support our hypothesis; specifically, a threefold increase in suicide rates in 15–24 year old men and a twofold increase in 15–24 year old women between 1970 and 1988.[2] Of interest, the most dramatic increase in suicides occurred between 1976 and 1977 in both 15–24 and 25–44 year old men – the first year of the asthma epidemic. Even in the UK, where there are no obvious financial barriers to health care, poverty has been shown to be associated with an increased risk of severe asthma[3] and such patients would be expected to have been further disadvantaged if they were confronted with escalating costs of primary health care such as those which have been imposed increasingly since the 1960s in New Zealand.

The direct costs of managing asthma in New Zealand are substantially higher for the individual patient than in any other developed country where such studies have been performed.[4,5] Using the same methods as Mellis *et al*[5] we found that up to 30% of the direct costs of managing asthma are borne by the patient in New Zealand (figure). Further, only a small proportion of the direct costs of managing asthma in New Zealand is spent on primary health care or specialist care, the majority being spent on medication. In the late 1970s, therefore, most of the financial support was for bronchodilator therapy (as opposed to inhaled steroids) and secondary health care with very little funding of primary health care. A multivariate linear regressional analysis of data collected in 1993/4 has shown that those on oral theophylline ($p = 0{\cdot}0006$) and with the highest serum salbutamol levels ($p = 0{\cdot}03$) had the most severe asthma at the time of attendance at two New Zealand emergency departments.[6] In the 1990s, therefore, some of the factors which contributed to the epidemic in the 1960s and 1970s are still apparent – namely, high unemployment, financial barriers to primary health care, and overreliance on bronchodilator therapy for the management of acute asthma (though acknowledging that this trend in behaviour may have improved). Such factors would be expected to have reduced the effectiveness of the various strategies developed to improve asthma care![7]

The evidence that inhaled corticosteroids reduce morbidity and thus mortality is overwhelming. In one prospective randomised study in a GP setting acute attacks were virtually abolished with the introduction of inhaled steroids.[8] In the most comprehensive randomised study of asthma education undertaken to date[9] we have shown significant improvement in knowledge and self management skills for those randomised to a community-based education centre, but no change in behaviour or hospital admission rates. Despite this, there was a 64% reduction in hospital admissions in the community targeted during the study, and one factor shown to have changed substantially was the sale of inhaled steroids (1·4 million to 2·7 million dollars) (table).

Asthma clinics had been established in most urban settings in New Zealand by 1985. Non-attendance rates for new references to our asthma clinics in Auckland are about 35% (not 80%).[10] Although we have not validated the efficacy of these multidisciplinary outpatient clinics directly, Allen *et al*[11] have. They had a similar non-attendance rate (38%) to us, despite which they were able to show a significantly lower readmission rate (OR 2·3) analysed on an intention to treat basis for those randomised to the clinic. Mayo *et al*[12] found a threefold reduction in readmission rate over three years in a group of patients randomised to intensive treatment compared with usual treatment within the outpatient setting, and this difference was largely due to the fourfold increase in use of inhaled corticosteroids in the intensive treatment group. This would explain why readmission rates began to drop in New Zealand in 1987 within two years of high dose inhaled steroids being registered for use by specialists only and two years before the debate on inhaled fenoterol began.

Whilst no study has yet shown benefit from changing from regular inhaled bronchodilator therapy to that of as required use only, there are many studies which show substantial benefit from the introduction of higher dose inhaled steroids. The major escalation in inhaled steroid sales in New Zealand therefore remains the most logical explanation for the reduction in both morbidity and mortality, particularly in view of the continued increase in sales of inhaled β agonists.

JEFF GARRETT
JOHN KOLBE
GLENN RICHARDS
TOBY WHITLOCK
HARRY REA
Department of Respiratory Services, Green Lane Hospital, Auckland 3, New Zealand

1 Kolbe J, Vamos M, Elkind G, Garrett JE. Socioeconomic and psychological factors and admissions for acute severe asthma (abstract). *Aust NZ J Med* 1995 (in press).
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In their review of asthma morbidity and mortality in New Zealand (March 1995;**50**:303–11) Dr Garrett and his colleagues argue that withdrawing fenoterol as a prescription medicine in New Zealand in late 1989 was only one among many factors that led to an overall improvement in morbidity and mortality from asthma in New Zealand. They cite no less than 11 initiatives which they consider to have been important contributors to the reductions in hospital admission and mortality rates which have occurred in New Zealand over the last 10 years, but exclude the withdrawal of fenoterol as one of them. Why?

We have no doubt that each of the initiatives discussed was necessary and important and contributed to the decline in mortality rates for some considerable time before 1989. However, in their evaluation of trends they do not account for the fact that first (fig 5) and total (fig 7) hospital admission rates remained obstinately unchanged until 1989, despite the fact that all of the measures outlined in their review were implemented on a nationwide basis well before that time.

The only new strategy adopted in 1989 was the withdrawal of fenoterol followed by recommendations to use β agonists as required rather than as regular treatment. This practice is now accepted in international guidelines for the management of asthma. The first of these steps was prompted by epidemiological data from Wellington,[1] and the second by the results of a study conducted in Dunedin which showed that control of asthma deteriorated when fenoterol was given regularly rather than as required to patients with mild to moderate asthma.[2] The results of these investigations were complementary. They indicated not only an increased risk of death from asthma among patients receiving inhaled fenoterol, but also that the underlying cause was likely to be an increase in baseline severity when inhaled fenoterol was being taken regularly. We have recently discussed the relationship between β agonist use and asthma morbidity and mortality in greater detail.[3]

It is therefore quite understandable that improvements in the delivery of and access to medical care – by whatever mechanisms – reduced asthma mortality before 1989, but the available data indicate that they had little impact on improving severity and hence morbidity, particularly first hospital admissions. Only when the major factor affecting severity was removed was there a subsequent reduction in morbidity.

DR TAYLOR
CS WONG
Department of Medicine, University of Otago Medical School, P.O. Box 913, Dunedin, New Zealand

1 Crane J, Pearce NE, Flatt A, Beasley R. Prescribed fenoterol and death from asthma in New Zealand. *Lancet* 1989;i:917–22.
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AUTHORS' REPLY Drs Taylor and Wong are correct in stating that it is our contention that the dramatic decline in asthma morbidity and mortality in New Zealand was due to multiple factors and that the precise contribution of individual factors cannot be accurately estimated retrospectively. However, on the one hand they agree that the possible contribution of the withdrawal of fenoterol was acknowledged, but immediately make the accusation that we "exclude(d) the withdrawal of fenoterol as one of them".

In a retrospective cohort study we have also shown that fenoterol was associated with an increased risk of fatal or near-fatal asthma (crude RR = 2·1).[1] However, because of more accurate, complete and detailed information on asthma severity we were better able to control for confounding by severity. After controlling for multiple risk factors, fenoterol was no longer associated with an increased

risk of severe adverse event (adjusted RR = 1·0) – that is, increased risk was entirely due to patients with more severe disease being prescribed fenoterol. The possibility that fenoterol was intermediate in the causal pathway (and thus responsible for an increased admission rate) was addressed by demonstrating that the lower strength preparation of fenoterol (100 μg in combination with ipratropium) was associated with a higher risk of serious adverse event (crude RR = 2·2, 95% CI 0·6 to 10) than the higher strength preparation (200 μg) (crude RR = 1·5, 95% CI 0·5 to 6·1), thus demonstrating biological implausibility.

It is incorrect of Drs Taylor and Wong to imply that all changes in morbidity and mortality occurred only after 1989; fenoterol was withdrawn in New Zealand in November 1989, but mortality had been steadily declining since 1981 (fig 7) and readmissions for asthma began to fall in 1987 (fig 5). However, first admissions for asthma began to fall only after 1989. Hence there appears to be a hierarchy in the response of mortality and indices of morbidity to intervention strategies. This is not at all surprising when one considers the nature of the interventions, the fact that they are usually initiated by hospital-based specialists and then "filter" out to the community, and that the initial strategies were directed at those at obviously highest risk (previous severe life threatening attacks or recurrent hospital admissions). The reduction in readmission rates (which began in 1987) followed by about two years the availability of high dose inhaled steroids to specialists, most of whom were in hospital based practice. Furthermore, the reduction in first admissions began about two years after high dose inhaled steroids became generally available to doctors in the community.

From our review it is patently incorrect and misleading to suggest that "the only new strategy adopted in 1989 was the withdrawal of fenoterol followed by recommendations to use β agonists as required . . .". The publicity associated with the "fenoterol debate" heightened general awareness about asthma morbidity and asthma management and led to continued initiatives along the lines outlined, particularly in the areas of asthma education and a multidisciplinary approach to the problem. A reflection of more fundamental changes in asthma management is the significant increase in sales of inhaled corticosteroids, specifically high dose preparations, after 1989 (fig 6). Although we have little information on how individuals are using inhaled β agonists now compared with the 1980s, we agree that patients are likely to have become more conservative in their use as a result of public awareness about the potential dangers of overuse of inhaled β agonists created by the fenoterol debate in New Zealand in 1989 and 1990. It is salutary to point out that these declines in morbidity and mortality occurred despite a continued increase in the total sales of inhaled β agonists in New Zealand.

JEFF GARRETT
JOHN KOLBE
GLENN RICHARDS
TOBY WHITLOCK
HARRY REA
Department of Respiratory Services, Green Lane Hospital, Auckland 3, New Zealand

1 Garrett J, Lanes S, Kolbe J, Rea H. Risk of severe life-threatening asthma (SLTA) and death and type of prescribed β-agonist. *Am J Respir Crit Care Med* 1995 (in press).

BOOK NOTICES

Arterial Chemoreceptors: Cell to System. Ronan G O'Regan, Philip Nolan, Daniel S McQueen, David J Paterson. (Pp 400; $95.00). New York: Plenum Press, 1994. 0 306 44824 6.

This book provides "an account of the papers" presented to the 12th International Meeting on Arterial Chemoreception held in 1993 in Dublin, under the aegis of the International Society for Chemoreception. It is a very good book . . . of its kind – and while some concerns may properly be raised about "its kind", these will hardly diminish the value of this text for anyone interested in ventilatory or circulatory control in general and arterial chemoreceptors in particular.

The volume comprises 64 concise reports by the contributors to the Dublin meeting. These are typically three pages long, including references and one figure, and are clustered under the section headings of: historical perspectives; molecular and ionic mechanisms of chemotransduction; neurotransmitters and putative neurotransmitters in the carotid body; chemosensory discharges; chemoreceptor reflexes; developmental aspects of chemoreception; morphological studies of the carotid body; and airway receptors. The list of authors is impressive – mostly a "who's who" of arterial chemoreceptor research.

The volume is bolstered by 10 invited reviews, each averaging 12 or so pages. These authors were therefore afforded the space to place the relevant issues into appropriate conceptual context. While it is perhaps unfair to single out a particular chapter from this very good collection of reviews, one feels compelled to draw attention to "International Meetings on Chemoreceptors: Historical Perspectives" by O'Regan and Nolan for their perceptive analysis of the developing (and fading) themes in almost half a century of chemoreceptor research. I have no doubt that it will prove to be a continuing source of valuable historical information, especially for young investigators preparing theses or dissertations on chemoreceptors.

While the review section of the volume may be unreservedly recommended, the section devoted to the concise reports of the presented papers justifies some reservations – precisely because of the concision. The space constraints do not allow the implications of the new results to be developed thoroughly or, in some instances, for contrasting viewpoints even to be introduced. One can imagine lively discussions on these presentations. The necessary consequence is the added editorial burden of ensuring that significant challenges to the authors' viewpoints arising from these exchanges are actually represented in the short discussion section. The papers themselves, however, are highly informative, reflect (and in large part define) the current "state of the art", and cover an impressive range of chemoreceptor-related topics, fully justifying the "Cell to System" subtitle.

The editorial standards are high, despite the occasional lapse such as allowing a figure to be attributed to the authors of a review chapter on the topic rather than to the authors of the original research paper from which it had been reproduced in the review.

I would recommend to anyone interested in arterial chemoreceptors that this book be put on their "high priority list" as a valuable source of up-to-date information from major investigators in the field. The volume is dedicated to Eric Neil (a charming sketch of whom graces the forepage); I judge it a further compliment to the enterprise to believe that it would have met his exacting standards. – BJW

Pulmonary Function: A Guide for Clinicians. Gabriel Laszlo. (Pp 245; £22.95 (US$37.95) paperback; £45.00 (US$69.95) hardback). Cambridge: Cambridge University Press, 1994. 0 521 44679 1 (paperback). 0 521 43050 X (hardback).

When approached for the first time, many find pulmonary physiology a complex and daunting subject. This most recent addition to the bookshelves now sits alongside more established texts which aim to enlighten.

Any new book, in order to be successful, needs to cover its subject in a new and original way, or be aimed at an audience previously poorly catered for. This book declares itself to be aimed at postgraduates entering the field of respiratory medicine, as well as all clinicians, scientists, and technical staff working with patients in the pulmonary function laboratory. In an attempt to achieve this broad aim, early chapters cover lung function testing and the physiological principles on which these tests are based. These are then followed by a number of chapters on pulmonary function in specific respiratory disorders, and chapters on physiological principles and testing in common clinical situations in respiratory medicine such as respiratory failure. Further chapters cover exercise testing, theoretical aspects of oxygen and carbon dioxide exchange, and the control of ventilation. The book ends with an overview of the rapidly expanding and changing field of sleep-related disorders of breathing, the investigation of which places an ever increasing workload on many lung function laboratories.

This volume is probably of greatest interest to those stated as the primary target – namely, physicians entering the field of respiratory medicine – for whom the disease and problem orientated chapters are of particular use for reference. The use of mm Hg in preference to kPa (though both are quoted) may just be the author's preference, but suggests that North America is seen as a target area. As with any text, the personal style of the author will appeal to some more than others, and I would suggest a quick trip to the library before purchase (do not be put off by the small errors in the first chapter). In paperback, particularly, this book represents good value for money. – JESW

NOTICE

1st European Forum of Quality Improvement in Health Care

The 1st European Forum of Quality Improvement in Health Care will be held at the QEII Conference Centre, London on 7–9 March 1996. It will allow the exchange of ideas on quality improvement in health care and provide education. The forum will consist of plenary lectures, parallel seminars and workshops and discussions and short educational courses. For more information contact: Clare Moloney, BMA Conference Unit, BMA House, Tavistock Square, London WC1H 9JP. Fax: 0171 383 6663. Tel: 0171 383 6478.