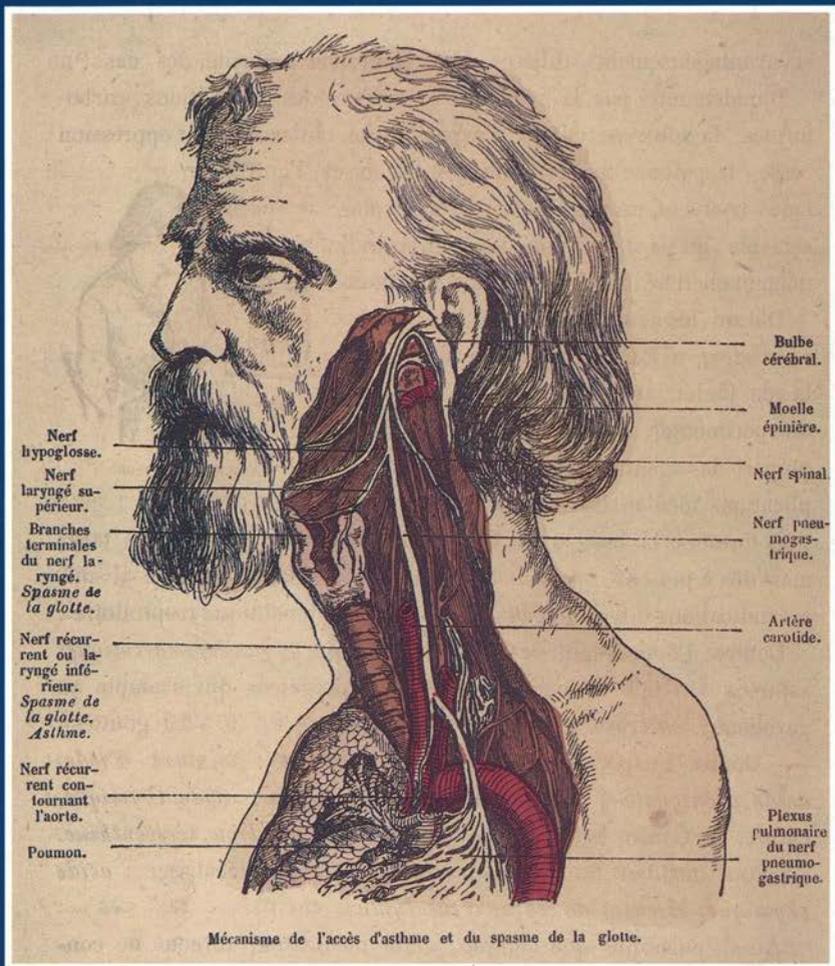


ASTHMA



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Office of Health Economics
12 Whitehall London SW1A 2DY

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Office of Health Economics

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To undertake research on the economic aspects of medical care.

To investigate other health and social problems.

To collect data from other countries.

To publish results, data and conclusions relevant to the above.

The Office of Health Economics welcomes financial support and discussions on research problems with any persons or bodies interested in its work.

Preface

It is fourteen years since the Office of Health Economics produced a paper on asthma (Studies of Current Health Problems No 57). During this time, asthma has been the focus for much research and more has become known about the distribution of asthma in the community, the disability that asthma causes, and the management of the condition, especially its prevention.

There is a perception that asthma is becoming more common, but whether this is because of a better understanding of the condition and improved diagnosis, or true proliferation, is not clear. Instruments for measuring air flow, for example, the peak flow meter, are more widely available now and clinic tests using more advanced techniques for measuring lung capacity and function (the vitalograph) and airway conductance (the plethysmograph) have been refined and are more widely applied. At the same time the importance of prophylactic medicines for asthma has been more fully recognised; this also has made it more desirable to diagnose cases which might previously have been given a more general respiratory label. While none of the changes which have occurred can be categorised as breakthroughs, the cumulative effect has been considerable.

The increased understanding of the disability that can be caused by asthma and the knowledge that much of this can be prevented or avoided is helping to remove the stigma from asthma as a disease and putting it into the category of a condition like migraine as something that cannot be cured but which can be successfully managed. It is therefore appropriate to review the situation once again to see what has been achieved in the intervening fourteen years, what still remains to be done and what the prospects are for improvements during the remaining years of this century.

George Teeling Smith

Introduction

In common with several other well-known conditions, such as epilepsy and migraine, asthma is not strictly a disease itself but a term used to describe a set of symptoms which result from the action of a number of incompletely understood mechanisms. The common factor is the hyperreactive nature of the airways which respond to a wide variety of stimuli.

Asthma is a word that comes to us from a Greek word meaning to breathe hard, or to pant. Hippocrates, in the fourth century BC described asthma but it was recognised 1,500 years earlier in China and was treated there with Ma Huang, the leaves and stems of the plant *Ephedra sinica*.¹ The alkaloid ephedrine, contained in species of *Ephedra*, was first effectively used for asthma in Western medicine in the 1920s, almost 4,000 years later.

In the second century BC Aretaeus of Cappadocia, who supposedly was the first to give an accurate description of an attack of asthma, acknowledged that if the symptoms increased the patient was liable to suffocate.² Moses Maimonides (AD 1134 to 1204) also alluded to the potentially fatal nature of asthma, especially if the patient ignored the rules of management and acted according to his own desires and habits.² He also recognised the idiosyncratic nature of the illness, including the tendency for attacks to occur at night,³ which was perceived by others in subsequent centuries.

In 1552, Cardan hinted at the possible hazard of feather pillows for asthmatics and in the same century, Van Helmont recorded cases that appeared to have been provoked by dust, the eating of fish and changes in the weather. However, until this century it proved to be extremely difficult to control attacks of asthma and attention became directed to obvious anxiety of patients during their attacks. This somehow led to the misconception that asthma is a psychologically based illness, a belief that still lingers in the prescription of sedatives, including barbiturates, which are inappropriate in this condition. Anxiety is an epiphenomenon of asthmatic attacks and is relevant only in so far as it may prolong symptoms through hyperventilation.

In contrast, others have not considered asthma as a potentially fatal illness and such was the teaching in medical schools well into the present century.² It was not until the well-published spate of deaths from asthma in the 1960s that attitudes began to change.

The advances made by medical science in the nineteenth and twentieth centuries have led to an understanding of the respiratory system and the scientific techniques which have been developed have allowed us to elaborate the process and control of breathing and its disordered course in attacks of asthma. The characteristic features of asthma are hyperreactivity of the trachea and bronchi with narrowing of the airways, which is variable and reversible. These properties are combined in the American Thoracic Society's definition of asthma:⁴

'Asthma is a disease characterised by an increased responsiveness of the trachea and bronchi to various stimuli, and manifested by a wide-

spread narrowing of the airways that changes in severity either spontaneously or as a result of therapy.'

The process of breathing consists of a muscular effort which enlarges the chest in all dimensions causing the lungs to fill with air. The muscles then relax and the expulsion of air depends largely on the elasticity of the lungs. In asthma there is narrowing of the airways through contraction of bronchial smooth muscle, blockage of the lumen with secretions and inflammation. The passive nature of normal expiration means that there is little that the patient can do to assist removal of gases from the lungs. The effort of breathing in the presence of this physical restriction usually produces a wheeze of variable audibility which may be accompanied by an irritating cough.

The classical asthmatic attack is relatively sudden in onset. However, there are considerable variations in the frequency, duration and severity of symptoms and this has important implications for the indiscriminate diagnostic labelling of people who experience breathing difficulties of this nature. Some individuals suffer attacks once or twice a year, lasting for a very short period of time, whilst others are in a state of chronic airway obstruction and develop recurrent severe exacerbations which may persist for several weeks.

Similarly the extent and permanence of the changes in the respiratory system are extremely variable. During remission the lungs may be clinically normal in cases of mild and infrequent asthma. At the other extreme respiratory obstruction may be readily detected even when a patient superficially appears to be free from symptoms. In addition to bronchial smooth muscle contraction, pathological investigations of patients dying *in status asthmaticus* often provide evidence of diminished elasticity of the lungs, swelling of the mucous membrane and excessive production of mucus.

This paper examines the nature and prevalence of asthma, at least in so far as it is possible to ascertain the latter given the lack of a generally accepted definition of the complaint and the problems of diagnosis at the extremes of age. It discusses the level of asthma mortality – which is low in relation to the overall morbidity generated by the condition – and the sudden increases in the number of deaths which have sometimes occurred. Consideration is also given to recent advances in the management of patients with asthma. Finally an attempt is made to calculate the costs of asthma in terms of both the expenditure incurred by the National Health Service and the social burdens imposed by the disorder.

The characteristics and natural history of asthma

The structural and functional changes in asthma are complex. Structural changes include damage to the cells (epithelium) lining the airways, thickening of the muscle of the airway wall, mucosal oedema and infiltration of cells, especially blood cells associated with inflammation (eosinophils, neutrophils and monocytes) into the air passages.⁵ The characteristic functional change is airway hyperreactivity in response to various stimuli.⁵ The mechanisms through which these changes occur are not completely understood but they probably involve chemical mediators such as histamine, leukotrienes, prostaglandins, bradykinins, sensory neuropeptides, platelet activating factor (PAF) and others, and interactions between these mediators and various cells in the airways.⁶

In asthma, narrowing of the airway is not due solely to constriction of the smooth muscle which lines it but it is also due to the accumulation of secretions and epithelial cells shed within the air passages. Furthermore, asthmatic subjects show increased lung epithelial permeability which might allow inhaled antigen or inflammatory mediators to penetrate more readily.⁷

Thus, the response of asthmatic subjects to various inhaled noxious stimuli is constriction of and inflammation in their airways.⁸ The mechanical consequence of constriction of the airways is hyperinflation of the lungs which helps to maintain patency at the expense of increased muscular work. Inflammation accounts for the increased ventilatory drive which results in alveolar hyperventilation (over inflation in the terminal airways of the lung) and dyspnoea (laboured breathing) which compromise delivery of oxygen to the tissues. Failure of patients with asthma to respond to a bronchodilator indicates the presence of continuing inflammatory activity.⁸ It is the inflammation in the airways which is associated with increased airway reactivity and worsening of the asthma.⁹

The distinction that was formally made between extrinsic asthma, in which an antigen-antibody reaction is involved, and intrinsic asthma, in which no allergic mechanism can be demonstrated by skin testing, is probably unhelpful. It has now been shown¹⁰ that immunoglobulin E (IgE), the antibody responsible for the immediate type of immune response, is markedly raised in the blood of nearly all patients with asthma and, therefore, there may be no basic difference between the so-called allergic ('extrinsic') and non-allergic ('intrinsic') forms of asthma. Atopy, a familial tendency to allergic disorders, is not related to persistent airway obstruction in asthmatic patients. In men, but not in women, increasing age is related to worsening of asthma.¹¹

Asthma is most prevalent in children, with an unexplained predominance among boys, and among older adults. The disease can begin to remit at any age.¹² Patients with asthma often have a history of atopy, that is, a familial tendency to allergies. Children with asthma usually have a complete remission of their disease unless the asthma is

severe, or associated with chronic sinusitis, nasal polyps or childhood eczema. If the onset of asthma occurs in childhood the outlook is better than if the onset is in adulthood. It has been found in Australian¹³ and British¹⁴ schoolchildren that only about one-fifth of those with wheezing at the age of eleven continued to wheeze throughout their life. The outlook for children with asthma is good with all but the most severely affected children, largely or completely losing their symptoms during the second decade of life.¹⁵

Remission of active disease becomes less common during middle age, but tends to rise slightly after the age of 60.¹⁶ Remission is closely related to the severity of the asthma with only 3 per cent of those with frequent attacks but nearly 40 per cent of those with less frequent symptoms being in remission after nine years.¹⁶ Quiescent asthma is very likely to relapse in later life.¹⁶

There is a circadian rhythm (24-hour cycle) underlying asthma which shows itself by an increase in bronchial hyperreactivity, disturbance of sleep, variable lung function at night and early morning tightness of the chest and wheezing.¹⁷

Environmental factors affecting asthma

Seasonal variation in the occurrence of diseases, including asthma, was recognised over 2,000 years ago when Hippocrates noted that asthma was more common and attacks were worse in the autumn.¹⁸ Autumn as the peak time for attacks of asthma in children has been confirmed in South East England¹⁹ and throughout England and Wales²⁰ as well as in Europe.²¹ Monthly admissions to hospital for attacks of asthma in children up to 14 years old were fewest in winter and rose in spring and early summer, although a trough of admissions was present in August.²⁰

There is evidence that climatic changes affect asthma. In Birmingham in 1983, it was noted that there was a striking increase in the number of patients admitted to hospital with acute attacks of asthma on two days in July.^{22, 23} The peak of admissions coincided with a violent thunderstorm when 26 asthmatic patients attended one hospital's casualty department over a 36-hour period compared with the normal rate for this number of 2 or 3 days;²² the situation elsewhere in Birmingham was similar.²³ There was a notable change in windspeed and relative humidity during the week preceding the sudden increase in hospital attendances with a steady rise in temperature and then a sudden fall when there was heavy rain. Air pollution was not a factor in this instance but a sudden increase in airborne fungal spores may have contributed to the outbreak.²³ Independent observations from Bermuda,²⁴ where airborne allergens and pollution tend to be absent, supports the view that episodes of acute bronchospasm in patients with asthma may be related to sudden changes in temperature and relative humidity.

The implications of these and other observations from elsewhere in the world are that there are mechanisms which link exacerbations of asthma to the weather and that this phenomenon is widespread and

independent of atmospheric chemical pollution and airborne pollen and fungal spores.²⁵

There are other environmental factors that can influence the frequency and severity of attacks of bronchospasm in patients with asthma. For example, in Barcelona it was noticed that outbreaks of asthma coincided with the release of dust from the unloading of soybean at the harbour.^{26, 27} Moreover, serum taken from patients with epidemic attacks of asthma contained antibodies specific for soybean antigen, whereas serum from matched control asthmatics without such episodic attacks, did not. There is also evidence from Canada that exposure to grain dust adversely affects lung function.²⁸ Dietary factors may also be important in those with a history of asthma as a high sodium intake may increase the reactivity of the airways.²⁹

It is commonly believed that emotional stress may either precipitate or aggravate attacks of asthma and therefore family relationships have been investigated in relation to asthmatic children. In one controlled study,³⁰ it was found behavioural disturbances and emotional stress were significant factors only in the small group of children with severe and continuing asthma. These children were those with serious chronic obstruction of the airways and with the most severe allergic manifestations. The families of these severely affected children showed the greatest evidence of emotional distress. Socioeconomic conditions were not significantly different in any group of children with asthma, compared with controls^{30, 31} but family social conditions may be an important determinant of whether wheezy children get access to appropriate medical advice and treatment for their condition.^{30, 33} This is important not only from the point of view of the health of the child, but also to prevent absence from school and interference with normal educational adjustment.³³

Exercise-induced asthma

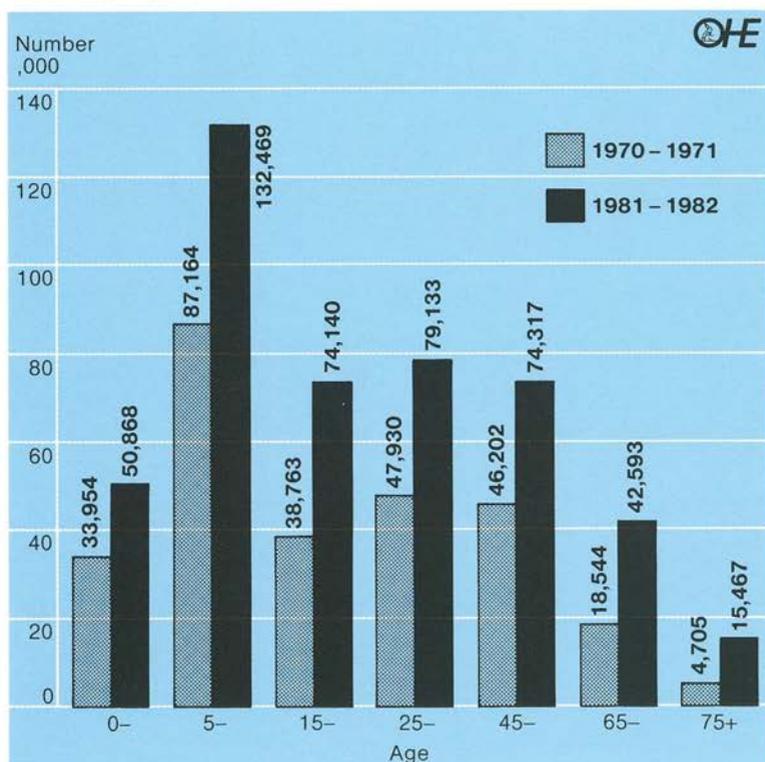
In many patients with asthma, exercise is quickly followed by wheezing and breathlessness and in a few this is the presenting symptom and most prominent feature. In such people, the term exercise-induced asthma is appropriate. However, most patients with uncontrolled asthma find that exercise induces some degree of wheezing and breathlessness with tightness of the chest and measurement of lung function in these circumstances will show increased resistance in the airways. Different types of exercise have a varying predisposition to induce an asthmatic reaction; swimming is much less likely than running to bring on an attack of asthma in those susceptible through exercise. This difference has been ascribed to loss of heat in the airways through humidification of the inspired air, especially as exercise-induced asthma is less troublesome in humid than in dry climates, but now there is some doubt as to whether this simple explanation is the sole reason for the phenomenon. In practice, people with exercise-induced asthma can be protected by inhalation of a beta-adrenoceptor stimulant (see page 24) or a cromolyn (see page 23) if it is given immediately before exercise. It has also been shown that alpha-adrenoceptor block-

ing agents can prevent bronchoconstriction in some of those who are susceptible to exercise-induced asthma.

The occurrence of asthma

Various reports have shown considerable differences in the incidence and prevalence of asthma. Estimates for children have ranged from a prevalence of 25.3 per cent in Tokelau Islanders living in New Zealand³⁴ to a virtual absence of the disease among Gambian children living in rural areas.³⁵ For adults, estimates have ranged from 0.2 per cent in Sweden³⁶ to 7.9 per cent in Tucson men.³⁷ These apparent differences may be due to different methods of study and classification but there may also be true differences between populations, for example, in their genetic susceptibility and in environmental factors. It is commonly accepted that approximately 5 per cent of the population in industrialised countries has asthma.

Figure 1 Number of males consulting (at least once) for asthma by age group, England and Wales



Source: OPCS 1974 and 1986.

England and Wales

The first National Study of Morbidity in General Practice undertaken in 1955 suggested a figure of 380,000 for the total number of people with asthma in England and Wales.³⁸ By the time of the Second National Study of Morbidity in General Practice sixteen years later, approximately 500,000 individuals in England and Wales consulted a doctor at least once for asthma during the year of the survey.³⁹ It was thought that the incidence of asthma had not increased over the intervening years³⁸ and that the apparent increase could be ascribed to difficulties in defining asthma, improvements in reporting and identification and by an increased willingness by patients to visit their doctors for treatment. However, the Third National Study of Morbidity in General Practice reported yet a further increase in consultations, suggesting that approaching 875,000 subjects in England and Wales consulted a doctor at least once for asthma in 1981–82.⁴⁰

Figure 2 Number of females consulting (at least once) for asthma by age group, England and Wales

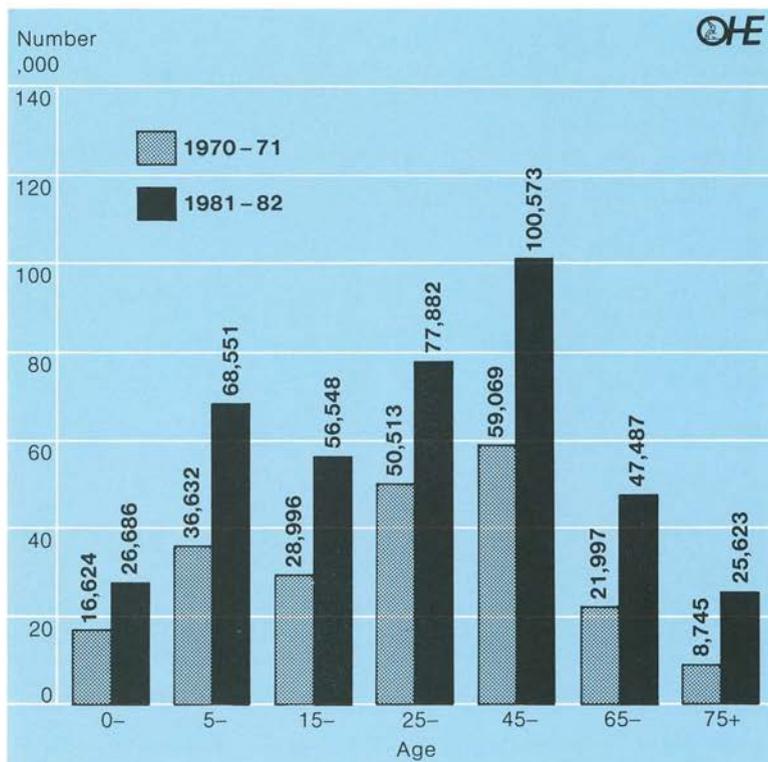
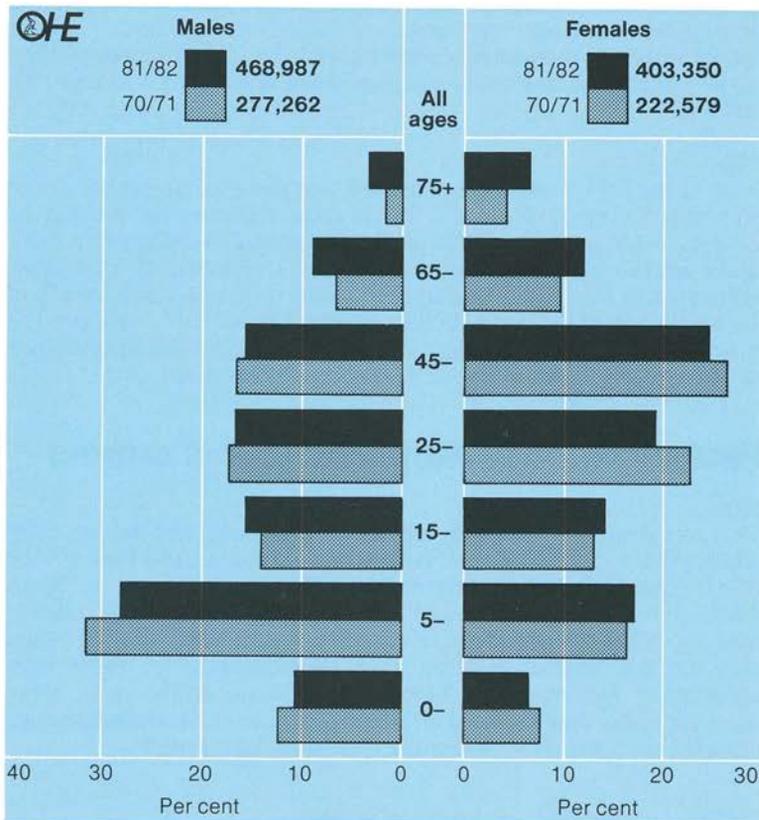


Figure 3 Number of individuals consulting (at least once) for asthma 1970/71 compared with 1981/82, England and Wales, by sex and age group



Source: OPCS 1974 and 1986.

Although the numbers of patients in each age group who consulted their doctors for asthma, increased substantially between 1970–71 and 1981–82 (Figures 1 and 2), the proportion of males and females in each age group remained fairly constant (Figure 3). Thus, comparison of the two periods studied shows that the prevalence of asthma in men increased from 11.6 to 20.5 people consulting per 1,000 population and in women from 8.8 to 15.9 per 1,000 population, to give an overall prevalence of 1.78 per cent. The prevalence increased in each of the age groups and the increase was said to be a real increase and not due to changes in diagnosis.⁴¹ However, a Public Health Service (PHS) survey in the United States of America in 1970,⁴² found a 12-month

prevalence rate of 3 per cent for asthma, so the situation in England and Wales may reflect at least, in part, an increased awareness of the condition and may still represent a degree of under reporting. Indeed, the prevalence of asthma in individual medical practices in England has been reported as 4 per cent⁴³ and more recently as 7 per cent.⁴⁴ If these figures were applied uniformly throughout the United Kingdom, they would represent a total asthmatic population of between 2.16 and 3.79 million.

USA

Even in the USA it was acknowledged that there is substantial under reporting of chronic illness and the prevalence rate for asthma may be as high as 4.3 per cent.¹² With a population of 234 million (the 1983 figure for the USA), this means that there could be 7 to 10 million asthmatics in the USA. This may well be the case, as a careful study of the population of the small Michigan town of Tecumseh, confirmed the apparently high prevalence found in the PHS survey, with a prevalence rate of 4.0 per cent in males and 3.4 per cent in females.¹²

Factors affecting the prevalence of asthma

Age

The prevalence of asthma is highest in childhood and among older adults (Table 1). This appears to be true for England and Wales,⁴⁰ the USA⁴² and Denmark.⁴⁵ About 1 in 10 schoolchildren in North Tyneside,⁴⁶ and in Middlesex,⁴⁷ are reported to have had clinical features indicative of asthma, although a report from Glasgow found only 4.4 per cent of children aged 18 months to 15 years were asthmatic.⁴⁸ However, the higher figure seems likely to be more accurate as the prevalence of asthma in children in Australia has been reported as 11 per cent¹³ and in the USA as 10.5 per cent.⁴⁹

Table 1 Age and the occurrence of asthma

Age group	Consulting rates per 1,000 patients at risk		Number of patients with asthma		Percentage of all patients with asthma	
	Male	Female	Male	Female	Male	Female
0-	33.3	18.3	50,868	26,686	10.8	6.6
5-	37.5	20.5	132,469	68,551	28.3	17.0
15-	18.6	14.7	74,140	56,548	15.8	14.8
25-	12.1	12.1	79,133	77,882	16.9	19.3
45-	13.9	18.1	74,317	100,573	15.8	24.9
65-	21.5	18.7	42,593	47,487	9.1	11.8
75+	16.2	12.9	15,467	25,623	3.3	6.4
Totals	20.0	15.9	468,987	403,350	100.0	100.0

Source: OPCS 1986.

Childhood asthma usually starts in the first few years of life, a third have their onset before two years of age and 80 per cent develop symptoms before the age of five years.⁵⁰ Most children stop having episodes of asthma as they they grow older, 70 per cent of the children in the Australian study¹³ did so. Nevertheless, 10 per cent of children continued to have symptoms at 10 years of age as earlier and in this group the onset of symptoms was usually before the age of three.¹³ After the age of 12 the prevalence of asthma drops to about 6 per cent.⁵¹

A survey carried out in South Wales showed that 6.5 per cent of people over the age of 70 had asthma, or a history of asthma.⁵² This second peak in later life has been confirmed in other surveys.^{12, 42, 45} Although some individuals have asthma from childhood to adult life, asthma can begin and remit at any age and groups of asthmatic subjects at various ages are composed of different individuals.

Sex

Boys outnumber girls among children with asthma; the sex ratios found in various studies of asthmatic children are shown in Table 2. However, this difference is lost in early adult life when asthma becomes as common in women as in men, but it returns in the elderly when asthma becomes more prevalent in men than in women (5.1 per cent compared to 1.8 per cent) and is also more severe.⁵²

Table 2 Sex ratio of children with asthma

<i>Country</i>	<i>Childhood population</i>	<i>Ratio</i>		<i>Reference</i>
		<i>Male:Female</i>		
England and Wales	Under 15 years	6:3		40
Denmark	Under 15 years	9:5		45
USA	6-11 years	3:2		51
USA	12-17 years	8:5		51
USA	Grades 1 and 6 at school	8:5		49
Australia	7-10 years	7:3		13
Switzerland	Children and adolescents	4:1		53

Race

In the USA, the PHS survey⁴² showed that for all ages asthma is less prevalent among whites than among other races. The prevalence (9.4 per cent) in black children in the USA was significantly greater than among white children (6.2 per cent) in a national survey,⁵¹ and also in the inner city of Baltimore, where the black to white ratio was 1.5:1.⁴⁹ These differences may be due, at least in part, to environmental factors because of the low prevalence of asthma in non-white developing countries.^{35, 54} This is suggested by the marked difference in prevalence among members of the same tribal group, depending on whether they lived in urban or rural areas.⁵⁵ However, asthma is uncommon among

American Indians and Eskimos.³⁶ In Britain,⁵⁶ the prevalence of asthma among Asians and blacks is higher in those children born in Britain compared with immigrants from abroad.

Heredity

Hereditary factors appear to play a part in the development of asthma. A predisposition to develop asthma is separate from a predisposition to atopy.⁵⁰ Studies in twins confirm that the propensity to asthma is under genetic control, although, since concordance of symptoms even in identical twins is incomplete, environmental and perhaps other factors must also play a role in the development of the illness.⁵⁰

Occupational factors

The occupation of adults may influence the prevalence of asthma in this age group. In one American study,⁵⁷ of more than 6,000 adults, the prevalence of asthma was 7.7 per cent and nearly one in six attributed their illness to exposure to environmental factors at their place of work. Thus, occupational asthma contributed 1.2 per cent to the total prevalence of asthma in these adults. The relative risk for occupational asthma was highest among industrial and agricultural workers. Environmental exposure to the dust of grain²⁸ and soya bean^{26, 27} has been shown to cause a reduction in lung function and epidemics of attacks of asthma.

Mortality

At the turn of the century it was standard teaching that asthma was never the cause of death⁵⁸ and yet mortality from asthma has been recognised from the start of the twentieth century⁵⁹ and deaths from asthma have occurred consistently in the UK, at approximately 1,500 per annum for the past 35 years, that is, apart from shorter periods of a transient increase such as occurred at the beginning of the 1960s.⁶⁰

The reasons for patients dying suddenly during an attack of asthma has been variously attributed to the improper use of inhalers^{61, 62} or to failure by the physician to appreciate the severity of an attack from the symptoms and perhaps to delay giving appropriate treatment.^{60, 63, 64} Overuse of bronchodilators has never been proved as the cause of death but the arguments about deaths of patients with asthma being linked to the use, or misuse, of their bronchodilator inhalers, continues with a recent report from New Zealand about one particular type of metered dose aerosol.⁶⁵ What is certain is that asthma consists of much more than just wheezing;⁶⁶ it is a serious illness and one that is potentially fatal. Most patients dying in asthma, die of asthma⁶³ although there may be the occasional misattribution of death.⁶⁷ It is distressing that avoidable factors have been identified in the events leading up to 82 per cent of deaths in the United Kingdom,⁶⁸ although this is not to say that the death could have been prevented. Examining the pattern of mortality might identify factors that will help to detect those who are at greatest risk of dying and help to close the gap between diagnostic and therapeutic knowledge and its general application.⁶⁰

Figure 4 Deaths from asthma: year of death, males, England and Wales 1951–85. Rate per million population.

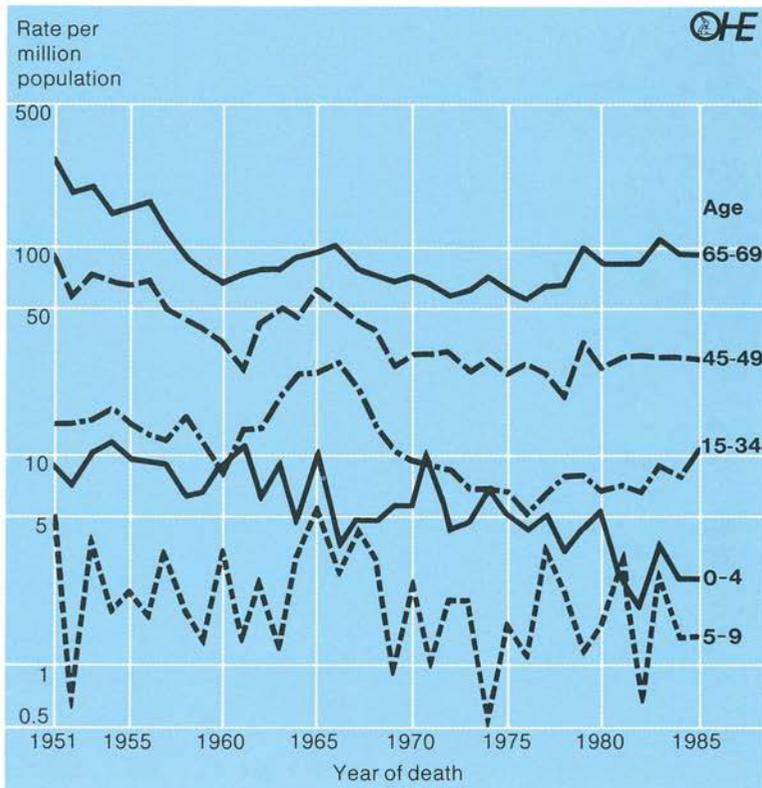


Source: The Annual Report of the Chief Medical Officer of the DHSS, 1986.

United Kingdom

The annual death rate from asthma per million population in England and Wales between 1951 and 1985 is shown in Figure 4 (males) and Figure 5 (females). Apart from the characteristic increase in deaths of the under 35s in the early to mid-1960s, the figures do not show any recent increase in the death rate, except in boys below the age of five, or any overall consistent trend.⁵⁹ Nevertheless, Khot and Burn⁶⁹ defined that there was a small upward drift in the death rate from asthma in England and Wales in the early 1980s, which was not significant; the most striking feature of the findings was a seasonal (summer) increase in mortality at all ages below 35 years; this was most pronounced in the 5 to 14-year-old age group. However, Burney⁷⁰ reported a consistent annual increase in mortality from asthma of 4.7 per cent in those below

Figure 5 Deaths from asthma: year of death, females, England and Wales 1951–85. Rate per million population.



Source: The Annual Report of the Chief Medical Officer of the DHSS, 1986.

35 years of age, over the ten-year period, 1975–1984, despite account being taken of changes in the coding of disease. This increase was said to be consistent and statistically significant and did not represent a change in diagnostic fashion or practice.⁷¹ The increase occurred principally among males in the age group 5 to 34 years (see Table 3). It has been pointed out⁷² that the death rate from asthma in females in the 15 to 44 year age group, has fallen from 15–20 deaths per million in the 1950s, to about 10 per million in the early 1980s, but in males of the same age group the death rate has hardly changed. This difference between the sexes in England and Wales remains unexplained and is not matched in Scotland where there is little difference in mortality from asthma between the sexes below the age of 45 years.⁷³

Table 3 Death rates/million and standardised mortality ratios (SMRs) for asthma in England and Wales by age group, sex, ICD classification, and year of death

Year*	Male—age in years			Female—age in years		
	1-4	5-14	35-64	1-4	5-35	35-64
Death rates/million						
1974	4.6	5.3	22.1	8.4	5.2	30.3
1975	4.2	7.0	26.3	6.6	4.8	30.3
1976	5.9	5.8	19.2	5.4	4.4	30.7
1977	5.5	6.2	20.1	5.8	5.9	28.6
1978	6.6	6.8	21.6	4.3	6.6	30.5
1979	1.7	6.8	28.0	6.2	6.7	33.7
1980	4.2	8.0	29.8	6.2	5.5	33.6
1981	2.5	10.9	32.9	3.5	7.1	41.2
1982	3.2	11.0	27.7	2.5	5.4	38.5
1983	2.3	10.3	33.2	4.9	7.7	35.1
1984	3.9	10.5	33.4	3.3	6.6	34.0
SMRs						
1974	100	100	100	100	100	100
1975	90	130	118	78	92	100
1976	127	107	86	65	85	101
1977	118	113	91	69	111	94
1978	142	123	98	52	123	100
1979	36	122	128	74	124	112
1980	91	143	136	74	101	112
1981	53	193	150	41	128	138
1982	69	194	126	30	97	129
1983	51	183	152	59	136	118
1984	84	186	152	39	116	115

*ICD 8 used for 1974-78, and ICD 9 for 1979-84

Source: *Lancet* 1986; 2: 323-6.

In Scotland, the death rate from asthma in those under 45 years of age fell over the period 1966 to 1975, from about 17-18 per million, to about 6 per million; an apparent increase to about 7 per million in the late 1970s and early 1980s is based upon too small figures to be meaningful. One factor identified in Scotland was the higher mortality rate in rural areas compared with urban areas.⁷³

In the United Kingdom, deaths from asthma are relatively rare; in England and Wales in 1985 there were 1,972 deaths from this disease and most were in the older age groups. Deaths from asthma account for 30 per cent of all deaths from respiratory diseases. Nevertheless, the British Thoracic Association's confidential enquiry into deaths from asthma in adults between the ages of 15 and 64 found that there were

preventable factors contributing to the death in 8 out of 10 cases.^{74, 75} Mortality from all causes, but especially from all respiratory diseases, is greater in patients with asthma than in non-asthmatic subjects.⁷⁶

France

France did not experience the 'epidemic' of deaths from asthma reported from several other countries. From 1968 and 1974, the death rate from asthma in females decreased, and it also decreased in males between 1968 and 1980. In 1984, there were 1,799 deaths from asthma or allergic alveolitis.⁷⁷ The death rate from asthma increased in France in the 5 to 34 year age group but, unlike the United Kingdom, this increase occurred in both sexes to a similar extent:

males : 1974 - 1.47/million; 1984 - 3.52/million

females: 1974 - 1.99/million; 1984 - 3.95/million

In other age groups (0-4 and over 35 years), there has been no consistent change in mortality from asthma between 1974 and 1984.

Scandinavia

Denmark

There was a progressive increase in the overall death rate from asthma from 12 per million in 1972 to 32 per million in 1982.⁷⁸

Sweden

The death rate from asthma rose from about 46 per million in 1974 to a peak of 71 per million in 1980 and fell to 57 per million by 1982.⁷⁸

USA

Asthma was reported as the underlying cause of 1,674 deaths in 1977, 1,872 deaths in 1978, 2,598 deaths in 1979, 3,054 deaths in 1981, 3,564 deaths in 1984, and about 3,800 deaths in 1985.^{78, 79, 80} The apparent sudden increase between 1978 and 1979 can be accounted for by the adoption of the Ninth Revision of the International Classification of Diseases (ICD). But between 1979 and 1985 there was a real increase in deaths from asthma of 46 per cent. Rates of death from asthma reached a low of 8 per million population in 1977 and 1978 but had doubled to 16 per million by 1985, part of this increase being accounted for by the change in ICD coding of 1979.⁷⁹ The death rate from asthma has increased in all age groups, except the under 5s, and in both sexes.⁷⁹ The increase in the death rate from asthma between 1980 and 1985 was 23 per cent.⁸⁰ The death rate is highest in the black population (25 per million in 1984) and lowest in the Indians (8 per million in 1980). The increase in the death rate between 1979 and 1984 was greater in black than in white subjects, 39 per cent versus 27 per cent.⁷⁹ Increases in deaths have not been localised to any region.⁷⁹

Canada

Deaths from asthma increased from 324 in 1974 to 513 in 1984, an increase of 58 per cent; the change in ICD classification would have

accounted for the 31 per cent increase which occurred between 1978 and 1979. Deaths from asthma as a percentage of total deaths increased by 50 per cent, from 0.194 per cent to 0.292 per cent, between 1974 and 1984. The increase in the death rate was most marked in the 15 to 34 year age group, in both sexes.^{81, 82}

Australia

After the 'epidemic' of asthma deaths in the mid-1960s, the death rate from asthma in Australia declined to an annual rate of about 0.9 per 100,000 throughout the 1970s.⁸³

New Zealand

National asthma mortality statistics for New Zealand showed that the incidence of deaths from asthma started to rise in 1965, rather later than in other countries which experienced the 'epidemic' of asthma deaths, and rose to about three times the pre-epidemic level in the late 1960s before declining.⁸³ However, in 1976 the mortality rate began to increase again and reached levels greater than those of the late 1960s, 6.6 to 7.2 per 100,000 in the early 1980s.⁸⁴ Demographic analysis showed markedly different death rates between the three racial groups: 18.9 per 100,000 in Maoris; 9.4 per 100,000 in Polynesians; 3.4 per 100,000 in Caucasians.⁸⁵ As was the case elsewhere, mortality rates increased more markedly in the under 35s, especially in the 15- to 24-year-olds in the years between 1977 and 1982, but they have declined since.

Possible explanations

There have been many attempts during the past 20 years to explain the variable mortality rates from asthma. Read⁶³ and Gandevia,⁶⁴ suggested that spontaneous changes in the natural history of asthma accounted for the raised level of mortality during the 'epidemic' of the 1960s. This was the only explanation that could be proffered when the authors failed to find evidence in Australia that confirmed earlier speculation, from investigations in England and Wales, into the use of drugs preceding death from asthma, that new treatments, specifically bronchodilator aerosols, might have contributed to the increase in mortality.^{61, 62} This has not prevented subsequent speculation on the possible links between treatment and death from asthma with oral sustained-release theophylline preparations,⁸⁶ bronchodilator solutions administered with electrical air compressor pumps,⁸⁷ or a specific bronchodilator claimed to be long-acting,⁶⁵ for none of which has a relationship been reliably shown with clinical or epidemiological evidence. Data on the sales of remedies for the treatment of asthma could not be linked temporally with mortality from asthma in the three countries which experienced the most marked increase in the incidence of deaths from asthma.⁸⁸ Thus, it is unlikely that modern remedies are directly associated with the deaths of asthmatic patients.

In fact, a confidential enquiry into the deaths from asthma of adults under 65 years old,⁷⁴ showed that no single contributory factor could be identified as responsible for the deaths of the 90 patients who were investigated. The interaction of several adverse factors tended to contribute to patients' deaths. Those who died generally suffered from chronic asthma and had a medical history of intermittent severe attacks. Bronchodilators and corticosteroids were generally under-prescribed, or underused, in that they were given in insufficient doses. Indeed, preventive measures (inhaled corticosteroids and cromoglycate) had often not been tried. Supervision of the patients and compliance with treatment were unsatisfactory in about half the patients and about half did not receive medical attention during the period immediately preceding their fatal attack. Failure to recognise the severity of the asthma by patients, their relatives, and their doctors, often delayed the start of appropriate treatment.

Of the 90 deaths, 77 (86 per cent) occurred at home or at work. In Scotland, too, the majority of deaths from asthma, 72 per cent in patients below 45 years of age, occur at home.⁷³ This reflects both the suddenness with which deterioration can occur and the failure to recognise the severity of an acute attack.

Death rates from asthma vary more than six-fold among the 14 countries which have suitable statistics for making comparisons.⁸⁹ However, a gradual increase in mortality seems to have occurred since the middle to late 1970s among 5 to 35-year-olds in these 14 countries. This increase is not able to be explained wholly by changes in diagnostic convention, but it could reflect an increase in the incidence and severity of the disease.

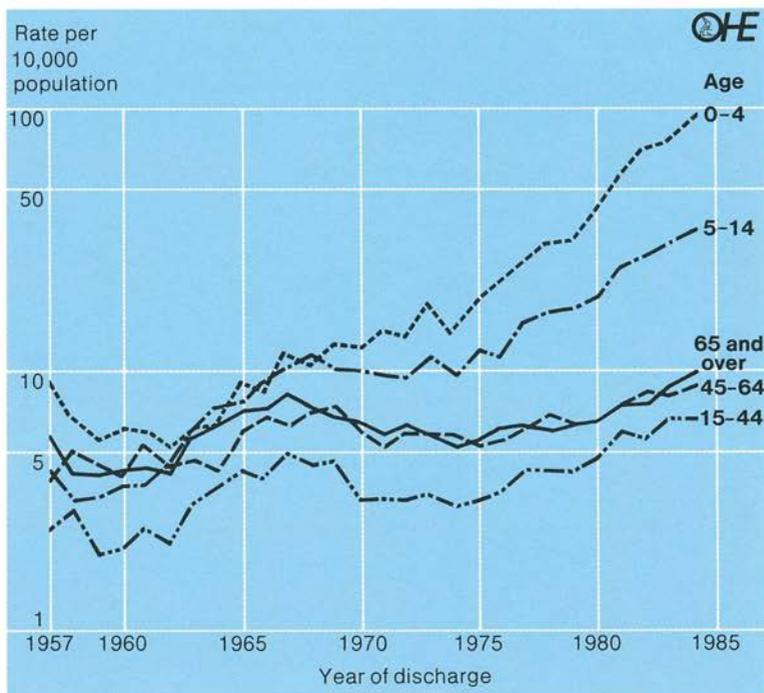
Morbidity

In Denmark, it has been shown⁴⁵ that, in general practice, the mean consultation rate for patients with asthma was 5.7 times a year, with variations between 7.9 (highest) and 2.8 (lowest) per patient per year. This is somewhat higher than the mean consultation rates, 2.62 per man and 2.82 per woman with asthma, for England and Wales in the early 1980s.⁴⁰ In the USA, 60 per cent of all patients with asthma make one, or more, visits per year to their physician to consult for their asthma.⁴²

Thus, with a mean general practitioner consulting rate approaching 3 per year and an average general practice having up to 140 asthmatic patients,⁴⁴ asthma represents a considerable part of a practice's work. However, it does not reflect the distress suffered by the patients.

It has been reported from the USA⁴² that nearly one-third of all patients with asthma are confined to bed with their condition for some period in each year, and also about one-third are bothered a great deal by symptoms frequently or all the time. In American children, this means that about half miss six days, or more, of schooling per year and about one in ten are admitted to hospital for up to ten days in every year because of their asthma.⁴⁹ In England, absence from school

Figure 6 Hospital discharges from asthma: year of discharge; males 1957-81, England and Wales, 1982-84 England



Source: The Annual Report of The Chief Medical Officer of the DHSS, 1986.

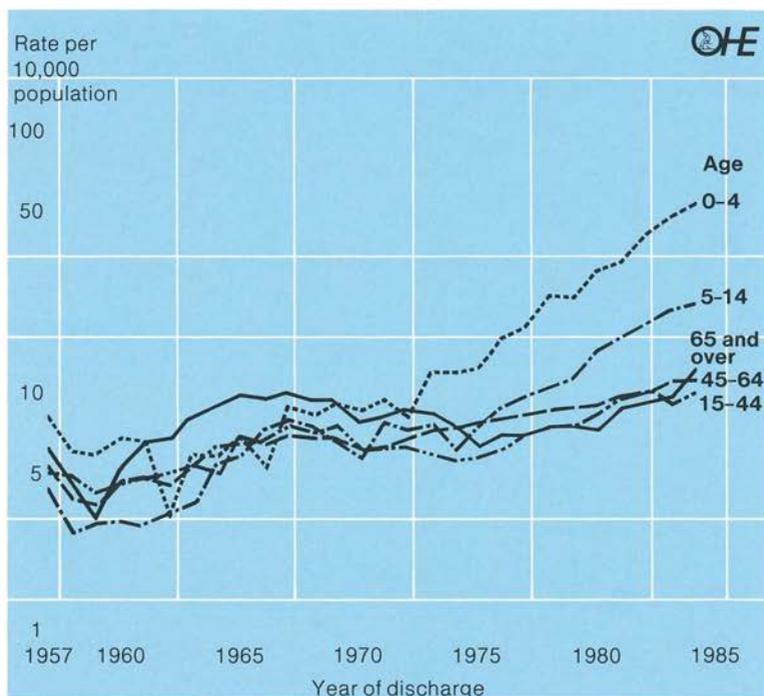
because of asthma has been reported in 58 per cent of asthmatic children with 12 per cent of them being away from school for more than 30 days.³³

There has also been an increasing rate of admission to hospital for asthma.²⁰ Hospital discharges for asthma over the years, 1957 to 1984, for five age groups, are shown in Figure 6 (males) and Figure 7 (females). The increase is particularly noticeable in the younger age groups, especially since 1970. There is some evidence that this is due, at least in part, to an increase in the re-admission rate and of self-referrals.⁵⁹

As in the USA, about one-third of asthmatic children are confined to bed with their asthma for up to 10 days.³³ About a quarter of asthmatic children have some limitation to their participation in physical education and games at school.³³

Home visits by general practitioners are made to 10 to 15 per cent of their asthmatic patients in a six month period.⁹⁰ More than one-third of asthmatic patients have two, or more, severe attacks of asthma in a six

Figure 7 Hospital discharges from asthma: year of discharge; females 1957-81, England and Wales, 1982-84 England



Source: The Annual Report of The Chief Medical Officer of the DHSS, 1986.

month period; over a third have an attack which lasts for at least a day;⁹⁰ and a third of those with persistent asthma have substantial time off work.⁹¹ In a survey⁹² of adults with asthma, measurement of their breathing capacity was normal in under half of the patients and morbidity from their asthma, including restrictions in life-style, was correlated with these measurements.

Nocturnal asthma, waking during the night with tightness of the chest and wheezing or coughing, is an especially troublesome feature of asthma which occurs at least once a week in nearly three out of four asthmatic patients, and every night in two out of five:⁹³ nocturnal asthma is often an indication of the severity of their asthma.

It is not really surprising that patients with chronic asthma have a higher than expected level of anxiety and restrictions on their life-style.⁹⁴ Chronic suffering from wheezing and breathlessness with its attendant restrictions on a normal life can become accepted as normal by the patient with asthma and contribute to the morbidity of the condition.

The clinical management of asthma

Asthma is a variable condition; not only does it differ between patients, but also in the same patient at different times. The degree of reversibility of the obstruction in the airways differs considerably between patients. Where irreversible obstruction has developed, there is a consequent reduction in reserve capacity in the lungs which leads to the development of severe symptoms from only a slight additional narrowing of the airways. It is not possible, therefore, to be specific about the management of asthma, only to generalise about the various medicines which can be used and to put these into some kind of context.

Allergy and Immunotherapy

In a few cases, where the asthma has been shown to be caused by exposure to an allergen such as animal fur or dander, removal of that allergen from the environment can relieve patients of their symptoms. However, only a limited number of patients can benefit from such management. Likewise, specific immunotherapy, the injection of allergens to which the subject reacts, is of limited value and can be hazardous. It is not a suitable treatment for general use. It may have some value in those few patients with seasonal allergic asthma due to pollens and also some young patients whose asthma is due to such perennial allergens as the house dust mite, although simple hygienic measures, such as regular vacuum cleaning, the use of synthetic bedding materials and impervious mattress covering can be helpful.

Anti-allergic medicines

The cromolyns, disodium cromoglycate (DSCG) and nedocromil sodium, were formerly thought to act by preventing the mast cells in the lungs from releasing spasmogens and inflammatory agents which started attacks of bronchospasm. It is now known that their effects are rather more complicated than this simple picture suggests. However, they do tend to prevent asthmatic attacks occurring in some patients, especially young atopic asthmatics, when they are used regularly and their use can lead to a reduction in other types of medication.

Ketotifen and azelastine are antihistamines with other 'anti-allergic' properties. Ketotifen appears to act on the mast cells* in the lungs and it may also act on other systems involved in allergic reactions such as the blood platelets. Neither compound is widely used in the clinical management of asthma, although they might prove to be useful as adjuvant treatment in some patients, especially those with a demonstrable allergic component.

*Mast cells are cells which contain granules in their cytoplasm and which are widely distributed in the connective tissues, including the respiratory tract. Large numbers of these cells are found in the walls of the airways and alveoli. Mast cells release chemical mediators, which cause bronchoconstriction and inflammation, in response to inhaled stimuli such as allergens.

Bronchodilators

Bronchodilators are substances which open up the airways by causing the smooth muscle which lines them to relax, reversing the constriction and bronchospasm, two of the functional changes in asthma which were described earlier. Bronchodilators fall into two distinct groups, known as beta-adrenoceptor stimulants and xanthines. Both groups of compounds have essentially similar effects on the airways but they act by different mechanisms and have different patterns of untoward effects.

Beta-adrenoceptor stimulants

These compounds are the mainstay of treatment in patients with asthma. They are available in various formulations (metered-dose aerosols, dry powder inhalers, solutions for nebulization, tablets and liquids for oral administration, injections) designed to achieve distinct effects in differing circumstances. The familiar, small, pressurised aerosol is used to relieve quickly acute attacks of wheezing and breathlessness that are the primary feature of asthma. To achieve the optimum results, it is necessary to co-ordinate actuation of the inhaler with maximum inspiration. For those patients who find this difficult, breath-actuated inhalers and spacer devices are available to allow them to get maximum benefit from this type of treatment. The advantages of inhalational treatment are two-fold: a rapid response and the use of minimum doses, both of which derive from delivery of the active compound directly to the airways where it is needed.

In chronic asthma, regular medication can improve control of the symptoms throughout the day and this can be achieved by inhalation or oral administration. The sustained-release oral preparations are particularly useful at night to avoid disturbance of sleep from night-time attacks of breathlessness and to inhibit the characteristic 'morning dipping' (reduction of inspiratory flow rate on waking) that is commonly a feature of unstable asthma. Solutions for nebulization, preferably administered with oxygen, and injections are used to control severe attacks of asthma which require admission to hospital.

Xanthines

The xanthines, theophylline for oral administration and aminophylline for injection, also cause bronchodilatation by affecting the smooth muscle lining the airways but, unlike the beta-adrenoceptor stimulants, their mechanism of action is probably indirect. Oral xanthines are useful for regular administration in preventing attacks of asthma, although they are less well tolerated than other bronchodilators. Sustained-release formulations for preventing nocturnal asthma and early morning wheeze are probably the most familiar forms of this group of compounds. Aminophylline is used by direct injection into a vein in many patients admitted to hospital with severe, perhaps life-threatening, attacks of asthma, especially those who have not already been taking a xanthine compound by mouth. Xanthines are not considered to be the treatment of choice in childhood asthma because of concern about their apparent effects on sleep, concentration and school performance.

Corticosteroids

It has long been known that corticosteroids were very effective in the management of chronic asthma but their regular use was formerly restricted to those most severely affected by the disease because these compounds needed to be given daily by mouth for long periods, with the attendant risks of side-effects that accompanies this type of treatment. With the development of corticosteroid derivatives in which the local effect on the tissues of the lungs was dissociated from the systemic side-effects, it became possible to administer these valuable compounds regularly by inhalation, so largely avoiding the unwanted effects of oral administration.

Corticosteroids act differently from the bronchodilators. They do not relieve the symptoms of acute attacks of wheezing and breathlessness. They have to be administered regularly to achieve their effect of alleviating inflammation in, and reducing the hyperreactive state of, the airways. Indeed, it is becoming increasingly recognised that the regular use of inhaled prophylactic agents such as the corticosteroids reduces morbidity from asthma, improves the quality of life of sufferers from the disease and can lead to lower usage of bronchodilators and fewer admissions to hospital for the treatment of severe attacks.^{44, 95}

Corticosteroids may occasionally need to be given by mouth to some asthmatic patients, preferably in short courses, to overcome particularly severe attacks, especially after admission to hospital where these compounds have already been given by injection.

Possible future developments

Activation of platelets, a formed element in the blood, may be an important mechanism in asthma. A substance termed platelet activating factor (PAF) is released in the airways as part of the inflammatory process in asthma. This substance causes immediate bronchoconstriction in the airways and also increases their hyperreactivity, a property not possessed by other inflammatory agents.⁹⁶ Research is being conducted on the effects of various compounds with activity against PAF and it is possible that these studies will lead to the evolution of a new and distinctive type of medicine for use in the management of patients with asthma.⁹⁷ Other new approaches include the development of anti-inflammatory compounds based on the pharmacological role of leukotriene in producing allergic and inflammatory reactions and of bronchodilators from the naturally occurring smooth muscle relaxant, atrial natriuretic peptide.

Other likely developments are new beta-adrenoceptor stimulants and corticosteroids with improved efficacy and tolerance.

While these developments are eagerly awaited, there is still much room for improvement in the diagnosis of asthma and its effective treatment with existing medicines. There is evidence that preventive treatment remains under-prescribed^{92, 98} and that the under-supervision and under-treatment of patients with asthma are common and not confined to those dying from the condition.⁹⁹ Asthma remains

a challenge to the doctor and there appears to be ample scope for improving the management of this very common disease.¹⁰⁰ This has been recognised with the development of international guidelines for the clinical management of children with asthma.¹⁰¹ These recommendations stress the importance of a full clinical history with tests of respiratory function and allergy being used to support a clinical diagnosis and to guide treatment. If treatment is properly prescribed and used and full use is made of other supportive measures, there is no reason why asthmatic children should not have a normal life-style, comparable to their more fortunate friends.

Quality of life

Disturbed sleep, through waking at night with symptoms of asthma, is one of the features which distinguishes the impaired quality of life of the asthmatic subject from the non-sufferer. This is of particular importance in children whose development can suffer from a prolonged disturbance of the normal rhythm of life. An objective measurement of the quantity and quality of sleep, by monitoring the nocturnal electroencephalogram (EEG), has shown that the administration of a long-acting bronchodilator before bedtime improves the quality of sleep without inducing any untoward effects.¹⁰² Furthermore, an inadequate understanding of asthma and its management in childhood leads to restriction of physical and sporting activities later in life, as well as to inadequate treatment and substantial absences from work.⁹¹ The restriction of physical activity may be measured objectively by the straightforward six-minute walking test which has been shown to relate to the quality of life of the patient with asthma better than any other single measurement of activity or lung function.¹⁰³

It is pertinent to mention that although the better management of the patient with asthma may require the use of modern remedies, rather than cheaper medicines of an earlier era, which superficially seem expensive, their use can actually reduce the overall medical costs. In one study,¹⁰⁴ the annual cost of bronchodilator inhalers increased by \$12.81 (£8.10) when isoprenaline was superseded by salbutamol, but the resultant annual saving in other asthma-related medical costs was more than forty times the relatively modest increase in the cost of medicines arising from the change to the more beneficial remedy. It must not be forgotten that there are also hidden, or indirect, costs associated with asthma, such as time lost from work or school, travel to surgery or hospital, miscellaneous purchases, etc. In the USA, it has been calculated that the total costs of managing a child with asthma account for 6.4 per cent of the average family's income.¹⁰⁵ The indirect costs are a relatively greater burden to those families with below average income and they have an impact on the quality of life not only of the patient, but also of the entire family.

Table 4 Cost of asthma to the National Health Service, UK, 1987

	<i>Asthma</i> £ million	<i>Total</i> £ million	% of Total
Hospital services	52.84	12,403	0.43
General medical services	17.62	1,634	1.08
Pharmaceutical services	29.16	2,281	1.28
Dental and ophthalmic services	—	1,086	—
Total	99.62	17,404	0.57

Notes

The hospital services figure includes capital costs.

The cost of pharmaceutical services is calculated using the average cost for all prescriptions.

Source: Office of Health Economics estimates.

The cost of asthma

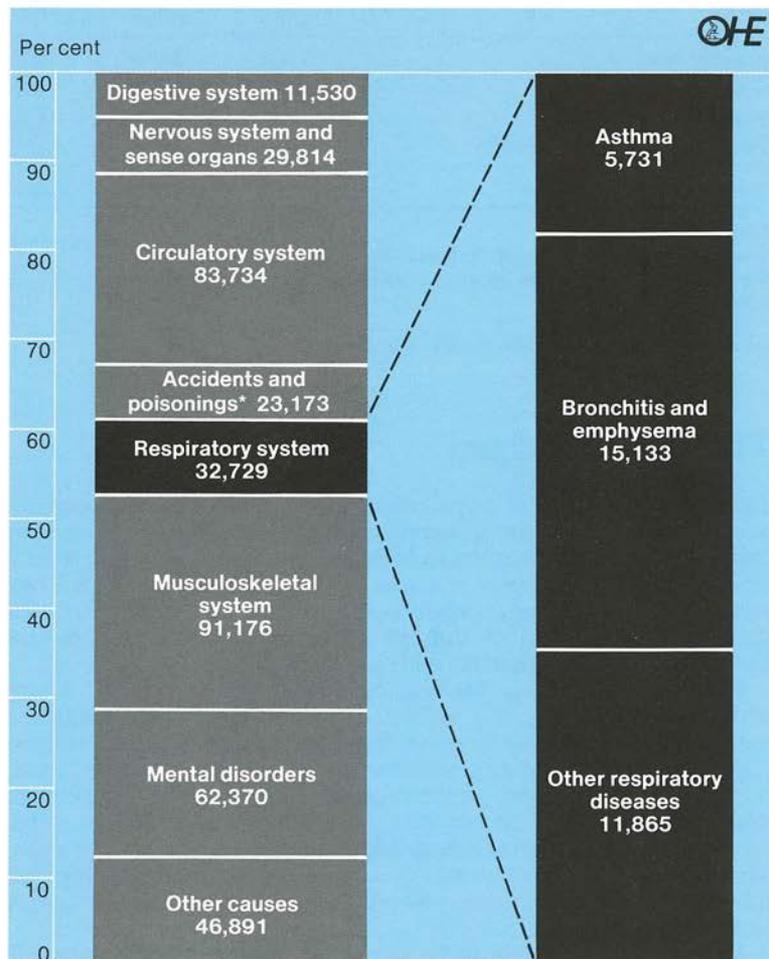
The financial burden of any disease can only be calculated approximately and such estimates should not be interpreted as if possessing importance in absolute terms. Rather, their value is in providing a measure of comparison between diseases. The costs can be divided into three main groups: the expenditure on medical and welfare services, the value of production lost through sickness absence, and the personal hardships suffered by patients and their families.

The cost of asthma to the National Health Service in 1987 is estimated to be around £100 million, of which more than half (£52.8 million) is accounted for by expenditure on hospital inpatient services (Table 4). The latter figure is probably an underestimate as a large proportion of asthmatics are admitted as emergencies to the more expensive acute hospitals. The hospital sector cost figure would be raised further by the inclusion of outpatient treatment but the relevant statistics are not available. Pharmaceutical services account for about one-third of the total medical cost, reflecting the importance of drug therapy in asthma.

Another economic cost to be taken into account is the amount of sickness absence resulting from a particular illness. In 1987/88 asthma accounted for 5.7 million days of certified absence (Figure 8) which represented 1.6 per cent of all recorded lost working days in Britain, thereby incurring an expenditure of just over £15 million in sickness benefit grants.

The value of production which is lost through sickness absence can be derived crudely from estimates of earnings foregone and on this basis asthma cost approximately £223 million in 1987. This estimate is based on average weekly earnings (1987) for full-time adult workers in manufacturing and certain other industries of £194.90 for men and women. Alternatively, if the cost of certified incapacity is expressed in

Figure 8 Days of certified incapacity for the period 1987/88, by cause, Great Britain, (thousands)



Note: *Accidents, poisonings and violence.

Source: DSS.

terms of foregone Gross National Product a rough estimate of £260 million may be obtained. It is, of course, extremely difficult to evaluate accurately the financial significance of sickness absence when so many factors, particularly the level and adaptability of economic activity and the effects of inflation, have to be taken into account.

The personal costs of distress and lost potential vary considerably between individuals. However, it is possible to calculate the loss of earnings due to the premature death of asthmatics, on the assumption that otherwise they would have had a normal life span. In England and Wales, there were 1,898 deaths from asthma in 1987 and 764 of these (40 per cent) occurred before the normal age of retirement. Deaths in males outnumbered those in females by a ratio of 1.5 to 1. When adjusted for age and sex, these deaths represented a total loss of earnings of £508 million for England and Wales, or £564 million for the United Kingdom.

One final aspect of the cost of the complaint is the amount spent on research, although it is not always possible to identify specific expenditures, partly because advances may accrue from investigations in other areas. Thus the Medical Research Council does not provide a breakdown of the funds allocated to asthma research but of the £133 million spent in 1987/88, approximately £8.3 million was accounted for by the unit investigating respiratory diseases. About 8 per cent (or £53 million) of the British pharmaceutical industry's research budget (estimated to be £668 million in 1987) was devoted to asthma and allergy. This is an area which is currently attracting considerable research interest not only in this country, but also throughout the world, thereby enhancing the likelihood of therapeutic advance. The main clearly identifiable source of finance is the Asthma Research Council. In 1987/88, grants totalling more than £788,000 were allocated to a wide variety of projects.

Social aspects

Emotional disturbance has long been regarded as having a bearing on attacks of asthma but it is difficult to separate emotional from purely physical factors in a complex condition such as asthma. The obvious distress of a child suffering from wheezing and breathlessness will provoke anxiety in the family which will be communicated to that child. Furthermore, in an illness which starts as an allergic reaction to physical stimuli emotional factors may later come to play a significant, if not dominant, role. Even when anxiety has not been responsible for starting an attack, once the asthma begins fear may tend to perpetuate it and create a vicious circle from which it is difficult to break out.¹⁰⁶ For example, parental anxiety can lead to excessive and unnecessary restrictions being placed on asthmatic children which may induce a feeling of isolation, reduce normal social relationships and condition them to regarding themselves as 'sick' people.

It is not surprising that, in such circumstances, personal and family relationships influence the course of the illness and create diverse

social problems which are difficult to evaluate and quantify. In asthma, as in many other illnesses, a holistic approach to the management of the patient is essential and the family and social aspects have to be elicited as part of the history taking, and considered in conjunction with the medical management of the condition.

Conclusion

Asthma is a common disability with all general practitioners having very many asthmatic patients of all ages in their practices. The evidence suggests an increasing prevalence of the condition which causes considerable morbidity with much time lost from schooling and, in adults, productive capacity and earnings. This increasing prevalence has not been accompanied by an increase in the mortality rate in the United Kingdom. However, the annual number of deaths remains disappointingly high in view of the advances made in medical treatment and the avoidable factors present in a high proportion of the deaths which have been investigated. There remains a considerable job to do in educating patients about their illness, in the regular monitoring of their respiratory function and their proper use of medicines, in increasing the preventive treatment of their illness, and in boosting the confidence of patients and their families about the ability of asthmatic subjects to lead normal active and social lives. With the medicines and facilities for monitoring respiratory function that are now available, there is no need for patients to accept as normal a chronic condition of ill health. Improved education about the long-term control of their asthma and a better understanding of the illness and its treatment should avoid the problem reportedly found in 9 out of 10 patients of failure to use preventive treatment by regular inhalation because of the lack of immediate relief of their symptoms.¹⁰⁷

The better management of asthma may, on superficial examination, appear to require increased expenditure on medicines, especially preventive treatment, and on patient education but the use of improved treatments and successful preventive measures can actually bring about a reduction in the total burden on health expenditure and a positive contribution to the productive capacity of the nation. There may already be some evidence of this in the reduced proportion of total NHS expenditure accounted for by asthma between 1976 and 1987. But with the annual number of deaths in the United Kingdom persisting at about 2,000, many of which occur tragically early in life, 40 per cent before retirement age, there is no room for complacency.

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