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 0.8-5.5%; the 5.5% 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 15-24.¹ 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