a drug that may improve performance on a mountaineering trip is an ethical question that should be left for individual mountaineers to consider.

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Asthma deaths in New Zealand

EDITOR,—In a recent letter members of the Wellington research group¹ repeat their previous suggestions that changes in mortality from asthma in New Zealand can be explained by shifts in use of fenoterol.² Their accompanying figure is misleading as it leaves out critical information on trends in asthma treatment, and the hypothesised association cannot be reproduced if drug consumption is used instead of market share. Furthermore, their figure starts with a non-zero y axis and includes the preliminary half year mortality estimate for 1990, which is regularly lower than the final rate.

In fact, whereas mortality from asthma rose from 1974 to 1979 and fell steeply thereafter, consumption of β agonists, including fenoterol after its introduction in 1976, increased steadily (fig 1). Concomitantly, the consumption of anti-inflammatory agents, in particular inhaled steroids, also increased appreciably, while higher dose beclomethasone formulations were marketed for the first time in 1979, just before the start of the decline (fig 2). In 1988 and 1989 new high dose steroid powder formulations were introduced and rapidly assumed a major role in treatment, especially of patients with severe asthma.

Outside New Zealand increases in mortality from asthma have occurred in countries with little use of fenoterol (for example, Australia) and

countries that do not use fenoterol (the United States). Conversely, Germany and Belgium have high use of fenoterol, measured both as per capita use and as market share, but have not experienced epidemics of asthma.

As with the role of isoprenaline in epidemics of asthma experienced by some countries in the 1970s,¹ the data can be regarded as consistent with the hypotheses but only at the cost of stretching some, and ignoring other, information.

Since 1989 asthma in New Zealand has continuously been in the public eye. In accordance with the recommendations of the Australian and New Zealand Thoracic Society, further improvements in the management of asthma have occurred, as evidenced by the considerable increase in the appropriate use of inhaled anti-inflammatory drugs.

The nested case-control study within the Saskatchewan asthma epidemiology project³ differed from the New Zealand group's case-control studies in design and results. The Canadian investigators found an association between mortality and the amount of β agonist administered over 12 months. Fenoterol and the reference β agonist had similar risks when doses of equivalent effectiveness were considered. The message from this study is that long term, excessive use of β agonists is a sign for the clinician to re-evaluate the patient's management, especially the appropriate use of anti-inflammatory treatment.

To ignore the effect of major developments on the management of asthma and selectively to present and interpret data do not contribute to understanding the causes of death from asthma. Nor will this approach support the current improvements in care of patients with asthma.

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Row over Maori cot death rate

EDITOR,—E A Mitchell comments¹ on my report of the cot death rate among Maoris.² The preliminary figures released by the monitoring group for the campaign to prevent cot deaths did suggest that the Maori cot death rate for 1991 was 20-40 times higher than the non-Maori rate. The number of sudden infant deaths among Maoris was reported as 123 and the total number as 147. There were 6946 Maori live births in 1991, using the Department of Statistics definition of Maori, and 53055 non-Maori births. This gives a Maori cot death rate of 17.7 per 1000 and a non-Maori rate of 0.45 per 1000—a 39-fold difference between the rates. But the definition of Maori used by the monitoring group was wider than that used by the Department of Statistics. If the Maori denominator population is expanded by 50% to account for either parent self identifying as Maori then the difference between the Maori and non-Maori rates drops to a 24-fold difference.

In contrast, the number of postneonatal deaths among Maoris, based on the Department of Statistics classification of ethnic group, was 64, with 175 such deaths among non-Maoris. The Maori postneonatal mortality was 9.2 per 1000 and the non-Maori rate 3.2 per 1000, a difference in line with

the results of the case-control study of cot death showing a two to three times higher rate. It was this discrepancy that led epidemiologists to question the monitoring group's classification of ethnic group. Undoubtedly the monitoring group will sort this discrepancy out. The reason for bringing it to notice was concern that the public should not be misled into believing that the situation for Maoris was worse than it is.

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Vertebral fractures

EDITOR,—We agree with Alison L Armstrong and colleagues that rates of admission to hospital for vertebral fractures might be lower in the United Kingdom than in the United States.¹ Indeed, we emphasised this in our editorial² and believe that one of the challenges to those studying the epidemiology of osteoporosis lies in explaining the considerable differences in fracture rates among white populations in different countries. The paucity of information on the occurrence of vertebral fractures in the United Kingdom, however, forces initial estimates of the size of the problem to be derived from other sources of epidemiological data. We share the Nottingham group's concern over the use of Hospital Activity Analysis data for this purpose. Several prevalence surveys are, however, currently under way in the United Kingdom, and more reliable data on the subject should be available in the near future.

Armstrong and colleagues suggest that we failed to refer to two of our own previous studies, yet we referenced both of these in our editorial. The results of these studies merely add weight to our suggestion that around 35% of vertebral deformities come to clinical attention.

We agree that the clinical consequences of different vertebral deformities need further study. Fortunately, relevant data are beginning to appear. A large population based survey in the United States indicates that elderly women with vertebral deformities at least four standard deviations from the mean (our grade 2) were at 2.6-fold greater risk of disability and were 1.9 times more likely to have moderate or severe back pain.³ There was no difference in mild back pain, which was as common in those with lesser deformities or no vertebral deformity at all. The results were thus concordant with those of our own study, which found no difference in the frequency of "persistent back pain in the last year" between women with and without vertebral deformities,⁴ presumably because back pain in general is ubiquitous.

Even if the definition of vertebral fracture was restricted to the most extreme deformities, as Armstrong and colleagues suggest, the burden in the United States at least would be considerable, with about 10% of women aged 65 and over affected.³ We think, however, that this approach is unwise because it confines the condition to end stage disease and precludes the possibility of earlier diagnosis, when treatment could halt the progression of vertebral fractures to the chronically symptomatic stage. In addition, Armstrong and colleagues ignore the problem of acute pain that accompanies the onset of vertebral fractures. Thus the need to diagnose and manage considerably more men and women with recent onset of, or less severe, vertebral fractures is superimposed on the need to rehabilitate those with end stage disease. Together, these requirements are likely to impose

FIG 1—Use of β agonist inhalers measured as number of aerosol puffs and mortality from asthma per 100 000 people aged 5-34 in New Zealand, 1970-89

FIG 2—Use of steroid inhalers measured as number of aerosol puffs and mortality from asthma per 100 000 people aged 5-34 in New Zealand, 1974-89