24 changes from another beta agonist to fenoterol as a result of the admission, and 46 changes in the other direction (most of the latter patients were switched to salbutamol). On the other hand, there were substantial changes for other classes of asthma drugs. In particular, as a result of the admission, the proportion of patients prescribed oral corticosteroids increased from 28% to 62%. Thus patients were often prescribed prophylactic medication as a result of their severe attack, but there is no evidence that sicker patients were switched to fenoterol.

The comment about substandard care in the United States among blacks is not relevant to our findings in the Maori. It is well known that Maori have a higher asthma death rate than non-Maori. We have shown something quite different: that the Maori who use fenoterol have a higher death rate than the Maori who do not use fenoterol. The implication that this finding is due to confounding by ethnicity is nonsense as the comparison was made within the one ethnic group.

More generally, are Professors Spitzer and Buist suggesting that the standard of medical care for New Zealand declined so rapidly in 1976 (when fenoterol was introduced) that this accounted for a doubling of the mortality in two years? If so, what evidence do they have for this? Do they also imply that the standard of medical care also declined suddenly in the six countries which had mortality epidemics in the 1960s when isoprenaline forte was introduced? Furthermore, if any bronchodilator can be harmful when prescribed in the context of poor medical care, why did New Zealand not see an epidemic when salbutamol was introduced?

Professors Spitzer and Buist suggest that we have challenged the scientific community with the possibility that good drugs when used poorly may be potentially harmful. We have not. The scientific community already knows this. Rather we have suggested that a poorly selective $beta_2$ agonist, which is more potent than salbutamol[5] but available by metered dose inhaler at twice the dose of salbutamol, may have been responsible for an epidemic of asthma deaths in young people with severe asthma in New Zealand.

Rather than repeatedly raising the same criticisms of our work, Professors Spitzer and Buist, or Boehringer Ingelheim, should inform the scientific community of the following: (1) Why was fenoterol marketed as a "forte preparation" (200 μg/puff compared with salbutamol at 100 μg/puff) when it was known to be more potent and to have greater cardiac effects than other commonly available beta agonists? (2) Why was the nebuliser formulation made available in New Zealand (5 mg/ml) five times the concentration used in Canada (1 mg/ml)? (3) Why was fenoterol never licensed in the United States? (4) Why was fenoterol marketed as a highly selective $beta_2$ agonist when Boehringer Ingelheim's own funded experiments had indicated that in the clinical situation it was no more selective than orciprenaline,[6] the poorly selective agent it was designed to replace?

In conclusion, we concur with the independent report commissioned by the New Zealand Health Department that the evidence proposed by the Boehringer Ingelheim reviewers in favour of the confounding hypothesis is indirect, circumstantial, and considerably subjective.[7] Although further research would clearly be valuable we also agree with the New Zealand Health Department's conclusion that the current balance of evidence is now in favour of a causal association between fenoterol use and asthma mortality. As a result, the New Zealand Minister of Health has moved to severely restrict the availability of fenoterol by removing it from the Drug Tariff, and a similar policy has now been adopted in Australia.

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Local anaesthesia for fibreoptic bronchoscopy

Dr AC Davidson and colleagues (March 1990;45:239) were impressed with the local anaesthesia produced by a transcricoid injection of 4–6 ml 5% cocaine (200–300 mg). They went on to state that they were unaware of a formal comparison of cocaine and lignocaine as local anaesthetics during bronchoscopy but recommended that other centres consider changing to the transcricoid instillation of cocaine for fibreoptic bronchoscopy.

In a double blind, randomised study of 60 patients we recently compared the local anaesthetic effects of intratracheal injections of lignocaine (4 ml of 4%: 160 mg) with cocaine (4 ml of 2·5%: 100 mg)[1]. Local anaesthesia was assessed by numbers of coughs, operator acceptability, and patient discomfort; in all areas cocaine scored only slightly better than lignocaine (for example, there was a mean of eight coughs per procedure with cocaine compared with 11 with lignocaine), none of the differences achieving statistical significance. The impression of Dr Davidson and colleagues of the superiority of cocaine may reflect their use of a dose two to three times higher than in our study. It has been recommended that no more than 1·0–1·5 mg/kg cocaine should be applied to mucous membranes in adults, while others have suggested a maximum dose as low as 50 mg[2]; the use of higher doses may increase the risks of toxicity.

We agree with Kinnear *et al*[3] that transcricoid injections of local anaesthetic are well tolerated and produce effective local anaesthesia for fibreoptic bronchoscopy. When used in the doses recommended above cocaine and lignocaine appear equally effective.

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AUTHOR'S REPLY We are grateful to Dr Teale and colleagues for drawing our attention to their study of local anaesthesia for bronchoscopy, which we had overlooked. The main point that we were making was that patient tolerance is dependent on the effectiveness of local anaesthesia and two studies now attest to the superiority of the transcricoid route.[1,2] Our impression of the greater effectiveness of cocaine may be the result of our use of a higher dose (200–300 mg) than that used by Dr Teale and colleagues in their study (100 mg); it is interesting that a trend towards a superiority of cocaine is apparent in their abstract. The question of the safe maximum dose of topical anaesthetic agents is controversial and surveys of practice in the UK suggest the use of more lignocaine at bronchoscopy than many authorities recommend. That this is safe practice is suggested by a study demonstrating serum concentrations well below the toxic range with topical doses in excess of 500 mg,[3] presumably because only a proportion of the administered dose is actually absorbed through mucus membranes. The recommended doses quoted by Dr Teale are unrealistically low and the *British National Formulary* recommends a maximum topical dose of 3 mg/kg for cocaine. We now aim to measure serum cocaine levels to ensure that the doses we employ do not produce toxicity. An internal audit of complications in nearly 1000 bronchoscopies has, however, shown no evidence of cocaine induced neurological or cardiac toxicity.

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BCG vaccination of schoolchildren in England and Wales

One aspect of discontinuing routine BCG not discussed by Drs V H Springett and I Sutherland (February 1990;45:83–8) is the possible increase in mortality which may result.

Using published data[1,2] from the Office