## Classifying suicide

EDITOR,—A report by the Samaritans, *Reach Out. . . . We'll Be There*, was accompanied by the claim that published suicide figures are the "tip of the iceberg."[1,2] It is suspected that the increasing suicide rate among young men has been underestimated because cases are effectively concealed by the coroners' courts to spare families further grief.[3] It has long been acknowledged that suicides are underestimated because some are categorised as "undetermined."[4] Recent figures, however, suggest a deterioration in reliability, with increasing numbers miscategorised as undetermined.

From 1968 an ICD code has been used for "injury undetermined whether accidentally or purposely inflicted" (E980-989) as well as a code for suicide (E950-959). For the first five years (1968-72) undetermined deaths as a proportion of all deaths classified as suicide or undetermined averaged 22·6%. In England and Wales, allowing for fluctuations from year to year, there was a steadily upward trend, and for 1986-90 the average was 33·2%. Scottish figures show wider fluctuations, but the overall trend was opposite: for 1968-72 the average was 37·5% but for 1986-90 this fell to 26%. These opposite trends probably account for much of the change in the relative suicide rates between Scotland and Britain.[5]

Figures for death by hanging highlight the problem. Sibbald pointed out that "a death by hanging can seldom be concealed and when known, it can seldom be regarded as otherwise than suicidal."[6] ICD code E953 combines suicides by hanging, strangulation, and suffocation, although the overwhelming majority of cases are hangings. There is a corresponding undetermined category, E983. In England and Wales the average percentage of these deaths categorised as undetermined was 6·0% over 1968-87 (range 4·4-9·0%; the 9·0% occurred in 1969). Recent years show a considerable increase: 1988, 11·4%; 1989, 13·4%; 1990, 15·2%. Such figures lack credibility. By contrast, the average percentage of such undetermined deaths in Scotland for 1968-90 was 2·7% (range 0-8·5%; the 8·5% occurred in 1968). For 1988, 1989, and 1990 the percentage undetermined was 2·1%, 0·8%, and 1·5%, figures that are realistically low.

Since 1979 British statistics on hanging have been more precise, separating true hangings (suicide E953.0 and undetermined E983.0) from other forms of asphyxia. The table gives the absolute numbers and the percentage undetermined.

The statistical impact of misclassification is well shown in the group of greatest concern, young men aged 15-24.[3] During 1979-90, 1195 suicidal hangings (E953.0) and 231 undetermined hangings (E983.0) occurred in this group. Reclassification of these undetermined cases as suicides would increase the number of suicidal hangings by 19·3% and of suicide by 5·8%.

The essential claim of the Samaritans that the coroners' system conceals suicides seems justified.[2]

*Numbers of suicides by hanging (ICD code E953.0) and of deaths classified in corresponding undetermined category (code E983.0), and these undetermined deaths as percentage of total*

| | 1979-84 | 1985 | 1986 | 1987 | 1988 | 1989 | 1990 |
|---|---|---|---|---|---|---|---|
| Suicide by hanging | 794-937 | 1004 | 1003 | 1024 | 978 | 940 | 982 |
| Corresponding undetermined category | 37-47 | 72 | 87 | 113 | 132 | 142 | 180 |
| Undetermined deaths as % of total | 4·2-5·6 | 6·7 | 8·0 | 9·9 | 11·9 | 13·1 | 15·5 |

### Advice to authors

Priority will be given to letters that are less than 400 words long and are typed with double spacing. All authors should sign the letter. Please enclose a stamped addressed envelope for acknowledgment.

The situation might be improved by changing the burden of proof for a determination of suicide from clear evidence of intent to a balance of probabilities and by ending the mandatory requirement for a public inquest into unnatural deaths. Under the Scottish procurator fiscal system such cases are investigated privately rather than publicly.

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## Lipoprotein(a) and coronary heart disease

EDITOR,—Melanie Davies and colleagues observe that plasma lipoprotein(a) (Lp(a)) concentrations are largely genetically determined, the extent being 40-70%.[1] At the gene level most subjects with a short apoprotein(a) gene have a small apoprotein(a) molecule that is associated with high circulating concentrations of Lp(a), while subjects with a large apoprotein(a) molecule have low plasma Lp(a) concentrations; people with a large apolipoprotein(a) molecule make up the bulk of Western white populations. Given the strong inverse relation between the size of the apoprotein(a) molecule and plasma Lp(a) concentration, simple differences in Lp(a) concentration cannot be compared between small groups with any confidence that they arise from the pathological condition studied since they may simply be due to dissimilarity in the size of the apoprotein(a) molecule. This influence can be determined either at the genetic level by pulsed field gel electrophoresis to examine gene size or by measurement of the size of the apoprotein(a) isoform directly by SDS gel electrophoresis with immunostaining; the second method is less arduous.

In Davies and colleagues' study the group with impaired glucose tolerance may have contained more subjects with a smaller apoprotein(a) molecule and, therefore, higher Lp(a) concentrations than the control group. The same problem will occur in any small cross sectional study designed to investigate an effect of a pathological condition on Lp(a) concentration, such as the study by Kapelrud *et al*, which examined the effect of microalbuminuria on Lp(a) concentration.[2] The importance of this is shown by a study of Lp(a) concentration and its association with coronary heart disease in patients with familial hypercholesterolaemia, which showed that the higher Lp(a) concentrations in patients with the disease were reflected in a different distribution of apoprotein(a) sizes. The group with coronary heart disease contained a higher proportion of subjects with smaller apoprotein(a) isoforms and, hence, higher Lp(a) concentrations than did the group without the disease.[3]

This problem in study design and interpretation of results can be overcome by using large enough groups to avoid a possible bias from the effect of the isoform inherited. Alternatively, subjects can be studied longitudinally as the genetic influence will thereby remain comparable.

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AUTHORS' REPLY,—As Mary Seed and Joseph Loscalzo emphasise, the plasma concentration of lipoprotein(a) (Lp(a)) is largely genetically determined, although metabolic effects such as hyperglycaemia may have an influence.[1] Seed and Loscalzo point out that the size of the apoprotein(a) isoform is strongly adversely related to the plasma Lp(a) concentration, and, certainly, differences in the size of the apoprotein(a) molecule in the group with impaired glucose tolerance may have accounted for the differences in Lp(a) concentration that we observed. This in itself may be an important finding as the size of the apoprotein(a) molecule is known to influence its pathogenicity, and we endorse the view that apoprotein(a) size should be measured by the techniques described in subjects with impaired glucose tolerance.

Many of the studies that have looked at the role of Lp(a) in the development of coronary artery disease have examined subjects in whom the disease is already well established.[2] Seed and Loscalzo refer to the problems of studying Lp(a) concentrations in small groups of subjects as differences arising from the pathological condition may be due to differences to the apoprotein(a) size. This reference to impaired glucose tolerance as a pathological process is interesting. Impaired glucose tolerance may be present in 10-20% of the adult population, and certainly some subjects with it do have an increased risk of subsequently developing both non-insulin dependent diabetes and coronary artery disease.[3,4] The nature of the

pathology present in impaired glucose tolerance is complex. We approached the problem of determining whether Lp(a) is a possible factor in why such subjects develop coronary artery disease by examining them before clinical coronary artery disease was established.

Rather than suggesting that the pathological process—for example, impaired glucose tolerance—accounts for differences in Lp(a) concentration, we suggest that differences in either Lp(a) concentration or the size of the apoprotein(a) isoform in such subjects could account for their subsequent development of coronary artery disease. To this end a larger long term prospective study is required, not so much to avoid the bias from the effect of inheritance of the apoprotein(a) isoform, but to examine the effect of the potential differences in this on the development of coronary artery disease in what is a common condition.

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## Diets that protect against coronary heart disease

EDITOR,—As we share the same views we enjoyed John Yudkin's letter[1] about our article.[2] We agree that an association between raised blood cholesterol concentration and coronary disease does not denote that cholesterol in itself causes the disease and that changes in diet and lifestyle will reduce the incidence of and mortality from chronic diseases. There is a need to define the extent of lifestyle changes required to modify risk factors in coronary disease. In the United Kingdom a modest increase in certain risk factors among South Asians is enough to cause more coronary deaths than in British people with higher overall levels of risk.[3] In India chronic diseases including coronary disease are emerging rapidly,[4] although dietary saturated fat and cholesterol and energy intakes are as good[2] as those in the American Heart Association's step I diet. These paradoxical observations may be related to the duration of lifestyle changes; such changes are relatively recent among Indians compared with the British.

The development of a risk factor seems to be a protective response of the body in an attempt to fight against changes in lifestyle. Most investigators believe that obesity is the best example of maladaptation of our bodies in response to increased intake of energy and reduced physical activity. Hypertension develops when the body is unable to adapt to increased consumption of salt or alcohol. Hypercholesterolaemia may be the result of maladaptation to increased intakes of saturated fat and cholesterol. Reducing any of these risk factors without altering the lifestyle factor concerned would mean preventing our bodies from fighting the effects of the lifestyle factor. A gradual increase in a risk factor, as has happened in British people, may allow the body to fight the lifestyle factor more efficiently and provide better adaptation, as compared with an abrupt increase in risk factors, as has been observed among South Asian immigrants, in whom lifestyle changes are of more recent origin.[3]

In the Indian study of survival after infarction our aim was to achieve maximal change in nutritional lifestyle.[2] Our success in this may explain the improved survival compared with survival in other cholesterol lowering studies. Many *BMJ* readers have written to us asking how we achieved such good adherence to dietary advice. The answer is that we emphasised foods to eat rather than those to restrict. Patients were advised to eat tasty fruits such as guava, papaya, banana, and musk melon before meals to displace foods rich in saturated fat and cholesterol.

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## Value of Dundee coronary risk-disk

EDITOR,—Soaked in Hugh Tunstall-Pedoe's bath water and left at the bus stop,[1] the rejected baby[2] thinks it appropriate to comment. We set out to enhance the usefulness of the Coronary Prevention Group and British Heart Foundation's guidelines by showing what happens if you put them into practice.[2] We were not trying to assess the Dundee risk-disk. We discovered what we already knew—that the guidelines are complicated—but we also made some unexpected discoveries. One of the most important was the high proportion of patients at high unifactorial risk who are likely to be excluded from special care if the guidelines are implemented without modification. This does not mean that we are Luddites who reject the multifactorial approach—indeed, we have often written in its support—but we realise that we are not just dealing with cardiovascular morbidity in our clinical practice. We do not advise patients who smoke 20 cigarettes a day to stop smoking just to reduce their risk of a heart attack, but we do regard smokers as needing special care.

As working general practitioners, we found that the guidelines left some unanswered questions. How many patients would be in the general risk group? How many of the patients allocated to special care would already be under regular review? We think that the tables and figures 3 and 4 in our paper are an important service to users of the guidelines. We did not intend to "cause unnecessary difficulties for readers." In contrast, figure 5, which indicates the percentage of those at risk who might slip through the guidelines, is perhaps a difficulty for the guidelines' originators.

Our main comment on the Dundee disk was that the Dundee score is of more use than the rank. Our reasons are clearly stated. Firstly, the distribution of risk is highly skewed, so the actual difference in risk between those in the back two thirds of the metaphorical bus queue is relatively small. To make the queue analogy work you have to arrange the queue at different distances from the stop so that those at the back are bunched together and have a long way to run to catch the bus.

Secondly, you cannot use the rank directly to predict workload, as had been suggested. The difference between the OXCHECK and Dundee figures does not imply that the Dundee figures are wrong but simply that populations vary and that the Dundee rank can therefore be misleading as a measure of workload. So, although we see the attractions of the concept of the bus queue, particularly for ease of explanation to patients, we still prefer the score as a practical measure of risk. We also challenge the statement that our preference is "at odds with most users." This statement is based on responses to a questionnaire sent to about 300 purchasers, of whom just over half had used the disk: 37% consistently preferred the rank, 20% preferred the score, 26% used both, and 17% had no preference.

For the record, we think that some of the "unexplained" difference between the OXCHECK and Dundee population risks can be explained by the fact that the Scottish data were accumulated during 1984-6. For example, smoking rates among 50-59 year old men and women in England and Wales fell by more than a quarter between 1984 and 1990.[3] We would also draw attention to the perception in Dundee that Luton is in "one corner of southern England." It is in fact as near to Birmingham as to the south coast and is nearer to Scotland than to Penzance.

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EDITOR,—It is difficult for a non-cardiologist to understand the controversy about the extent to which the Dundee coronary risk-disk has value when the risk calculations on which it is based seem to be flawed.[1,2]

The disk calculations do not take into account women's hormonal status: whether they are premenopausal or postmenopausal or are taking hormone replacement therapy, as increasing numbers are. Yet there is accumulating evidence that hormone replacement therapy has a major effect in reducing fatal coronary heart events, especially in women with pre-existing coronary atheroma.[3,4] Over the years the topic has been reviewed by Gambrell, Beaglehole, and, more recently, Bush.[5-7] Bush used epidemiological criteria for causation in assessing the strength of the association between hormone replacement therapy and reduction in risk of coronary heart disease. These criteria included consistency of the association, a proper time sequence, a strong association between the two variables, a change in risk with change in exposure, and biological plausibility.

Tunstall-Pedoe is reported as dismissing the evidence as a myth,[8] relying instead on a logarithmic plot of coronary mortality in British women which showed no change in gradient around age 50 but failed to take account of time since the menopause.[9]

Women receiving hormone replacement therapy seem to face not only a better quality of life but also a longer life, extended by a decrease in mortality of up to 40% (depending on duration of use of oestrogen) and largely attributable to a reduction in coronary heart disease.[10]

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