matters arising

The design of wildlife preserves

MAY[1] has drawn attention to the need to make nature reserves sufficiently large, arguing from both theory and observations on island biogeography. I would like to add two comments to reinforce his case. The first is that a similar species–area relationship is found in quadrats of different size and in other studies of continuous ecosystems, as well as across sets of islands. This means that when a boundary is drawn around a nature reserve in a larger area of the same habitat, the number of species in the reserve will be less than that in the region as a whole. The other point is that if the remainder of the habitat is then cleared away, there will be an edge effect around the new reserve. Species suited to the edge will frequently be different from those suited to the centre of the reserve. If the reserve is intended to maintain the 'central' species, then its effective size is smaller than its apparent area.

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[1] May, R. M., *Nature*, **254**, 177–178 (1975).

Sudden death in infancy

I AM pleased to learn that Carpenter and Emery[1] have succeeded in identifying a limited number of high-risk cases in the field of child-death prevention. But their conclusion that "there are encouraging indications that the study may be preventing some deaths" is not supported by the data.

Carpenter and Emery[1] compared a high-risk group which was not selected for follow-up health care with a sample of a low-risk group, and found that the former had significantly higher mortality. But this result merely reflects the original group assignments. The relevant issue is rather, whether or not the high-risk group which was followed up differs from the high-risk group which was not selected.

I have reanalysed Carpenter and Emery's data (Table 1) using χ-square tests corrected for continuity. Two groups (high-risk subjects not selected, and high-risk subjects either not selected or not participating) are compared separately with the high-risk group receiving aftercare. The null hypothesis for each test is that mortality will be at least as great in the follow-up group as in the non-treatment group. As statistical convention requires significance at the 0.05 level, the null hypothesis cannot be rejected in either case.

Perhaps a larger sample will yield different results in the future.

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[1] Carpenter, R. G., and Emery, J. L., *Nature*, **250**, 729 (1974)

CARPENTER AND EMERY REPLY—

Magura[1] is mistaken when he says that the significant difference we reported[2] "merely reflects the original group assignments". The group assignments were based on a discriminant function that had been constructed before the prospective study began. The significant difference referred to is fundamental to our main point, which is that it is possible to identify babies who will be at risk of sudden death in infancy within 24 hours of their birth. We also showed that the high-risk group is sufficiently sharply defined that it can be studied prospectively.

Magura shows that the observed difference in mortality rates between the high-risk group followed up and the high-risk controls is compatible with the null hypothesis, as we were well aware. But the data are equally compatible with the alternative hypothesis that the follow-up study is preventing deaths, and on a likelihood-ratio criterion the latter hypothesis is more strongly supported than the former. This common sense view is also reinforced by case reports[3]. We think that our conclusion that "there are encouraging indications that the study may be preventing some deaths" summarised this situation accurately.

We are confronted with a difficult ethical problem. To know whether or not the discriminant function and the follow-up study are effective a high-risk control group is essential. But in the light of results such as those summarised by Magura, for how long does it remain ethical to exclude high-risk babies from the follow-up study? The classical significance test approach followed by Magura gives one answer to the problem. It would be interesting to know if this view is generally shared by your readers.

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[1] Magura, S., *Nature*, **256**, 519 (1975).
[2] Carpenter, R. G., and Emery, J. L., *Nature*, **250**, 729 (1974).
[3] Emery, J. L., and Carpenter, R. G., *SIDS* 1974 (edit. by Robinson, R. R.,) 97-106 (Canadian Foundation for the Study of Infant Deaths, Toronto and London, 1974).

Table 1 Number of infants and number of sudden deaths among high-risk groups

	No. in group	No. of deaths	Significance (one-tailed test)
Followed up	354	0	
Not selected	477	4	$0.15 > P > 0.10$
Not selected or not participating	557	5	$0.10 > P > 0.05$

Proterozoic supercontinent: time duration and the Grenville problem

THE reconstruction of the Proterozoic supercontinent has been devised[1] by superimposing 2,000–1,100 Myr palaeomagnetic poles from two regions and was derived by rotating North America anticlockwise 146° about a Euler pole at 138°E, 73°N. With this operation most dated, Precambrian, palaeomagnetic poles from Africa correlate with comparable poles from North America with fields of agreement illustrated in a qualitative way in Fig. 1. The new data of McGlynn *et al.*[2] enable further comparison and the 2,090 Myr Indin dykes pole agrees closely with several poles 2,070–2,090 Myr in age from Africa[3]. Also, 2,150 Myr poles from western Africa correlate with some poles from the Nispissing diabase and Abitibi dykes (~2,150 Myr). It is not yet possible to match 2,200–2,300 Myr poles from Africa with North American data, and there are clearly two possibilities: either the reconstruction is not valid earlier than 2,150 Myr or apparent polar movements are more complicated than recognised.

The latter explanation is probably