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

I P HALL
A T COLE

Department of Therapeutics,
University Hospital,
Nottingham NG7 2UH

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Asthma deaths in New Zealand

EDITOR,—In a recent letter members of the Wellington research group[1] 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.

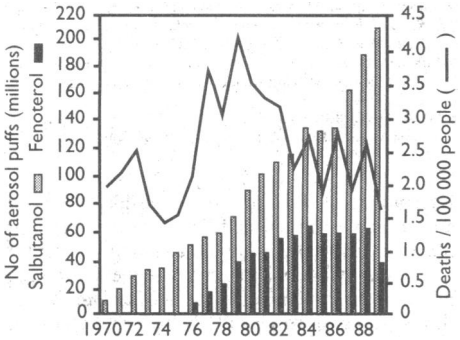

FIG 1—*Use of β agonist inhalers measured as number of aerosol puffs and mortality from asthma per 100 000 people aged 5-34 in New Zealand, 1970-89*

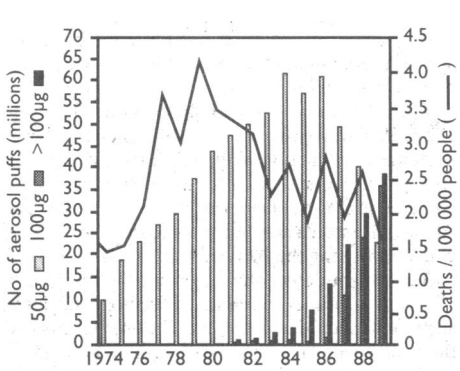

FIG 2—*Use of steroid inhalers measured as number of aerosol puffs and mortality from asthma per 100 000 people aged 5-34 in New Zealand, 1974-89*

As with the role of isoprenaline in epidemics of asthma experienced by some countries in the 1970s,[3] 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 project[4] 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 efficaciousness 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.

J F HAAS
H W STAUDINGER
C SCHUIJT

Boehringer Ingelheim,
6507 Ingelheim,
Germany

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Row over Maori cot death rate

EDITOR,—E A Mitchell comments[1] 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 53 055 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.

CHARLOTTE PAUL

Department of Preventive and Social Medicine,
Otago University Medical School,
PO Box 913,
Dunedin,
New Zealand

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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 editorial[2] 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

a considerable clinical and economic burden, as we indicated in our editorial.

CYRUS COOPER

MRC Environmental Epidemiology Unit,
Southampton General Hospital,
Southampton SO9 4XY

L JOSEPH MELTON III

Section of Clinical Epidemiology,
Mayo Clinic,
Rochester,
MN 55905,
USA

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Adrenaline in allergic emergencies

EDITOR,—We disagree strongly with Gregory Y H Lip and Malcolm J Metcalfe's view that the correct route for administering adrenaline during allergic emergencies is by an intramuscular injection.[1] The intravenous (rather than intramuscular or subcutaneous) route is best under these circumstances as it is safe and effective and a therapeutic response is both rapid and assured. During hypotension the absorption of drugs given intramuscularly and subcutaneously may be unreliable; furthermore, uptake of adrenaline given intramuscularly may be delayed. Venous access is usually not difficult to secure as vasodilatation is a major pathophysiological characteristic of anaphylaxis. Lip and Metcalfe state that absorption after intramuscular injection is "rapid and usually adequate if reasonable circulation is present." This seems highly questionable, considering that hypoperfusion is characteristic of anaphylxis.

Concerns for the potential dangers of intravenous adrenaline are largely misfounded; like any drug, adrenaline can be dangerous if given incorrectly. If given in a controlled titrated manner at a dose of 5-8 μg/kg in a dilution of 1:10 000, however, it is safely and rapidly delivered to its site of intended action. Of course, by the nature of its sympathomimetic action adrenaline may cause arrhythmias, but in anaphylaxis so might hypoxaemia, hypotension, and the effects of mast cell mediators. Anecdotal reports often cited by those who claim that intravenous adrenaline is hazardous either do not comment on the speed of injection of the dose given[2] or fail to exclude other causes of the arrhythmias witnessed.[3] We conclude that when adrenaline is required in an anaphylactic emergency it should be given in the correct dose intravenously.

G B SMITH
B L TAYLOR

Intensive Therapy Services,
Queen Alexandra Hospital,
Portsmouth PO6 3LY

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Vaccination and immunisation

EDITOR,—The Health Education Authority seems to be out of step with the World Health Organisation in wishing to discard the term vaccination.[1] The World Health Organisation uses "immunise" to refer to the process of conferring immunity, while "vaccinate" means administering a vaccine. There may be an important difference between the two. The two terms help us to remember that children who are "vaccinated" are not necessarily "immunised." Though this distinction may not be obvious to members of the public, it is valuable for health professionals and should be preserved.

TONY WATERSTON

Community Headquarters,
Newcastle General Hospital,
Newcastle upon Tyne NE4 6BE

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The GMC on performance

EDITOR,—Richard Smith mentions my bill in his editorial on the General Medical Council's proposals for performance assessment.[1] I have made it clear that whatever merits these proposals may have—in any case, as Smith points out, they would take some time to implement—I do not believe that they meet the major gap in the existing disciplinary procedure. This gap is greatly to the disadvantage of the profession itself, let alone the public.

Smith states that my bill would "produce a lesser charge than serious professional misconduct." This is not the case, although critics of my bill have on occasion implied or stated that it is so. The bill itself states that where in the course of an inquiry into a prima facie case of serious professional misconduct the professional conduct committee of the General Medical Council judges that a fully registered person has "behaved in a manner which cannot be regarded as acceptable professional conduct, the Committee may, if they think fit, direct that the registration shall be made conditional in accordance with the foregoing subsections of this section [36]."

Thus the preliminary proceedings and the prima facie charge of serious professional misconduct remain, and it would still be open for the professional conduct committee to find that any demonstrable conduct of a practitioner does not amount to serious professional misconduct. At the moment, however, if the committee finds that the misconduct is not acceptable professional conduct, as indeed it did in the case of Alfie Winn, which Smith mentions, it is debarred by law from taking any action other than the variable publicity.

Thus the professional conduct committee can find the conduct of a doctor towards patients unacceptable but then go on to accept it in practice since it has no legal remedy. But the one I suggest—that of "conditional registration"—is already provided for and used in the existing act. It is less penal than remedial since it would attempt to meet any deficiency of the practitioner in a way most suited to the practitioner's needs.

This modest change would not only be of direct benefit to the profession but add to its reputation of being capable of effective self regulation, which, alas, is not the case at present.

NIGEL SPEARING

House of Commons,
London SW1A 0AA

1 Smith R. The GMC on performance. *BMJ* 1992;**304**:1257-8. (16 May.)

EDITOR,—I read with interest your editorial on the new General Medical Council machinery for dealing with long term poor performance by doctors.[1] I share all your anxieties, but I have a further concern—namely, that this move by the GMC could blur responsibilities and hinder rather than facilitate the taking of effective action. I write as a clinician who is now involved in management of the service. It seems to me that as a profession we have evaded this issue. Many of the aspects of performance the new regulations cover are the legitimate concern of employing authorities and eventually can appropriately be dealt with only by these authorities. By involving itself in this new role the GMC may merely ensure that neither it nor the health service itself effectively deals with this situation.

MARTIN McNICOL

Central Middlesex Hospital NHS Trust,
London NW10 7NS

1 Smith R. The GMC on performance. *BMJ* 1992;**304**:1257-8. (16 May.)

EDITOR,—Before the General Medical Council starts disciplining doctors for rudeness and incompetence perhaps it might examine a few cases. I suspect that most of the incidents would feature doctors under extreme pressure—from fatigue, overwork, or unreasonable patients. It is the conditions of our work rather than personal deficiencies that produce conflict and poor performance.

As a general practitioner, I believe that our job is becoming untenable. On the one hand the government, family health services authorities, pressure groups, and members of our own profession are queueing up to complain about our standards; on the other hand we are expected to impose order on a system that allows the public free unlimited access to health care at any time of the day or night.

I do not believe that we can combine unlimited access with a consistently high quality service. If the quality of care is low it is because doctors have compromised their standards to ensure that every patient who presents is seen. To raise standards we need more time with fewer patients, which means placing some limitation on access. I can see no other way of resolving this dilemma.

CHRIS NANCOLLAS

Yorkley,
Gloucestershire GL15 4TX

Recording HIV status on police computers

EDITOR,—J K Mason's editorial on police practice suggests that information on HIV status is stored on the police national computer with the best of motives.[1] Subsequent letters from A J Lyons,[2] A Jeynes,[3] and D C Macallan[4] correctly point to errors in the accuracy of data and the deleterious impact of holding such data.

Possible uses of this information should be looked at more closely. Mason suggests that records are centrally controlled and have strictly limited objectives—namely, to protect members of the public and police by reducing the risk of infection. This belief seems naive, as shown by recent press reports.

The *Evening Standard* recently reported: "almost half of a survey sample of prostitutes working the Kings Cross red light area are drug abusers and of these 75% claimed to be HIV-positive, police said today."[5] This story was taken up by local and national papers. A second report with the headline "Met has Mugshots of Hookers with AIDS" quoted chief Inspector Derek Talbot, saying "of the 50 hard core regulars who work the streets around the station we reckon that three out of four have the virus."[6]

Subsequent discussions with the police at the relevant vice unit revealed that the reports were not based on a specific survey but on the collation of existing police information, including records held on the police computer. This information is there-