What would be much better would be for a single reliable answerable question to be included on the death certificate, which asks the informant (who is usually a member of the family of the dead person) whether the dead person has ever, at any time over the past 10 years, been a smoker. This question could be answered "Yes," "No," or "No information" and, together with knowledge of the relationship of the informant to the dead person, would be sufficient for epidemiologists to derive useful estimates of national mortality attributable to tobacco.

Irrespective of whether those completing death certificates are to be allowed to attribute particular deaths to tobacco, consideration should be given to the inclusion on death certificates of at least one simple question about tobacco use.

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1 Beecham L. Smoking accepted on death certificates. *BMJ* 1992;305:543. (5 September.)

EDITOR,—Doctors are to be allowed to mention "smoking" as a contributing factor on death certificates but what does "smoking" mean? Does it mean only active smoking of cigarettes, pipes, and cigars? Or are doctors also going to be allowed to indicate the other major type of tobacco smoking—passive smoking—on death certificates?

Coincidentally, this decision about death certificates was announced in the same month as the publication of the report on passive smoking and heart disease by the American Heart Association's panel of expert doctors and scientists. This "AHA medical/scientific statement" concluded that passive smoking is "a major preventable cause of cardiovascular disease and death."² Heart disease is the largest killer in Britain.³ Would it not, therefore, be correct now for doctors to take the connection of passive smoking with heart disease into account when writing out the newly permitted "smoking" death certificates? And what about lung cancer and the many other diseases which have now been linked to passive smoking? Will fatalities from these diseases in non-smokers sometimes be recorded as smoking deaths by doctors? The figures resulting from smoking death certificates will be noticeably askew if these passive smoking fatalities are omitted from the reckoning, since passive smoking has been proposed to be the third largest contributing factor in preventable early death in Western society, following on immediately after active smoking and alcohol abuse.⁴ We can only hope that doctors will not filter out this state of affairs by failing to use the new ruling on smoking to mention exposure to tobacco smoke in appropriate cases where the death being recorded is from a disease now linked with passive smoking.

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4 Glantz SA, Parmley WW. Passive smoking and heart disease: epidemiology, physiology and biochemistry. *Circulation* 1991; 83:1-12.

EDITOR,—The reasoning behind the decision that doctors can now put smoking as a cause of death on death certificates without the death having to be reported to a coroner is obscure.¹ Without clarification it might be considered a self fulfilling medico-political device to support the campaign against smoking with figures of dubious accuracy.

That smoking is associated with several potentially fatal diseases is not in dispute; the difficulty lies in applying an epidemiological and statistical association to individual cases. How is a doctor to determine that smoking is of sufficient causal importance to be separated out from other possible risk factors in those diseases—particularly coronary artery atherosclerosis—regarded as multifactorial in aetiology? Even those diseases with the highest smoking related mortality ratios—lung cancer and chronic obstructive pulmonary disease—can occur in non-smokers.

The claims that the change "will show unequivocally the link between smoking and death" and "will improve the quality and accuracy of statistics on tobacco related deaths" seem unlikely for several reasons.

Firstly, the accuracy of clinical death certification without validation by necropsy is known to be suspect.²

Secondly, doctors seem to find accurate formulation of causes of death difficult.³

Thirdly, it seems inevitable that doctors will differ over the precise role that smoking may have had in individual cases. Faced with the spectre of possible future litigation, will doctors feel sufficiently confident in their knowledge of the complexities of epidemiological association to specify smoking as the underlying cause of death? The World Health Organisation's format requires smoking to be specified below the smoking related disease in part 1 of the death certificate; smoking cannot be placed in part 2 without the implication that it is unrelated to the condition specified in part 1.

At first sight it seems paradoxical that deaths resulting from smoking, which is not a natural human activity and is regarded increasingly as socially unacceptable, should be removed from coroners' jurisdiction. This is in line, however, with previous guidance from the Office of Population Censuses and Surveys to registrars of births and deaths that deaths attributed to chronic alcohol misuse need not be reported to a coroner. Such guidance only weakens an already anomalous system, which, if it is to help in the production of accurate mortality statistics, should be subject to radical and rational revision rather than ad hoc, ambiguous amendment.

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Long term problems after obstetric epidural anaesthesia

EDITOR,—D B Scott and J D O Loudon have written about our reports of associations between epidural anaesthesia and a range of long term symptoms starting after childbirth.¹ We have considered many of their points in previous publications,²⁻⁶ but some of them need clarification.

Firstly, their statement that the study was retrospective is incorrect; it had a prospective design. These terms are often misused and misunderstood. We agree that the study was based on recall of symptoms, but this does not make the design retrospective. We carefully examined the effects of long term recall and showed that women who had delivered several years before questioning did indeed report fewer symptoms than those delivered more recently; but this did not account,

even partially, for the associations of symptoms with epidural anaesthesia.

A second point, that most of the symptoms are experienced by almost everyone at some time, ignores our strict inclusion criteria. We included in our main analyses only new symptoms, accurately dated, starting within three months after the birth and lasting over six weeks. On these criteria very many symptoms were excluded.

A long second stage labour was not, as Scott and Loudon say, a prime factor determining symptoms. It was an independent predictor of backache, but only a minor one. No other factor matched the predictive power of epidural anaesthesia, and none of the potential confounders explained more than a small fraction of that power.

Finally, we agree that we need to know more about the severity of symptoms. We are engaged in another study to examine this issue. Much more work is necessary before definitive recommendations for clinical practice can be made, and we hope that others, as well as ourselves, will undertake this work.

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Predicting preterm delivery

EDITOR,—The recent introduction of a commercial test to predict premature labour by measuring fetal fibronectin in vaginal secretions has received wide publicity and patients are already asking about it. The publicity material circulated to obstetricians suggested that the test should be performed every two weeks from 24 weeks, but no guidance was given on who should be screened. Furthermore, it was claimed that among high risk but asymptomatic women a positive result indicates a 94% chance of preterm delivery. We are concerned that patients and doctors might be misled by these claims.

Firstly, if the risk of 94% is derived from the paper by Lockwood *et al* in the *New England Journal of Medicine* it is simply wrong; the quoted 94% is the rate of test positivity among women with preterm rupture of the membranes.¹ Among the very high risk group studied by Lockwood *et al* the risk of preterm delivery (the positive predictive value) was 83.1%. The difference is not great, but either risk might mislead clinicians who fail to consider patients' prior risks of preterm labour. Lockwood *et al*'s subjects had an overall 50% chance of preterm delivery considerably inflating the positive predictive value.

In the presence of intact membranes, the test characteristics for prediction of preterm labour (delivery <37 weeks) are sensitivity 81.7% and specificity 82.5% (likelihood ratio of a positive result 4.28, 95% confidence interval 2.29 to 8.24). If these results are confirmed by others this is good test performance, but even so, prediction of preterm labour in low risk women is likely to be poor. Consider such a woman tested at 24 weeks whose prior odds of preterm labour are 5:100. In round numbers, a positive test quadruples the odds to 20:100 (say 20%). If we assume the same

test characteristics for delivery before 34 weeks (they are unlikely to be better) the prior odds would now be only 1:100 and the posterior odds approximately 4:100 (say 4%). The identification of women with a 20% risk of delivering before 37 weeks and 4% risk of delivering before 34 weeks is less impressive than a 94% chance of preterm delivery. Since 17.5% of women not delivering prematurely will have had a positive test much anxiety will be caused if such testing becomes routine.

We would not wish to suggest that this test is not an exciting development. It seems to perform better than cervical assessment, which until now has been the best single predictor of preterm labour. However, any test is only likely to be useful if there is an effective intervention that can follow a positive result. No such intervention has been shown to reduce perinatal mortality in properly conducted randomised trials, although tocolysis does prolong pregnancy and reduce the respiratory distress syndrome in women who are already having contractions. In such high risk groups fibronectin measurement may direct treatment to women actually likely to deliver prematurely.

Many people will argue that there should be a randomised trial of the fibronectin test. We disagree. Tests for preterm labour cannot in themselves improve outcome. Only the interventions which follow the test result can do this. These interventions might follow any risk factor such as an early pregnancy risk score, uterine activity monitoring, cervical microbiological culture, or assessment of cervical dilatation. Women identified to be at high risk of premature labour by one or more of these tests should logically be entered in trials of the relevant intervention, not trials of the test. The first step is proper evaluation of test sensitivity and specificity in a larger series of patients. Results must be concealed from clinicians to avoid the treatment paradox that true positive results which are followed by an effective intervention will seem to be false positives. If the fibronectin test continued to perform well in such studies it could be used to identify a high risk group for trials of possible treatments.

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1 Lockwood CJ, Senyei AE, Dische R, Casal D, Shah KD, Thung SN, *et al.* Fetal fibronectin in cervical and vaginal secretions as a predictor of preterm delivery. *N Engl J Med* 1991;325: 669-74.

Bovine insulin

EDITOR, — None of the contributors to the "human insulin and hypoglycaemia debate" mentioned bovine insulin.¹ In our fascination with the scientific minutiae of hypoglycaemic unawareness we seem to have forgotten that some of the worst problems occurred when middle aged or elderly people were unnecessarily switched, on a unit for unit basis, to human insulin from the once daily, long acting bovine insulin that had suited them for years.²

In a 1988 paper 22 diabetic patients with true sulphonylurea failure (mean age 64.5 (range 50-88) years) were randomly allocated to once daily long acting bovine lente insulin (Neulente, Nordisk/Wellcome) or once daily human insulin zinc suspension (Humulin Zn, Eli Lilly).³ The only striking difference between the two groups was that after six months' treatment the patients receiving Neulente had had only four episodes of hypoglycaemia, whereas those receiving Humulin Zn had had 46, 36 of which occurred between 3 am and 6 am. Humulin Zn is now described as an intermediate acting insulin, although studies in

normal volunteers had shown that its action was equivalent to a lente insulin, with the nadir of blood glucose concentration occurring 20 hours after injection.⁴

When acting as a referee for the 1988 study¹ I commented how strange it was that by the time the paper was published the insulin which "worked" would have been withdrawn. The fact is that there is no satisfactory long acting human insulin that can be used either for once daily treatment of older patients or as the basis of a basal bolus regimen for younger patients. The problem is compounded by the fact that human Ultratard is described as a long acting insulin when in fact its 50% disappearance time is 12-18 hours versus 35-53 hours for the now withdrawn bovine equivalent.⁵ One clinical study concluded that the onset of action of human Ultralente is two to four hours, with a broad and variable peak between six and 12 hours after injection,⁶ so it does not produce the constant basal insulin concentrations that the architects of the basal bolus concept originally envisaged.⁷

My experience is that attempts to treat older patients with a once daily injection of any human insulin formulation are often frustrated by nocturnal hypoglycaemia and morning hyperglycaemia, leading to one version of brittle diabetes in the elderly.⁸ Once daily bovine lente (Hypurin Lente) or protamine zinc insulin (Hypurin PZI) works well in these patients and is also sometimes a good choice as a basal insulin for younger people who are plagued by nocturnal hypoglycaemia on a four times daily regimen in which the human intermediate acting insulin last thing at night does not last long enough.

Why has bovine insulin apparently been consigned to the scrap heap?

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Practising intubation on cadavers

EDITOR, — Alison Tonks gives a misleading interpretation of the BMA's policy on training in intubation techniques and how that policy stance was reached.

The issue was initially raised at the September 1991 meeting of the BMA Ethics Committee, and after a long debate covering the philosophical, spiritual, legal, and ethical considerations it was agreed that the practice was ethical but that protocols should be established with regard to who should be taught and how training should be performed to maximise its usefulness while ensuring that the body was treated with due respect.

The establishment of a protocol was delegated to a joint working party of the BMA and the Royal College of Nursing which had already been convened to discuss resuscitation policy. At the

first meeting of this working party it became clear that many nurses were uncertain of the ethical basis of this training and that the medical profession was itself divided over whether this type of training was useful. The working party therefore decided to send a questionnaire to all interested parties to establish how widespread the practice was and whether it was thought to be useful, and the results of this survey confirmed a sharp divide within the medical profession—namely, that accident and emergency staff thought it extremely valuable while anaesthetists considered it unnecessary. At this stage there was a meeting of the British Association of Accident and Emergency Trainees in Liverpool, where it was unanimously agreed that this type of training was an extremely useful way of training junior staff in essential intubation techniques.

The survey results were discussed at a further meeting of the joint working party and it was decided to outline the principles that should be included in establishing protocols but to advise the British Association of Accident and Emergency Medicine and the Association of Anaesthetists to try and establish joint guidance. This stance was subsequently supported by the BMA Ethics Committee and by BMA Council.

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1 Tonks A. Intubation practice on cadavers should stop. *BMJ* 1992;305:332. (8 August.)

EDITOR, — The BMA and Royal College of Nursing have issued a statement about tracheal intubation of patients who have just died, which is often done for teaching purposes.¹ The statement suggests that this should be done only in accident departments on patients who have died after sustaining major head and neck trauma as these patients may be difficult to intubate and the technique cannot be learnt elsewhere. The statement concludes that most doctors and nurses who need to be proficient in intubation techniques can obtain sufficient experience through practising on manikins and in the anaesthetic room.

No mention is made of the problems associated with learning to intubate newborn babies, the largest group of patients who may need emergency resuscitation. It does not seem from the statement that any paediatricians were consulted. In large units experienced staff accompany new trainees to deliveries, but this may not always be feasible. Manikins of newborn babies, although a useful training aid, do not show anatomy clearly and are not realistic. For this reason we believe that supplementing the teaching of trainees by intubation of stillborn infants is appropriate. This can be done sensitively and privately but is not easily discussed with the parents at such an emotional time. In our unit it is always supervised.

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Drug resistant tuberculosis

EDITOR, — The report of the discovery that the deletion of a single gene is the cause of isoniazid resistance in *Mycobacterium tuberculosis* contains some misleading comments.

New procedures (such as the Bactec radiometric