MEDICAL PRACTICE

Process and Outcome

Asthma mortality in Birmingham 1975-7: 53 deaths

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Summary and conclusions

Out of 83 patients studied 72 were certified as dying from asthma, and 11 aged under 45 as dying from chronic bronchitis and pneumonia. Fifty-three deaths were thought to be due to asthma. There were avoidable factors associated with several of these deaths from asthma. Recent discharge from hospital (16%), non-availability of aerosol bronchodilators (45%), underuse of corticosteroids (66%), and lack of objective measurements of airflow obstruction (100%) were found in deaths outside hospital. Inadequate initial assessment including baseline spirometry and blood gases (50%), significant underusage of corticosteroids (93%) and intravenous and nebulised bronchodilators (100%), and failure to monitor treatment objectively (100%) were found in deaths in hospital. "False-positive" and "false-negative" certifications of asthma were studied, and the findings suggest that these may lead to appreciable inaccuracy in the reporting of deaths from asthma.

Introduction

Surveys of asthma mortality in which the lack of awareness of the severity of attacks[1] was pointed out have been conducted for over 25 years. A rise in the number of deaths due to asthma in the mid-sixties, especially in young people, focused attention on the possible role of non-specific beta-agonist aerosols.[2-5] This factor, however, was declining in importance before the introduction of more specific beta-agonists.

Deficiencies in case management for both outpatients[6] and inpatients[7 8] have been outlined more recently. These authors found that underuse of corticosteroids, inadequate assessment, and failure to use objective measurements of severity were major factors contributing to death. The present survey was undertaken to see what effect these findings had had on managing asthma and its mortality in our own area.

The recognition of asthma in life and the appreciation of its severity by both patients and doctors remains a problem. This is reflected after death in both over- and under-certification of the condition, which may lead to a false impression of the frequency of death from asthma. We present an analysis of cases that in our opinion were wrongly certified, adding some brief case studies that highlight a matter currently under debate. This provides further evidence of the weakness of our present methods of certification, already pointed out by the Medical Services Study Group of the Royal College of Physicians.[9]

Methods

Copies of all death certificates in which asthma was mentioned were obtained for 72 deaths occurring within the metropolitan area of Birmingham for the three years 1975-7 from an approximate population of one million. Death certificates giving the cause of death as chronic bronchitis or bronchopneumonia in 11 patients under the age of 45 years were also considered, because we thought that this group probably included unsuspected asthmatics. This age was chosen arbitrarily because it was not possible to survey the much larger numbers who were over 45.

The NHS records, except for seven patients which could not be traced, were obtained from the local family practitioner committee. Thirty-one patients had had coroner's necropsies, and access to the pathologists' reports was obtained. Twenty-two patients died in hospital, and these notes were traced in all but one patient.

Patients were included in the study only if there was (1) necropsy evidence of asthma with overinflated lungs that failed to deflate on

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opening the chest and extensive bronchial mucus plugging,[10 11] (2) a convincing clinical history of asthma, or (3) evidence of significant reversibility of airways obstruction as shown by objective measurement or prompt response to treatment with bronchodilators. We excluded from further study patients not fulfilling these criteria, those in whom the stated asthma was not, in our opinion, a contributory cause of death, and suspected asthmatics for whom there was insufficient information to form a judgment.

In categorising our patients we were aware of the difficulties of interpreting "a convincing clinical history," as asthma and other causes of chronic airways obstruction are not mutually exclusive. Thus our impressions were biased in favour of asthma and away from chronic bronchitis by the onset of symptoms of an episodic nature at a young age, by the absence of a history of smoking, and by the presence of atopy and associated disorders. Conversely, we were biased against asthma in favour of chronic bronchitis by an older age of onset, by chronic persistent wheeze or breathlessness with production of winter sputum, and a history of heavy cigarette smoking. There must therefore be important reservations about the accuracy of making a single diagnosis in some patients in any retrospective survey of asthma mortality.

Thirty-eight patients dying outside hospital (23 necropsies) and 15 in hospital (4 necropsies) were thought to have died from asthma (details below). Examples are given below of false-negative certifications (asthmatics not certified as such) and the more numerous false-positive certifications (patients certified as dying from asthma, in whom the above criteria were not met).

Results

A total of 83 death certificates were examined, comprising 61 (73%) deaths outside hospital and 22 (26%) inside. From both groups 72 certificates mentioned asthma as a cause of death, and the remaining 11 were examined because they fell into the under-45-year-old bronchitis and pneumonia group. We thought that a total of 53 deaths were due to asthma and 22 to other causes. In eight information was not adequate to form an opinion and in one the hospital case notes were not traceable. The number of patients fulfilling the diagnostic criteria for asthma were: necropsy evidence (27); convincing clinical history (52); and significant reversibility of airways obstruction (27). Twenty-seven patients fulfilled two criteria and 13 fulfilled three.

DEATHS FROM ASTHMA OUTSIDE HOSPITAL

General

There were 20 female and 18 male patients whose age at death ranged from 18 months to 81 years (mean 52·1 years for male and 50·4 year for female patients). Table I shows the age of patients at the time of onset of their asthma.

The mean length of history for male patients was 11·9 years (range 6-30) with 21·5 years (range 4 months to 40 years) for female patients.

TABLE I—*Age at onset of asthma of patients who died outside hospital*

	0–9	10–	20–	30–	40–	50–	60–9
No of patients	5	7	4	10	3	6	3

TABLE II—*Time from onset of asthma attack to death*

	<30 min	30 min–1 h	2–12 h	12–24 h	1–7 days	>7 days
No of patients.. ..	1	3	4	6	5	2

TABLE III—*Age at onset of asthma of patients who died in hospital*

	0–9	40–9	50–9	60–9
No of patients	2	7	5	1

The difference was significant ($t=3·55$, $0·01<p<0.001$). Deaths were distributed equally throughout the months of the year (χ^2 2·98, $p>0·99$). The time of death was known in 55% (21/38) of deaths outside hospital and showed a diurnal variation that peaked at 0800, which was, however, not significant (χ^2 8·43, $p>0·05$). Twenty-three patients had had previous hospital admissions for asthma—20 once and three more than three. Six patients (16%) died within three weeks of their discharge from hospital after an asthmatic attack. Twenty-two (58%) had never attended a clinic for assessment of asthma.

Maintenance treatment

Out of 38 patients, 14 were taking oral corticosteroids (prednisolone) in a dose between 5 and 10 mg daily, 25 were using oral bronchodilators, 21 aerosol bronchodilators, and eight were inhaling corticosteroid preparations.

Final attack

Details of duration were known in 55% (table II) and varied from 20 minutes to 12 days.

Fourteen patients (37%) had attended their general practitioner complaining of increased symptoms in the two weeks before their death, but no significant alterations to treatment were made. Eleven died within four days of the last visit.

Fourteen patients sought medical help in the final attack, but three died before the general practitioner arrived. Eight patients did not seek help, no information is available on 16, and 11 were alive when medical help arrived. Treatment was not changed in three cases, antibiotics were added in three, oral steroids were increased to 20 mg daily in three, two were given a bronchodilator aerosol, and in one an intramuscular bronchodilator was given. Arrangements for admission to hospital were made for two patients.

DEATHS FROM ASTHMA IN HOSPITAL

General

There were fifteen deaths from asthma in hospital; nine women and six men, aged from 42 to 81 (mean 54 for men and 63·9 for women). Table III shows the age of patients at the time of onset of their asthma.

The mean length of history for men was 11·8 years (range 6-16) with 17·7 years (range 1 month to 40 years) for women. This difference was not significant ($t=0·89$, $p>0·1$). Deaths tended to occur in spring and autumn, but this was not significant (χ^2 9·97, $p>0·1$). The time of death was known in all patients and did not show a significant diurnal variation (χ^2 5·72, $p>0·1$). Thirteen patients had had previous admissions to hospital for asthma, eight once and five on multiple occasions. Eleven patients (80%) had attended a clinic for asthma, six on one occasion only.

Maintenance treatment

Of the 15 patients with asthma, 12 were taking oral prednisolone 5-15 mg daily before admission. Twelve were taking oral bronchodilators and only seven were using aerosol bronchodilators. One patient was using aerosol corticosteroid.

Final attack

The length of the final attack ranged from 12 hours to 14 days before admission. Up to the time of admission three had continued in their job with difficulty and six had become completely bedbound when viewed in accord with the functional classification of Jones.[12] When first seen in hospital seven were fully alert, four were said to be drowsy, and three were unconscious. Central cyanosis was present in seven, the pulse rate was recorded as 88-120/min, but there was no mention of pulsus paradoxus. Spirometry performed on two of the conscious patients on admission showed a peak exploratory flow rate of 80 and 100 l/min. Arterial blood gases were measured on four patients.

In the first two hours seven patients were given intravenous hydrocortisone (100-200 mg), nine intravenous aminophylline (250-500 mg), and five oral steroids (prednisolone 5-10 mg). Over the first 24 hours after admission nine patients had received intravenous aminophylline (250-500 mg), seven intravenous hydrocortisone (100-800 mg), and eight oral prednisolone (5-40 mg); but no patient received an infusion of bronchodilator or nebulised bronchodilator over this period or subsequently. Similarly, in no case was treatment monitored over the initial 24 hours or subsequently with serial spirometric measurements or repeated blood gas analysis.

The mean survival time was 10 days, with a range of 12 hours to 30 days. Four patients were given assisted ventilation, three within the first few hours (unconscious on admission). Sedation was given in five patients and was related to death in one, no case being ventilated. Left lower lobe pneumonia was a complicating cause of death in one patient.

Death certification: false-positive certificates

Seven certificates that stated asthma as a cause of death did not meet our diagnostic criteria. Examples are given below.

Case 1—A woman aged 66 with a 20-year history of steroid-dependent asthma developed sudden cardiac pain and collapsed. Necropsy showed severe coronary atheroma with occlusions; there was no bronchial obstruction, plugging, or airtrapping, but the lungs were oedematous. The probable cause of death was left ventricular failure.

Case 2—This woman aged 60 had had progressive dyspnoea for six years. She was a heavy smoker, and symptoms did not improve with corticosteroids. The chest radiograph showed gross bilateral bullous emphysema. The certified cause of death was cor pulmonale due to asthma, but the probable cause was cor pulmonale due to emphysema.

Case 3—A woman aged 59, who was a heavy smoker with a 15-year history of winter bronchitis and intermittent right heart failure, was admitted with hypercapnoea. The certified cause of death was cor pulmonale, asthma, and chronic bronchitis, but the probable cause was cor pulmonale and chronic bronchitis.

Death certification: false-negative certificates

In the group aged under 45 11 were certified as having bronchitis, emphysema, or pneumonia. Six of these had pneumonia (five necropsies), and nine were certified as dying of bronchitis or emphysema (four necropsies). We think three of these probably died of asthma but in only two (cases 1 and 2) was the evidence sufficient for inclusion in the asthma group for further analysis. These three cases are detailed below.

Case 1—A man aged 36 had a 10-year history of wheezing. The certified causes of death were respiratory failure, chronic bronchitis, and bronchopneumonia. At necropsy the lungs were voluminous, and the distal bronchi were plugged with viscid grey sputum. As the necropsy criteria were satisfied he was included in the asthma group.

Case 2—A woman aged 42 had developed intermittent wheeze at the age of 3 years. She had had several hospital admissions with bronchospasm, the last after a three-week history of deterioration. She was certified as having died of respiratory failure, cor pulmonale, chronic bronchitis, and emphysema, but we think she died of asthma. There was sufficient evidence for her inclusion in the asthma group.

Case 3—A man aged 45 had a one-year history of intermittent wheezing and dry cough. There were numerous visits to his general practitioner, and improvement when taking bronchodilators was recorded. He was certified as having died of respiratory failure and chronic bronchitis but we think he may have died of asthma. There was insufficient evidence for inclusion.

Discussion

Of the 72 patients certified as having died from asthma, in only 53 was the cause thought to be consistent with the available clinical information. Thus in 29% (6/21) of deaths in hospital and in 27·5% (4/51) of deaths outside hospital, the cause of death considered by us was other than that certified. Of the 11 patients stated to have died from chronic bronchitis or bronchopneumonia under the age of 45, two patients were asthmatic and a further one almost certainly was but adequate records could not be traced for this to be confirmed. These findings are in keeping with those of the Medical Services Study Group of the Royal College of Physicians,[9] who found a major discrepancy in the death certificate of between 12·5% and 35%, depending on source. Undiagnosed asthmatics were present in the group dying under 45 years labelled as "chronic bronchitis." Since this group represents only a very small proportion of the total number of deaths from "chronic bronchitis/pneumonia" further asthmatic deaths are probably concealed in this manner, but because of the numbers concerned, this could not be investigated directly. This loss of cases, together with other causes of wrong certification—for example, left ventricular failure—makes us suspect that the number of deaths considered here is a substantial underestimate of the true numbers affected.

Analysis of the deaths thought to be due to asthma show broadly similar results to other surveys.[6-8] That deaths tend to occur in patients with longer histories is confirmed. Female patients dying outside hospital had a significantly longer history than men. The previously reported trend[8] of deaths in hospital occurring in the early hours is not shown in this survey, which may be partly explained by the age limits set by the previous survey.[8] Our finding that 65% of acute hospital admissions had had a severe episode of wheezing lasting longer than 24 hours agrees with recently published observations,[13] and the information from the deaths occurring outside hospital supports our findings that the more severe episode is preceded by a rather longer period of poor control.

Of those patients dying outside hospital, a minimum of 37% did not call their doctors. This underestimation of severity may be reduced by educating the patients and their relatives to call for medical help promptly when symptomatic relief fails. One-third of those dying outside hospital had sought medical help within a few days of death, but had not had their treatment altered. A further quarter had called for an emergency visit during their final attack, but did not receive sufficient treatment.[14 15] This failure to appreciate the severity was due, at least in part, to lack of objective measurement of the severity of the asthmatic attack in terms of airways obstruction, pulse rate, and pulsus paradoxus. Underusage of corticosteroids and effective bronchodilators also probably played a part.

Deaths in hospital showed the same trend. The severity of airways obstruction and its sequelae was not adequately appreciated, and objective measurements and blood gas analysis were too infrequently, if at all, performed. The initial treatment was inadequate by accepted standards[14 15] with underusage of intravenous and nebulised bronchodilators, and parenteral and oral corticosteroids. Again the treatment was not objectively assessed with serial estimations of airflow obstruction and repeat blood-gas analysis. This may account for the underuse of temporary artificial ventilation as a supportive measure until the therapeutic effects of treatment have had time to take effect.

Deaths cannot be attributed to these findings, since the care of this group is not compared with survivors. The results, however, support the views expressed by Seaton[16] that there is still a considerable discrepancy between the known availability of effective treatment and its logical application in the management of patients. The assumption that deaths from asthma occur because of shortcomings in the appreciation of severity and failure to apply generally accepted principles of treatment would need a further prospective study comparing survivors as controls for its resolution. To identify failure of care as a risk factor, matching for age, sex, atopic state, cigarette consumption, and severity of the final attack in patients treated in the same unit would be necessary. Only then could the question of identifying the specific risk factors be proved. We believe, however, that such a survey would be impossible because of the difficulty of finding a sufficiently close match for a death from asthma from within a single unit or hospital for the comparison to have any relevance. Instead, we suggest that a study of the management given to survivors of severe asthmatic attacks might be of interest, but we suspect that this would

be found to be no better than that given to patients who failed to survive, if cases were drawn from hospital populations similar to that in our study.

We thank the staff of the Birmingham Family Practitioner Committee for their assistance; HM Coroner for Birmingham, Dr R M Whittington, for access to pathologists' reports; all Birmingham physicians who gave us access to medical records; and Mrs P Ormerod and Mrs Pauline Taylor for secretarial help.

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(*Accepted 10 December 1979*)

MATERIA NON MEDICA

The Pope in Des Moines

When it was announced that Pope John Paul II would visit Boston, New York, Philadelphia, Des Moines, and Washington a great many probably reread the sentence to be sure that it was no mistake. Des Moines? Henry Kissinger makes the point in his recent memoirs when, wishing to ridicule the behaviour of President Nixon's advance men on the President's first trip to Europe, he writes " . . . they solved their problems as if they were running a political stopover in Des Moines." But Des Moines, which calls itself "the surprising place," is far from being a quiet country town.

A crowd of up to 400 000 could be expected for the Pope's visit, and immensely complicated arrangements were needed to control the flow of people. The site chosen was Living History Farms, an open-air museum of some 500 acres, located on the western fringe of the city. The farms are real, and past, present, and future methods of farming are authentically performed there. An altar of oak was built on a grassy knoll, with a logo representing the four seasons, each in a different colour, the patterns being that of contour ploughing.

The medical problems appeared immense, with huge estimates of myocardial infarctions, lost children, and large numbers of elderly and disabled requiring help. Mercy Hospital was, appropriately, chosen to organise medical care, but all hospitals, the Red Cross, and volunteers from all walks of life were involved. Four field hospitals were set up, with an elaborate communications system. There was talk of the city being inundated with cars and travel being impossible. To make matters worse a rock group called Kiss were giving a concert the same night and refused to cancel. They had a reputation for inducing hysterical behaviour in teenagers and were expected to add to the demand on hospitals.

I had to be on call and so could not attend the Mass. To be sure of reaching my hospital I had to sleep there on the night before the Pope's visit. On the day the hospital was quiet and I was finished by 11 am. Looking out I saw empty streets. Security informed me that all roads were clear and so I drove home to watch the Pope on television. The streets were deserted and the freeway almost empty. The temperature had suddenly fallen and clouds scudded across the sky.

Perhaps, I thought, they have overdone the warnings and everyone has stayed at home. Later, as the Pope arrived, the clouds blew away and when Mass was held a huge crowd was there under a bright blue sky with the burnished trees shimmering in the golden light. The scene was reminiscent of the Sermon on the Mount, and when the choir sang "Praise Him, Alleluia," one wondered if they meant him rather than Him. Afterwards the Pope flew on to Chicago, the great Polish city of America, and the day ended in Des Moines in a vast blazing sunset.

The next day it became clear what had happened. Many people came in chartered buses rather than cars. They followed instructions and arrived and left calmly. There was no damage to land or property despite a crowd of 400 000, and only a small amount of litter. There were a few minor accidents but no major medical crises. The organisation had been superb and the crowd immaculate. I criticised myself for having doubted that all would be well, and should have known better of the State whose flower is the wild rose and whose tree the oak.

Incidentally, the rock concert was a flop. It seems that the teenagers preferred the Pope, which is surprising, even in Des Moines.—C T FLYNN (consultant physician, Des Moines, Iowa).

Chemical producers

Not long back I saw some glow worms emitting their weak, cold light. What was happening, of course, was that the female beetle was trying to entice a male, presumably in the hope of closer acquaintance. It seemed remarkable that the glow worm was able to initiate, by enzyme action, the oxidation of the complex light-producing chemical, luciferin, to cause illumination and that, later, the oxidised product could revert to the basic chemical.

Obviously, specialised chemical substances are of great importance in the lives of many insects. A good example is the small ground beetle, the bombardier beetle; if threatened, this beetle will squirt a jet of caustic fluid from the anus and, not only is the liquid irritant, but it is very hot in temperature. Then, as is well known medically, blister beetles contain the irritating substance cantharidin, which is concentrated in the wing cases. Cantharidin is also present in the body fluid of the oil beetle; if this beetle is handled then the unpleasant juice is extruded through the joint tissues.

Wood ants will squirt a formic acid solution if they are provoked, and the same acid is also used as a repellent by the caterpillars of both the puss and lobster moths. The puss moth larva will eject formic acid at a strength of nearly 40% and, when the imago form is about to emerge from its chrysalis, it produces an alkaline solution from its mouth which softens the hard, silken pupal wall. More elaborate chemicals are emanated by female moths at mating time, which can be detected by the male at great distances.

As with moths, some butterfly species will produce deterrent chemicals; for instance, the larva of the swallowtail, if threatened, raises a spiky organ from behind the head which discharges an unpleasant and smelly gas. The caterpillars of the small tortoiseshell and peacock butterflies will regurgitate an acrid liquid if they are touched.

Most people fear the stings of bees and wasps and their insect or spider prey have reason to fear them also. It seems that many insects, some quite minute in size, are able to synthetise chemicals of great complexity, potency, and variety of action. Doubtless we should think twice before treading on that beetle or caterpillar on the garden path.—PHILIP RADFORD (general practitioner, Brenty, Bristol).