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 1988.1 Of interest is the increase in asthma-related hospital admissions 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 asthma2 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.3 Using the same methods as Mellis et al we found that up to 30% of the direct costs of managing asthma are borne by the patients in New Zealand (figure). In New Zealand, 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 regression analysis undertaken in 1993 has shown that those on oral theophylline (p = 0.0006) and with the highest serum salbutamol levels (p = 0.03) had the most severe asthma at the time of attendance at two New Zealand emergency departments. In the late 1970s, before the use of beta agonists, many of those with severe asthma were not taking any medications. In 1993, one third of those with the most severe asthma were not taking any medications. In 1993, one third of those with the most severe asthma were not taking any medications.

In the most comprehensive randomised study of asthma education undertaken to date 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 costs in the community participants in the intervention group. This suggests that education programmes for asthma management could reduce the cost of managing asthma. The major factor contributing to the increase in asthma-related hospital admissions was the introduction of inhaled steroids. In the 1980s, asthma-related hospital admission rates increased two-fold. In 1989, asthma-related hospital admissions were the highest in New Zealand. In 1990, asthma-related hospital admissions were the highest in New Zealand. In 1990, asthma-related hospital admissions were the highest in New Zealand.

In our 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 aspect of a range of measures which have contributed to the overall improvement in asthma morbidity and mortality in New Zealand. The city of Asthma has 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 in asthma mortality rates, they do not account for the fact that the 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 beta 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 are not surprising. 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 morbidity when inhaled fenoterol therapy was taken regularly. We have recently discussed the relationship between beta agonist use and asthma morbidity and mortality in greater detail.3 It is therefore quite understandable that improvements in asthma management 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, the use of fenoterol, was removed there was a subsequent reduction in morbidity.

In a prospective study we have also shown that fenoterol was associated with an increased risk of fatal or near-fatal asthma (crude RR = 2.1).4 However, because of more accurate, complete and detailed information on severity and asthma-related hospital care, the control for confounding by severity. After controlling for multiple risk factors, fenoterol was no longer associated with an increased

AUTHORS’ REPLY

Dr 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 a range of factors, as we have acknowledged, but immediately make the accusation that we “exclude(d) the withdrawal of fenoterol as one of them”.

In a prospective study we have also shown that fenoterol was associated with an increased risk of fatal or near-fatal asthma (crude RR = 2.1). However, because of more accurate, complete and detailed information on severity and asthma-related hospital care, the 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 increased 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 'hockey stick' in the postmarketing trends and the nature 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 "fades" with 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" heighten general awareness about asthma morbidity and asthma management and led to continuing changes 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 anti- 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 1980's, 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 satisfactory 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.

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BOOK NOTICES


This book presents "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 this kind - and while some concerns may properly be raised about "its kind", they 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 chemoreception; chemoreceptors and neurotransmitters; chemoreceptor reflexes; developmental aspects of chemoreception; and clinical studies of 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 of this 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" theme.

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 Neill (a charming sketch of whom graces the forepage); I judge it a fitting compliment to the enterprise to believe that it would have met his exacting standards. - BJW

Pulmonary Function: A Guide for Clinicians. Gabriel Lazzlo. (pp 245; $22·95) (US$37·95) paperback; £6·00 ($US96·95) 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 undergraduate nurses 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 followed by a number of chapters on pulmonary function in specific respiratory disorders, and chapters on thoracic radiology. The book ends with an overview of the rapidly expanding 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 oriented chapters are of particular use for reference. The use of imperial units is prevalent throughout, 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 and not to 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 November 1994. It will provide an opportunity to discuss issues on quality improvement in health care and provide education. The forum will consist of plenary lectures, parallel seminars and workshops and this cover lung function, technical courses. For more information contact: Clare Moliney, BMA Conference Unit, BMA House, Tavistock Square, London WC1H 9JP. Fax: 0171 383 6663. Tel: 0171 383 6478.

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