levels would be considered arrhythmogenic even in structurally and electrically normal hearts and if found in patients on a medical ward would require prompt emergency treatment. It is possible, therefore, that the physiological rise in arterial plasma potassium might explain sudden death on exercise in patients with normal cardiac and coronary anatomy and no known electrophysiological abnormalities.

Since sudden death on exercise is relatively uncommon in healthy people, it is pertinent to consider how the heart is protected from hyperkalaemia induced by exercise. Two mechanisms have been suggested.

Firstly, in animal studies hyperkalaemia has been shown to reduce the amplitude of single ventricular myocyte action potentials.⁵ Giving β agonists (adrenaline, isoprenaline, or terbutaline) restores the action potentials to normal.⁵ It is therefore possible that the raised catecholamine levels associated with heavy exercise⁶ protect the heart from exercise induced hyperkalaemia.

Secondly, levels of extracellular potassium of 7-8 mmol/l may (as calculated by using the Nernst equation) reduce the resting potential of all accessible excitable cell membranes by about 18 mV. This change would be sufficient to decrease skeletal muscle contractility and so lead to fatigue.⁷ Pain afferents (C fibres) would also be depolarised, leading to muscular pain. Fatigue and muscle pain induced by hyperkalaemia may therefore be limiting factors during heavy exercise and prevent arterial plasma potassium rising further to increasingly dangerous levels. Some support for this suggestion comes from the observation that patients with myophosphorylase deficiency (McArdle's syndrome) become fatigued at very low work loads (60-80 W), when the plasma arterial potassium has risen to about 6-7 mmol/l.⁸ At this level of work normal subjects are not fatigued, and their potassium rises by only 0.5 mmol/l.⁸

It is possible that sudden death on exercise in fit people with normal hearts may be related to a failure of one or both of these possible protective mechanisms.

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Emergency feeding programmes

EDITOR,—Jeremy Shoham is correct in saying that giving a child a small supplementary food ration is pointless if the child has no access to food at home.¹ In using for illustration the current crisis in Somalia, in which the Save the Children Fund is doing much to feed children, he has made sweeping charges which he does not sustain.

Supplementary rations are intended to supplement a basic family ration that is marginally inadequate in terms of quality or quantity or to

ensure more rapid recovery of malnourished children, or both. Such programmes are often run by giving the supplement in a dry form and are generally phased out as nutritional status improves. But, particularly in Africa, basic family rations are now often grossly inadequate in quantity and quality. Supplementary cooked food is then distributed for broadly three reasons.

Firstly, such food is used where the general ration or other food supply is grossly inadequate or wholly lacking. Here the supplement tends to be a full ration. Save the Children Fund has selected children in terms of weight for height, attempting to ensure the survival of the greatest number for the longest period in the anticipation of improved supplies.² Such feeding programmes are not in any sense supplementary—they seek to direct limited supplies to a defined population, using malnutrition as the basis for definition. This requires administration of cooked food. We have suggested that the term supplementary should be dropped.³

Secondly, this food is used where domestic fuel supply is limited—for example, in highland Ethiopia, in Iranian Kurdistan, and where providing cooked food can achieve large fuel savings.

Lastly, it is used where it is important to have access to the children concerned. Supplementary centres allow children to be monitored for growth; sick children can be identified and treated quickly and economically. A large part of many feeding programmes is primarily concerned with the medical care of severely malnourished children.

The effectiveness of feeding programmes may be limited by low attendance, but generalisation is impossible. For example, the attendance of Rohingya refugees currently in Bangladesh relative to expected attendance from a population nutrition survey is over 90%, and drop out rates are low. Wet feeding of children can be very effective, and the routine collection of information in many feeding programmes shows high recovery rates.

Cooked feeding programmes are often socially undesirable and administratively complex, and as an agency we would prefer not to use them. That we continue to do so in some cases reflects the abysmal supply and quality of food. As a matter of written policy we do not arrange emergency programmes for fundraising purposes, and we refute the charge that we would misuse children to this end.

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EDITOR,—In his editorial on emergency feeding programmes Jeremy Shoham raises several issues but offers no solutions for field workers faced with famine or refugee crises.¹ He criticises agencies' "lack of institutional memory and guidelines," yet every practical point and dilemma he raises is covered in current agency guidelines.^{2,4} These guidelines are a great help to relief workers in emergencies and are surely an indicator that agencies' institutional memories are functioning.

For instance, an agency's decision to implement a supplementary feeding programme is usually based on the nutritional status of children under 5 determined by a sample survey and on the availability of staff and food. The guidelines produced by agencies for this are explicit and clearly state that such supplementary feeding programmes are justified only if there is an adequate basic ration for the whole population. If the basic ration is inadequate then, clearly, a targeted supplementary

feeding programme is of little value and diverts attention away from the main issue.

Likewise, in deciding whether to implement a dry or wet feeding programme field workers have to assess the capability of the feeding agency to organise wet programmes in feeding centres and the availability of fuel for families to prepare dry rations. Agency guidelines cover this issue. The problem of micronutrient deficiency is also addressed, with suggestions for types and quantities of supplements to the basic general ration. The difficulty lies with the field staff, who must find adequate supplies and transport to distribute the foods that are not packaged and are difficult to store.

The guidelines for emergency feeding programmes are clear that the whole population must have a basic ration with an agreed minimum energy content with supplementation of micronutrients. Without these basic requirements any response will be inadequate. Depending on the emergency, additional energy may be required for vulnerable groups.

The real issue is not one of guidelines but of resources and logistics. If the international community in the form of the United Nations High Commission for Refugees and the World Food Programme fulfil their mandate to provide adequate food supplies where and when they are needed and listen to the accumulated experience of implementing agencies such as Oxfam and Save the Children Fund the response to nutritional emergencies will be less likely to fail "predictably and often spectacularly."

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Smoking accepted on death certificates

EDITOR,—The recently proposed change in death certification rules, whereby deaths attributed to tobacco would no longer automatically be referred to a coroner,¹ will lead to a number of death certificates attributing death to tobacco. There is, however, little reason to expect that it will be the correct number, for there are substantial scientific difficulties in assessing the external causes of any one particular death from the main chronic diseases to which the use of tobacco can sometimes lead. These difficulties can, of course, be circumvented by using appropriate epidemiological methods when assessing the proportion of deaths that is attributable to tobacco in a population. But for most deaths that were in fact due to tobacco there is no reliable way to know that those particular deaths were due to tobacco. Consider, for example, 1000 habitual smokers who die of myocardial infarction at about 65 years of age. Even though the epidemiologist may be able to say with confidence that about half of these 1000 deaths were due to tobacco, there would be no way for the medical practitioners certifying those deaths to know which ones to attribute to the habit. Should all be attributed, should none be attributed, or should about half be attributed—and, if half, which ones?

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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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