# Morbidity from chronic asthma

JL STELLMAN, JE SPICER, RM CAYTON

*From East Birmingham Hospital, Birmingham*

ABSTRACT   Seventy asthmatic patients newly referred to a hospital outpatient clinic have been studied. In one-third of the patients the diagnosis of asthma had not been considered. Eight patients presented with an $FEV_1$ of one litre or less. Acute severe asthma is commonly believed to precede death from asthma, but patients with more chronic symptoms may have equally severe airways obstruction without appearing acutely ill. Failure to diagnose and treat such patients may be contributing to our present inability to reduce the death rate from asthma.

The 1960-65 epidemic increase in asthma deaths[1] has stimulated interest in the management of acute severe asthma. Nevertheless about 1500 patients continue to die from asthma each year in the UK;[2] a similar number were dying in 1960 immediately before the epidemic began.[1] Thus a graph of asthma deaths now shows a plateau, uninfluenced by the use of selective $\beta$-sympathomimetic drugs, intensive care units, and self-admission units. The natural history of chronic asthma and its relationship to life-threatening asthma is poorly documented. A recent study of the treatment of asthma in general practice has shown that symptomatic patients suffering from airways obstruction may be receiving inadequate treatment.[3] We have investigated asthmatic patients newly referred to a hospital outpatient clinic, comparing initial lung function measurements with those made after stabilisation on an adjusted treatment regimen.

## Methods

All patients had bronchial asthma, defined as a condition in which widespread narrowing of the airways changes its severity over short periods of time, either spontaneously or under treatment.[4] Consecutive patients referred by their general practitioner to a single hospital outpatient clinic were studied. Details of drug treatment were documented with the history and physical examination. The severity of dyspnoea was graded.[5] The forced expiratory volume ($FEV_1$) and forced vital capacity (FVC) were measured at the initial and each subsequent attendance with a Vitalograph spirometer. Results were compared with predicted values taken

Address for reprint requests: Dr RM Cayton, East Birmingham Hospital, Bordesley Green East, Birmingham B9 5ST.

from Cotes.[6] The patient's asthma was stabilised, primarily on an inhaled bronchodilator drug, with the addition of an oral bronchodilator preparation, taken at night, inhaled beclomethasone dipropionate and oral prednisolone, as required. Mean values were recorded $\pm$ 1 standard deviation, and Student's *t* test used for paired comparisons.

## Results

Seventy asthmatic patients (30 males and 40 females) aged 37 ($\pm$ 15) years were included in the study. Asthma had not previously been diagnosed in 22 (31%) patients, who had been symptomatic for a mean of 3·8 ($\pm$ 4·7) years. The remaining 48 patients were referred for reassessment and had a longer mean history of asthma (13·6 $\pm$ 12·3 years).

The episodic nature of the symptoms of cough, wheeze, and breathlessness made it impossible to grade the patients' dyspnoea with any degree of accuracy. The initial $FEV_1$ ranged from 0·65-4·9 litres (23-132% predicted) in male and from being unrecordable to 3·9 litres (unrecordable-134% predicted) in female patients. The very low values measured in some patients are disguised by the mean presenting $FEV_1$, which was 2·2 ($\pm$ 1·0) litres or 71 ($\pm$ 27)% predicted. Significant improvement in $FEV_1$ followed stabilisation on an adjusted treatment regimen (p < 0·001). The mean stable $FEV_1$ was 2·7 ($\pm$ 0·85) litres, or 91 ($\pm$ 21)% predicted. There was a 41·5 ($\pm$ 62)% improvement in $FEV_1$ which was similar in males and females (fig 1).

Eight patients (seven female) aged 23-71 years presented with an $FEV_1$ of 1·0 litre or less (table 1). Four patients required immediate hospital admission. One had an unrecordable $FEV_1$ and three had received no treatment for asthma, despite being symptomatic for three months, one month, and
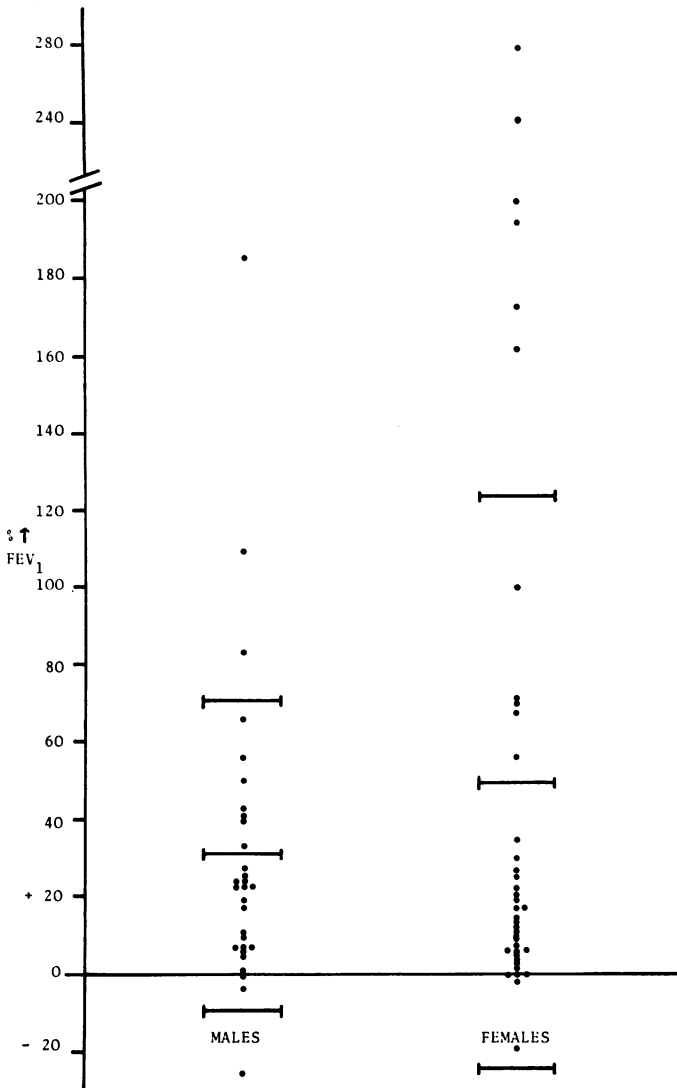
Fig 1 *Percentage increase in FEV₁ after restabilisation of asthma on adjusted treatment regimen. Bars represent mean ± 1 standard deviation.*

Table 1  *Details of patients presenting with FEV₁ one litre or less*

| | Age (yr) | Sex | Duration of asthma (yr) | Rx | FEV₁ (l) | Pao₂ (kPa) | PaCo₂ (kPa) |
|---|---|---|---|---|---|---|---|
| AL* | 31 | F | 3/12 | Nil | NR | 8·5 | 4·4 |
| DB* | 53 | F | 1 | Nil | 0·5 | 8·9 | 4·0 |
| GC* | 23 | F | 1/12 | Nil | 1·0 | 8·7 | 3·9 |
| GP* | 51 | M | 4 | B | 0·65 | 9·7 | 4·9 |
| PH | 37 | F | 27 | S/B | 0·85 | 10·3 | 4·0 |
| MT | 47 | F | 26 | S/B | 0·65 | 9·2 | 5·2 |
| DC | 57 | F | 26 | B | 0·5 | — | — |
| MT | 71 | F | 3 | B | 0·5 | — | — |

S = steroids, B = bronchodilators, NR = not recordable,
* = hospital admission.

seven weeks respectively. All patients had severe airflow obstruction as indicated by the FEV₁ and five had an arterial oxygen tension (Pao₂) less than 10·0 kPa. After treatment, the mean FEV₁ in these eight patients increased from 0·67 (± 0·21) litres to 2·04 (± 0·56) litres (fig 2). The percentage improvement was 205 (± 41)%.

Eight patients (11%) had received no treatment for their symptomatic asthma (table 2). Oral bronchodilator drugs had been prescribed for 45 of the 70 patients (64%) and inhaled bronchodilators for 37 (53%). Initially 23 (33%) patients were taking
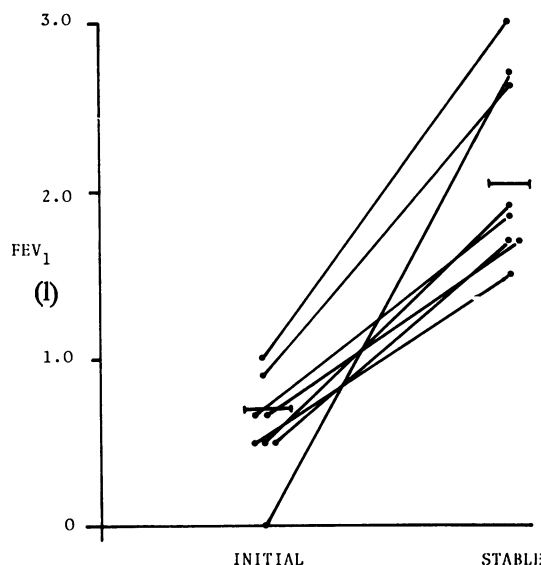
Fig 2 *Comparison of initial and stable recordings of FEV$_1$ in patients presenting with FEV$_1$ one litre or less. Bars represent mean.*

Table 2 *Summary of initial and stable drug treatment DSCG = sodium cromoglycate*

| | | Initial | | Stable | |
|---|---|---|---|---|---|
| | | *No* | *%* | *No* | *%* |
| Nil | | 8 | 11 | | |
| Bronchodilator | Oral | 45 | 64 | 24 | 34 |
| | Inhaled | 37 | 53 | 70 | 100 |
| Steroids | Oral | 23 | 33 | 11 | 16 |
| | Inhaled | 6 | 9 | 28 | 40 |
| DSCG | | 9 | 13 | 13 | 19 |

oral corticosteroids (mainly prednisolone) and 6 (9%) inhaled steroid preparations. The major change in treatment pattern was the prescription of an inhaled bronchodilator drug for all patients. When stabilised on treatment rather more patients (28, 40%) were using a steroid aerosol but fewer patients (11, 16%) required oral steroids.

## Discussion

Bronchial asthma follows an unpredictable course in many patients. It is a disease punctuated by relapses and remissions. Asthmatic attacks that appear clinically identical may vary widely in their pathology and physiology.[7] The airways obstruction in asthma is caused by mucous plugging and increased bronchomotor tone.[8] The relative importance of each component in an individual patient is speculative and may be related to pathogenesis.

In patients with acute exacerbations of asthma the

symptom of dyspnoea correlates imprecisely with measurements of lung function such as FEV$_1$ and Pao$_2$.[9-11] Nevertheless dyspnoea continues to be regarded as the cardinal symptom of bronchial asthma and progress and treatment monitored by its severity.

The diagnosis of asthma requires the demonstration of reversible airways obstruction.[4] Serial measurements of peak flow (PEFR) or FEV$_1$ in patients complaining of recurrent cough, wheeze, and breathlessness may be revealing. Failure to make a confident diagnosis in almost one-third of the patients in this study probably contributed to inadequate treatment being prescribed. A patient will seek medical attention if he suffers persistent and distressing symptoms of wheeze and breathlessness and is then likely to receive treatment. Patients with chronic or episodic symptoms may either not seek medical advice or be misdiagnosed and receive inappropriate or inadequate treatment. In this study of patients with chronic asthma the symptom of dyspnoea proved impossible to quantitate. Nevertheless, once the diagnosis of asthma was established, the patients responded to treatment with a mean 40% increase in FEV$_1$.

Several patients had evidence of severe airways obstruction. They were not suffering from "status asthmaticus" nor from "acute severe asthma".[13] Both these terms attempt to define an acute and potentially fatal asthma attack. However, it is not unreasonable to postulate that these patients with symptoms of cough, wheeze, and breathlessness lasting several weeks, a low FEV$_1$ and often a low Pao$_2$ were in a critical phase of their disease.

Patterns of airflow obstruction in bronchial asthma may relate to pathogenesis.[14] It has been suggested that they may be used to identify patients at high risk and that patients with gradually deteriorating airflow obstruction have an increased risk of sudden death from asthma.[15] The clinical categorisation of patients into those with acute and those with chronic symptoms neglects the severe functional impairment which may be present in the latter group. The diagnosis of bronchial asthma embraces a spectrum of disease processess. Identification of the pattern of airflow obstruction may not only lead to better patient management but also provide information about pathogenesis.

The symptom of dyspnoea has once again been shown to be an unreliable guide to the severity of asthma. The combination of a diagnosis of asthma' symptoms of cough, wheeze, and breathlessness with serial measurements of lung function should be used to alert the medical practitioner to a potentially critical situation. Neither "status asthmaticus" nor

"acute severe asthma" necessarily precedes death from asthma.[13] Patients with chronic asthma may be equally at risk.

Diagnosis and treatment of these patients is rewarding and may help to reduce the number of asthma deaths.

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