Correspondence

Prevalence of bronchial reactivity to inhaled methacholine in New Zealand children

SIR,—The paper by Dr MR Sears and others (March 1986;**41**:283–9) on bronchial reactivity and symptoms in New Zealand schoolchildren is to be welcomed. We feel, however, that the authors do an injustice to their results if their conclusion is simply that tests of non-specific bronchial responsiveness are of little use in epidemiological studies of asthma. Their results, describing the epidemiology of this phenomenon, are of great interest to those investigating the aetiology of symptoms in hyperresponsive individuals.

We find that some of the conclusions reflect illogicality of a sort that has crept into many papers on this subject. Measurement of non-specific bronchial responsiveness is not a test for the presence or absence of a phenomenon but simply a measurement in its own right, such as a measurement of height. To say that a subject does not have bronchial reactivity is no more logical than claiming that a man has no height because the ruler is too short to measure him. It has recently become clear that there is an overlap between degrees of bronchial responsiveness in the normal and asthmatic population when measured at a single point in time since no studies have yet been published where such measurements have been made repeatedly in these two groups over a period of time. On the basis of this concept, the term bronchial hyperresponsiveness might be defined as a degree of methacholine or histamine responsiveness not present in 95% of the symptomless population.

While the authors' conclusion that measurement of bronchial responsiveness is not useful for refuting or confirming a diagnosis of asthma is certainly correct, this measurement remains a useful research tool and may even be of some benefit clinically in certain patients to monitor response to treatment. Differences in the spectrum of bronchial responsiveness to methacholine between populations, between different groups within populations, and within individual subjects with time may all be important in elucidating the aetiology of asthma and other chronic forms of obstructive airways disease.

The authors' concluding remarks on the benefits of a detailed history are attractive but no more practicable. In their own study more than one quarter of the children studied had a history of current or past wheeze and many more a history of cough. Clearly, not all of these children had asthma, whereas the drawing of a line through any one symptom group is as arbitrary as using a particular concentration of metha-choline or histamine to define hyperresponsiveness. If the relationship of hyperresponsiveness and symptoms to the pathogenesis of airflow obstruction in children is to be better understood, it is important that the relationship of symptoms to the physiological abnormality is sought.

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***This letter was sent to Dr Sears, who replies below.

SIR,—Drs Clifford and Holgate have interpreted our paper as saying simply that tests of non-specific bronchial responsiveness are of "little use in epidemiological studies of asthma." Our conclusion was that "bronchial challenge by methacholine inhalation was not sufficiently sensitive or specific to be useful as a major criterion for the diagnosis of asthma in epidemiological studies" (of the cumulative prevalence of asthma in a childhood population). We suggested that a carefully taken history remains the best available tool for determining the prevalence of asthma. We agree, however, that bronchial responsiveness is a useful and very interesting measurement in its own right.

As reported, some children had histories compatible with asthma but their measured airway responsiveness did not fall within the usually accepted "asthmatic" range, while other children with no history of respiratory symptoms showed mildly or moderately increased responsiveness. For detecting subjects in our cohort with any history of recurrent wheezing, tests of airway hyperresponsiveness (defined as reversal of spontaneous airflow obstruction by salbutamol or PC_{20} $FEV_1 \leqslant 25$ mg/ml methacholine by our method) had a sensitivity of 50% and a specificity of 84%; for detecting subjects with "current" asthma (symptoms within the past year), the sensitivity of the test was 59% and the specificity 83%. Using a lower concentration of methacholine to define hyperresponsiveness increased the specificity of the test but reduced the sensitivity considerably.

We found that the degree of bronchial responsiveness correlated broadly with both a history of wheezing and the interval since symptoms were last experienced. Had we been able to measure airway responsiveness at the time when symptoms were present, the sensitivity and specificity of the test may have been much greater. Airway hyperresponsiveness is closely related to the severity of asthmatic symptoms and therapy needed for control,[1] and reflects immediate asthma more reliably than past asthma or even asthma within the past year.

As Drs Clifford and Holgate point out, our study showed that there was an overlap in airway responsiveness between "normal" and "asthmatic" populations when measured on one occasion. We are presently completing studies in which we have measured airway responsiveness in most of these children on three occasions over four years. These data may provide more insight into the relationship between a history consistent with current or past asthma and persistent or transient airway hyperresponsiveness. Defining airway hyperresponsiveness as the degree of responsiveness not present in 95% of the "symptomless" population would exclude a small proportion of currently asthmatic subjects who by history and spirometric testing clearly have variable airflow obstruction; further, some "symptomless" children (or adults) with hyperresponsiveness might have asthma with variable airflow obstruction but be poor perceivers of symptoms. Further prospective studies in other populations and age groups are required to determine the epidemiology of airway hyperresponsiveness and its relationship with asthma and asthma like symptoms.

In their last paragraph, Drs Clifford and Holgate highlight the problem that we sought to resolve by including

bronchial challenge testing in our study of the prevalence of asthma. In commenting on our finding that over one quarter of children had a history of current or past wheeze, they state that "clearly, not all of these children had asthma." What did they have? Many but not all of these children showed increased airway responsiveness, as did a proportion of those with a history of dry cough. If, as advocated by Woolcock and colleagues,[2] asthma is diagnosed only when symptoms are accompanied by demonstrable airway hyperresponsiveness, much past asthma, and even mild current asthma, will not be diagnosed. Perhaps our problem is the use of the word "asthma." We can demonstrate and measure degrees of bronchial responsiveness, and we can obtain and clinically categorise a history of wheezing or cough. While symptoms and hyperresponsiveness are often found together, they are not synonymous, and neither is exactly equivalent to variable airflow obstruction, which is the hallmark of asthma. We should report both the history of symptoms and the degree of airway responsiveness rather than use their dual presence to make a diagnosis of a condition whose precise definition continues to elude us.

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1 Juniper EF, Frith PA, Hargreave FE. Airway responsiveness to histamine and methacholine: relationship to minimum treatment to control symptoms of asthma. *Thorax* 1981;**36**:575–9.
2 Britton WJ, Woolcock AJ, Peat JK, Sedgwick CJ, Lloyd DM, Leeder SR. Prevalence of bronchial hyperresponsiveness in children. The relationship between asthma and skin reactivity to allergens in two communities. *Int J Epidemiol* (in press).

Book notices

Recommended Health Based Limits in Occupational Exposure to Selected Mineral Dusts (Silica, Coal). WHO Study Group 734. (Pp 82; Sw Fr 12.) Geneva: World Health Organisation, 1986. ISBN 92 4 120734 5.

This report is the latest of a series of publications produced by groups of experts for the World Health Organisation. Its object is to make recommendations for exposure limits to fibrogenic mineral dusts, including coal and silica. The group initially defines pneumoconiosis and details the factors that influence the retention and elimination of airborne particles in the respiratory tract and their eventual fate. The methods of measuring airborne particulates are clearly described. Subsequent sections describe the pathology, pathogenesis, clinical manifestations, and complications of silicosis and coalworkers' pneumoconiosis. The published evidence relating exposure to dust and the development of pneumoconiosis is comprehensively reported. The group finally makes recommendations of exposure limits for free crystalline silica and coalmine dust and regarding the surveillance of the workers and their environment. This publication is short and easy to read. It provides comprehensive current information on two of the pneumoconioses with appropriate references. It is recommended for those practitioners directly concerned with this industry.—CACP

Inhaled Aerosol Bronchodilators. ER McFadden jun. (Pp 122; £25 softback.) Baltimore: Williams and Wilkins, 1986. ISBN 0-683-05867-3.

This is a small, compact paperback which, although primarily about aerosols and delivery systems, covers many aspects of drug therapy in the treatment of diseases of airflow obstruction. The book is unusual in that it has no foreword by the author, and thus leaves the reviewer undecided on the audience the author intended to reach—a problem exacerbated when he is reviewing North American publications for British readers and not entirely resolved after it has been read in its entirety. The history of inhalation therapy is reviewed, and followed by an excellent chapter on aerosols, their deposition and generation. This is followed by a discourse on the pharmacokinetics of inhaled substances, which does highlight the present paucity of data on bronchodilators. Patients' and doctors' errors in the use of hand held aerosols and means of overcoming such problems are well described. Metabolism, structure, and function followed by pharmacology make up the major portion of the book, but the inherent safety of selective β stimulants is also covered. Drug dose differences between the various delivery systems are highlighted. The vexed problem of bronchial tachyphylaxis to selective β stimulants is reviewed, and sensibly dismissed as of little clinical relevance in asthmatics. The remainder of the book deals with drug interactions and specific problems such as aerosol use in pregnancy and exercise induced asthma. The book is clearly written, remarkably readable, and illustrated clearly and mainly appropriately. The bibliography is up to date and extensive, suggesting that the author is aware of the limited readership. I believe that the book should be read by all medical students and general practitioners, but it is probably of less interest to the specialist thoracic physician. The bibliography, however, extends the readership to all trainee thoracic physicians. I am pleased to have the book for the undergraduate and postgraduate library, but at £25 it is an expensive book for the solitary purchaser.—GMC

Correction

Surgical pathology of the thymus: 20 years' experience
We have learned of the following errors in the references to the paper by Mr S Large and colleagues (January 1986; **41**:51–4). Reference 5 should read: Wychulis AR, Payne WS, Clagett OT, Woolner LB. Surgical treatment of mediastinal tumours. A forty year experience. *J Thorac Cardiovasc Surg* 1971;**62**:379–92. It is stated in error in the text that this work was published in 1964. In reference 6 "Claget" should read "Clagett."