J G SCADDING on 2 May 1987. 2024 by guest. Protected by copyright

was necessary and doubts about its applicability to a patient who when seen shows little variability of airflow resistance.

Some patients with asthma have a special liability to type 1 IgE mediated hypersensitivity reactions, and it used to be customary to refer to allergy in defining asthma. But these are a minority; and not only they but other patients with asthma and some with other respiratory disorders show abnormal bronchoconstrictor responsiveness to many chemical and physical stimuli. This has been called non-specific bronchial or airway reactivity; quantitative tests for it may use physical agents, such as cold air and non-isotonic aqueous aerosols, but most look for enhanced responses to pharmacological bronchoconstrictors such as histamine or methacholine.7-9 The smallest dose that induces a specified diminution in expiratory airflow may be determined. These tests thus use the same sort of physiological measurement as the 1971 definition of asthma.

Two groups, both from Southampton, have sought to clarify these issues by population studies. Mortagy et al surveyed 2145 subjects sampled from an electoral roll. 10 They sent a postal questionnaire about respiratory symptoms; took random samples from four groups of respondents, three with selected combinations of symptoms and one denving symptoms; and performed spirometry and a test of bronchial reactivity to histamine in these. No subject without symptoms was hyperreactive to histamine by their test. Among 51 with shortness of breath, wheezing, or both, nine were hyperreactive; all of these had noticed that their symptoms were provoked by physical or chemical factors in air. In a second survey a simplified questionnaire identified 63 subjects with symptoms resembling those of these nine. Twenty two of these were challenged with histamine, and all were hyperreactive. Fifteen said that they had asthma currently and five said that they had had it, whereas only one subject among 68 in other symptomatic and asymptomatic groups gave a history of asthma. These studies led the authors to define a "bronchial irritability syndrome" as bronchial hyperreactivity to histamine together with one or more of three clinical features, provocation of symptoms by environmental factors, nocturnal dyspnoea, and tightness of the chest in the morning for more than one hour, and to claim that it constitutes a clinical entity.

What is meant by a "clinical entity" is as usual not clear, but it certainly carries essentialist overtones. Presumably it implies more than clinical recognisability. The authors suggest that the bronchial irritability syndrome might be useful in decisions about treatment; but these can be made more simply by trial of available agents controlled by spirometry. They also claim that the bronchial irritability syndrome is a clearer diagnostic term than asthma, but since in this paper asthma appears to be defined as the disease from which people who say they have asthma suffer this claim is hardly surprising. "Clinical entity" may suggest a common pathogenesis, but this cannot be claimed for the bronchial irritability syndrome. It must include some cases with episodic symptoms caused by IgE mediated reactions to identifiable allergens; a few with similar symptoms, eosinophilia, and possibly systemic vasculitis; many with persistent smoking induced airflow limitation; and some with none of these features.

Burney et al studied adults in two villages and one market

town by similar methods and added prick tests of skin reactivity to three common allergens. 11 One in seven subjects aged 18 to 34 had bronchial hyperreactivity to histamine by their test; the proportion was slightly lower in middle age, \leq and higher, one in four, over 55. Atopy, assessed by mean skin weal diameter, diminished with age. In the younger age groups bronchial reactivity to histamine was strongly correlated with atopy; this relation may be causal since nonspecific bronchial reactivity increases in atopic subjects after $^{\mathfrak{D}}$ challenge with allergen¹² and diminishes after avoidance of [7] exposure.¹³ In subjects over 45 bronchial reactivity was strongly correlated with smoking. There was no relation \vec{a} between smoking and skin test sensitivity.

These two studies raise the question whether bronchial 5 hyperreactivity might be a useful criterion for a diagnostic $\frac{\overline{\omega}}{2}$ category to replace asthma. Compared with the 1971 definition, which takes note of abnormal bronchoconstrictor abnormal responses to any stimulus, a definition that required response to a specified test of so called non-specific hyperreactivity would be restrictive. The bronchial irritability syndrome, as ω defined in the first study, is thus a subset of asthma. As the $\frac{1}{2}$ second study confirmed, the category so specified would be aetiologically diverse and thus need subcategorisation similar $\overset{\text{N}}{\circ}$ to that currently advocated for asthma.14

If we accept the rules of nominalist definition, which prevent us from regarding diseases as causes, 15 we can continue to use asthma as a diagnostic term.

Emeritus Professor of Medicine, University of London Correspondence to: 18 Seagrave Road, Beaconsfield HP9 1SY.

- Porter R, Birch J, eds. Identification of asthma. Edinburgh: Churchill Livingstone, 1971. (Ciba Foundation Study Group No 38.)

 American Thoracic Society. Chronic bronchitis, asthma and pulmonary emphysema: diagnostic standards for non-tuberculous respiratory disease. Am Rev Respir Dis 1962;85:762-8.
- 203-4.
 Popper KR. The open society and its enemies. Vol 2. London: Kegan Paul, 1945:chapter 11.

- Fopper RR. In open society and its enemies. Vol 2. London: Regain Faul, 1993: Enapter 11.
 Scadding JG. Talking clearly about broncho-pulmonary diseases. In Scadding JG, Cumming G, eds. Scientific foundations of respiratory medicine. London: Heinemann, 1981:727-34.
 Scadding JG. The classification of disease. In: Rose J, Mitchell JH, eds. Advances in medical computing. Edinburgh: Churchill Livingstone, 1975:5-9.
 Boushey HA, Holtzman MJ, Sheller JR, Nadel JA. State of the art: bronchial reactivity. Am Rev
- Respir Dis 1980;121:389-413. argreave FE, ed. Airway reactivity: mechanisms and clinical relevance. Hamilton, Ontario: McMaster University, 1980.
- 9 Hargreave FE, Ryan G, Thompson NC, et al. Bronchial responsiveness to histamine or ethacholine in asthma; measurement and clinical significance. I Allergy Clin Im 1981:68:347-55
- 10 Mortagy AK, Howell LBL, Waters WR. Respiratory symptoms and bronchial reactivity: identification of a syndrome and its relation to asthma. Br Med J 1986;293:525-9.
- Burney PGJ, Britton JR, Chinn S, et al. Descriptive epidemiology of bronchial reactivity in an adult population: results from a community study. Thorax 1987;42:38-44.
 Cockcroft DW, Ruffin RE, Dolovich J, Hargreave FE. Allergen-induced increase in non-allergic
- 12 Cockcroft DW, Ruffin RE, Dolovich J, Hargreave FE. Allerge Induced bronchial reactivity. Clin Allergy 1977;7:503-13.

 13 Platts-Mills T, Tovey E, Mitchell E, et al. Reduction of bronchial hyperreactivity during prolonged allergen avoidance. Lancet 1982;ii:675-8.

 14 Scadding JG. Definition and clinical categorisation. In: Clark TJH, Godfrey S, eds. Asthma. London: Chapman and Hall, 1983:1-11. Also in: Weiss EB, Segal MS, Stern M, eds. Bronchial between Roston: Little Brown, 1985:3-13.
- 15 Campbell EJM, Scadding JG, Roberts RS. The concept of disease. Br Med J 1979;ii:757-62.

Correction

The genetics of diabetes: from nightmare to headache

Professor Harry Keen's address is United Medical and Dental Schools of Guy and St Thomas's Hospitals (Guy's Campus), London SE1 9RT (11 April, p 917)