpreting the incidence rates of a particular condition among users of a drug without knowing the incidence measured by the same methods among similar people who had not received the drug. Another problem is the establishment of similar data about drugs already in common use and not those coming into use for the first time.

The potential already exists for selected testing of a suspected association of morbidity with a specific drug in the disease indexes,¹ maintained by over 100 general practitioners. These disease indexes now contain approximately two million patient years of records of total morbidity—that is, some three million episodes of illness.² The indexes can be used for investigating any adverse effect which is included consistently in the disease classification used.³ This approach was used in a study to establish whether women recorded as suffering from thromboembolic phenomena were more likely than other women to be using oral contraceptives.^{4 5} In that study a relationship between the use of oral contraceptives and venous thromboembolism was established at a statistically significant level. Other surveys carried out retrospectively from disease indexes have included linking the use of isoprenaline inhalers with a raised mortality rate in asthma. A major advantage of hypothesis testing in this way is that the studies are carried out retro-