Tuberculosis at the end of the 20th century in England and Wales: results of a national survey in 1998

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Abstract

Background—A national survey of tuberculosis was conducted in England and Wales in 1998 to obtain detailed information on the occurrence of the disease and recent trends. This survey also piloted the methodology for enhanced tuberculosis surveillance in England and Wales and investigated the prevalence of HIV infection in adults with tuberculosis.

Methods—Clinical and demographic data for all cases diagnosed during 1998 were obtained, together with microbiological and geographical data where available. Annual incidence rates in the population were estimated by age, sex, ethnic group, and geographical region using denominators from the 1998 Labour Force Survey. Incidence rates in different subgroups of the population were compared with the rates observed in previous surveys. The tuberculosis survey database for 1998 was matched against the Communicable Disease Surveillance Centre HIV/AIDS database to estimate the prevalence of HIV co-infection in adult patients with tuberculosis.

Results—A total of 5658 patients with tuberculosis were included in the survey in England and Wales (94% of all formally notified cases during the same period), giving an annual rate of 10.93 per 100 000 population (95% CI 10.87 to 10.99). This represented an increase of 11% in the number of cases since the survey in 1993 and 21% since 1988. In many regions case numbers have remained little changed since 1988, but in London an increase of 71% was observed. The number of children with tuberculosis has decreased by 10% since 1993. Annual rates of tuberculosis per 100 000 population have continued to decline among the white population (4.38) and those from the Indian subcontinent, although the rate for the latter has remained high at 121 per 100 000. Annual rates per 100 000 have increased in all other ethnic groups, especially among those of black African (210) and Chinese (77.3) origin. Over 50% of all patients were born outside the UK. Recent entrants to the UK had higher rates of the disease than those who had been in the country for more than 5 years or who had been born in the UK. An estimated 3.3% of all adults with tuberculosis were co-infected with HIV.

Conclusions—The epidemiology of tuberculosis continues to change in England and Wales and the annual number of cases is rising. More than one third of cases now occur in young adults and rates are particularly high in those recently arrived from high prevalence areas of the world. The geographical distribution is uneven with urban centres having the highest rates. The increase in the number of cases in London is particularly large. Tuberculosis in patients co-infected with HIV makes a small but important contribution to the overall increase, particularly in London. To be most effective and to make the most efficient use of resources, tuberculosis prevention and control measures must be based on accurate and timely information on the occurrence of disease. A new system of continuous enhanced tuberculosis surveillance was introduced in 1999, based on the methodology developed in this national survey.

Keywords: tuberculosis; national survey; HIV co-infection

National surveys of tuberculosis have been carried out in England and Wales every 5 years since 1978. These surveys have generally collected clinical, microbiological, and demographic data on notified cases for the first 6 months of the year and, in some, more limited data (on a restricted age group) in the second 6 months. The surveys have supplemented information on the incidence of tuberculosis from notifications and laboratory reports, permitting analyses of the trends in incidence in population subgroups defined by ethnic group and country of birth. The detailed information on the epidemiology of tuberculosis provided by the surveys has underpinned decisions on national treatment, control and prevention guidelines. A final survey was carried out in 1998 with the additional objective of piloting the methodology for a new system of continuous enhanced tuberculosis surveillance.

Methods

The survey was carried out by the Public Health Laboratory Service in collaboration with the British Thoracic Society and the Department of Health. The methodology of the survey differed from that used in previous...
surveys in certain key ways—namely, data were collected on all patients identified in the calendar year 1998, whether or not notified, and a coordinator was nominated by each district health authority with responsibility for ensuring that completed survey forms were collected and forwarded to the survey team at the Communicable Disease Surveillance Centre (CDSC), London.

Microbiological information, including drug susceptibility, on isolates reported to Mycobnet (the UK Mycobacterial Resistance Network, a surveillance system for all culture confirmed cases of tuberculosis) was matched by name and date of birth with the tuberculosis survey database. This supplemented the information provided by clinicians.

Matching was carried out between the 1998 tuberculosis survey database and the CDSC HIV/AIDS database, using previously described methodology, to estimate the extent of co-infection of tuberculosis and HIV in adults.

Population figures for England and Wales were obtained from the 1998 Labour Force Survey (winter quarter and 1998 local area database). Rates were not calculated for population subgroups of less than 10 000 as the relative standard errors would have been too large.

In earlier surveys most analyses were of new (previously untreated) cases. Analyses of trends from 1988–98 used data from the earlier survey databases held at CDSC. To permit comparison, data from earlier surveys were re-analysed to include all patients, whether or not previously treated. As the earlier surveys had collected patient information for only 6 months in some age groups, these data were multiplied by scaling factors (as published previously) to give annual estimates for comparison of trends. Numbers cited in this article for the 1988 and 1993 surveys are therefore the annual numbers.

The 1998 survey data were entered and held in Access 95 Version 7 software and exported to SPSS Version 10 (SPSS Inc, Chicago, Illinois) for analysis. Analysis of HIV prevalence was conducted using Access 95 Version 7.

Results
In all, 6032 cases of tuberculosis were formally notified during weeks 1–52 of 1998. A revised estimate (5876) of the total for the study period (1 January to 31 December 1998) was derived from figures provided by district coordinators which took into account local over- and under-notification. A total of 6400 survey forms was received, of which 343 were excluded on the basis of information on the form (230 patients received chemoprophylaxis only and 113 were diagnosed or notified outside the survey period). A further 399 were excluded after they were subsequently found not to have tuberculosis. Thus, 5658 patients were eligible for inclusion in the survey. This represented 94% of all those formally notified and 96% of the district coordinators’ estimate.

A total of 3506 laboratory reports of tuberculosis isolates were reported to Mycobnet in 1998; 629 of these could not be matched with patients on the survey database. Insufficient information was available at the time of the survey to determine their eligibility so they were excluded. A subsequent audit of Mycobnet reports for 1998 carried out in 2000 (PHLS, unpublished data) determined that half of those reports for which information could be obtained would not have been eligible for inclusion in the survey—for example, patients notified outside the survey period or not resident in England and Wales.

Four hundred and forty five patients (7.9%) had been previously treated and 98 (1.7%) were known to have been diagnosed post mortem. The overall annual rate of tuberculosis in England and Wales was 10.93 per 100 000 (95% CI 10.87 to 10.99).

Age and sex
The greatest numbers of cases were reported in those aged 15–34 years, followed by the 35–54 and 55–74 age groups (fig 1A). More cases overall were reported in males in all age groups, except in children where similar numbers were reported in boys and girls (fig 1A). The number of male patients was equal to or greater than the number of females in all but the Indian subcontinent (ISC: India, Pakistan and Bangladesh) ethnic group in which there were more females (fig 1B–D).

The lowest annual rates per 100 000 were seen in children (<15 years of age): males 3.5 (95% CI 3.4 to 3.6); females 3.7 (95% CI 3.6 to 3.7). In all the adult age groups males had higher rates than females (fig 1A). In males the annual rate per 100 000 generally increased with increasing age, peaking in the elderly (>74 years) at 24.5 (95% CI 23.7 to 25.3). In contrast, the annual rate in females was highest in the 15–34 year age group and did not increase in the elderly. These overall rates (fig 1A) hide substantial differences in age specific rates in ethnic subgroups of the population (fig 1B–D).

Ethnic group
In 1998 38% of patients with tuberculosis were reported from the ISC ethnic group. The proportion of white patients was also 38%, and 13% of patients were of black African ethnic origin (table 1). As in previous surveys, the numbers of cases in both the black Caribbean and Chinese ethnic groups have remained small, comprising 2.2% and 1.8% of all cases, respectively. The highest annual rate per 100 000 was observed in the black African ethnic group (over 200), and an increased rate since the last survey was seen in the Chinese ethnic group which was nearly three times higher than the rate for those of black Caribbean origin. Rates in the white ethnic group were low (less than 5), while rates in the ISC ethnic group have remained high (over 100).

Place of birth and year of entry to the UK
Fifty six per cent of patients were born abroad compared with 45% in 1988 and 50% in 1993 (data not shown). Of those born in the UK,
most (77%) were of white ethnic origin. Most of those born abroad were of ISC ethnic origin (56%). Of all patients with known year of entry, 60% had been in the UK for longer than 5 years; 57% of patients of black African ethnic origin born abroad (where year of entry was known) were recent entrants to the UK (during the 5 years before the survey), whereas only 32% of ISC patients were recent arrivals (table 2).

For all ethnic groups except black Caribbean the rates of tuberculosis were highest in those born abroad who had recently entered the UK, about one third lower in those born abroad but who had been in the UK for more than 5 years, and lowest in those born in the UK. Patients of ISC ethnic origin had the highest rates of all those born in the UK. The proportion born abroad for whom year of entry was unknown ranged from 22% (in the ISC ethnic group) to 39% (white ethnic group); had these patients’ year of entry been known, corresponding rates of disease may have been higher than stated in table 2.

Geographical distribution
Of all cases reported, 40% were from London. The annual rate per 100 000 in London (31.6 (95% CI 31.2 to 32.1)) was four times the rate for the rest of England and Wales (7.65 (95% CI 7.61 to 7.70)). Annual rates for the remaining regions ranged from 4.22 (95% CI 4.16 to 4.29) in the Eastern region to 11.9 (95% CI 11.7 to 12.0) in the West Midlands (table 3). Ten of the 15 district health authorities with more than 100 cases and/or an annual rate of >25 per 100 000 were in London (table 3).

TRENDS IN NUMBER OF CASES AND ANNUAL RATES OF DISEASE, 1988–98
The total number of patients with tuberculosis in 1998 (5658) represents an increase of 11% since the last national survey in 1993 and 21%
High numbers of patients born abroad with unknown year of entry for some ethnic groups means that some rates for either recent entry or longer residents may be higher than stated.

**Arrived in the UK prior to 1994.**

*Arrived in the UK from 1994 to 1998.

Rate per 100,000 population.


††Rate per 100,000 population.

**Source of population data: Labour Force Survey Local Area District database to 1998.**

Since the survey in 1988, annual rates of disease have also increased (table 1).

**Age group**

After an increase of 42% between the 1988 (286 patients) and 1993 surveys (407 patients), the number of children with tuberculosis decreased to 365 in 1998. The number of children with tuberculosis in London, however, has doubled since 1993 (to 90 patients). The number of elderly patients (over 74 years) showed little change between 1988 and 1998 (from 546 to 551). Other age groups (15–34, 35–54, 55–74 years) showed an increase in numbers of patients over the last decade which was greatest in the 15–34 year age group (1428 in 1988; 1554 in 1993; 1977 in 1998).

**Ethnic group**

The proportion of patients from the white ethnic group declined from 54% in 1988 to 44% in 1993 and 38% in 1998. The proportion of patients in the black African ethnic group, however, has risen from 1.7% to 7.0% and 13% in 1988, 1993, and 1998, respectively. The number of patients from the black Caribbean and Chinese ethnic groups decreased slightly between 1988 and 1993. Between 1993 and 1998 the numbers of patients of black Caribbean origin increased slightly, and those of Chinese ethnic origin increased by a factor of 2.5.

The rate in the black Caribbean ethnic group did not change substantially between 1988 and 1998. In the ISC population the rate declined slightly, although it was 30 times higher than the rate for the white ethnic group in 1998. During the same period the rate for patients from the black African ethnic group increased by about a factor of three. The decline in the rate in the ISC population and the rise in the rate in the black African ethnic group remained even after standardising to the 1988 population, taking into account age, place of birth, and year of first entry to the UK (data not shown). More detailed analysis of the contribution of different demographic factors will be carried out separately.

### Table 2 Number of patients with tuberculosis and rate of disease by place of birth, year of entry to the UK, and ethnic group in England and Wales, 1998

<table>
<thead>
<tr>
<th>Ethnic group</th>
<th>UK born</th>
<th>Born abroad (total)</th>
<th>Born abroad (recent entrants)*</th>
<th>Born abroad (longer residents)**</th>
<th>Born abroad (year of entry unknown)†</th>
<th>Unknown/missing place of birth</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>Rate††</td>
<td>95% CI</td>
<td>No</td>
<td>Rate†</td>
<td>95% CI</td>
<td>No</td>
</tr>
<tr>
<td>White</td>
<td>1784</td>
<td>3.89</td>
<td>3.86 to 3.91</td>
<td>219</td>
<td>9.85</td>
<td>9.61 to 10.1</td>
<td>55</td>
</tr>
<tr>
<td>ISC</td>
<td>373</td>
<td>44.8</td>
<td>43.0 to 46.7</td>
<td>1656</td>
<td>177</td>
<td>170 to 184</td>
<td>411</td>
</tr>
<tr>
<td>Black</td>
<td>40</td>
<td>28.7</td>
<td>26.1 to 31.9</td>
<td>657</td>
<td>308</td>
<td>285 to 335</td>
<td>277</td>
</tr>
<tr>
<td>Caribbean</td>
<td>56</td>
<td>20.4</td>
<td>19.1 to 22.0</td>
<td>59</td>
<td>20.6</td>
<td>19.3 to 22.2</td>
<td>2</td>
</tr>
<tr>
<td>Other</td>
<td>55</td>
<td>9.50</td>
<td>9.1 to 10.0</td>
<td>362</td>
<td>82.1</td>
<td>77.8 to 87.0</td>
<td>143</td>
</tr>
<tr>
<td>Missing ethnic group</td>
<td>6</td>
<td>**</td>
<td>**</td>
<td>**</td>
<td>**</td>
<td>**</td>
<td>**</td>
</tr>
<tr>
<td>Total</td>
<td>2308</td>
<td>4.83</td>
<td>4.81 to 4.86</td>
<td>2959</td>
<td>73.6</td>
<td>72.3 to 75.0</td>
<td>891</td>
</tr>
</tbody>
</table>

*Arrived in the UK from 1994 to 1998.

**Arrived in the UK prior to 1994.

†High numbers of patients born abroad with unknown year of entry for some ethnic groups means that some rates for either recent entry or longer resident categories may be higher than stated.

††Rate per 100,000 population.


### Table 3 Number of patients with tuberculosis and rate of disease by NHS region in England and Wales with more than 100 patients with tuberculosis and/or a rate of >25 per 100 000 in 1998

<table>
<thead>
<tr>
<th>Region*</th>
<th>No of cases</th>
<th>Rate**</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>London</td>
<td>2244</td>
<td>31.6</td>
<td>31.2 to 32.1</td>
</tr>
<tr>
<td>West Midlands</td>
<td>625</td>
<td>11.9</td>
<td>11.7 to 12.0</td>
</tr>
<tr>
<td>North West</td>
<td>662</td>
<td>10.2</td>
<td>10.1 to 10.4</td>
</tr>
<tr>
<td>Northern &amp; Yorkshire</td>
<td>605</td>
<td>9.67</td>
<td>9.52 to 9.81</td>
</tr>
<tr>
<td>Trent</td>
<td>443</td>
<td>8.68</td>
<td>8.53 to 8.82</td>
</tr>
<tr>
<td>Wales</td>
<td>163</td>
<td>5.70</td>
<td>5.58 to 5.83</td>
</tr>
<tr>
<td>South West</td>
<td>212</td>
<td>4.41</td>
<td>4.34 to 4.49</td>
</tr>
<tr>
<td>Eastern</td>
<td>244</td>
<td>4.22</td>
<td>4.16 to 4.29</td>
</tr>
</tbody>
</table>

*April 1999 boundaries.

**Rate per 100,000 population.


Decline in the rate in the white population in England, 1978–98

The annual rate of disease in the white ethnic group has continued to decline with each survey between 1978 and 1998 (fig 2). The general trend of increasing rate with age persisted in both male and female patients, and was more marked in males. In females the rate per 100,000 only rose above 5 for those aged 65 years and over while in males this rate was reached by 35 years of age, increasing by a factor of three for men over 65 years.

Geographical distribution

With the exception of London, little change in the number of patients with tuberculosis has been observed in the NHS regions of England and Wales over the last decade. In London 927 more patients (an increase of 71%) were reached by 35 years of age, increasing by a factor of three for men over 65 years.

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There were urban areas other than London such as Leicester and Blackburn where the annual rates of tuberculosis were in the range of 30–50 per 100 000. However, although the rate in Leicester was greater than that for London, it has declined since 1988 (data not shown). Most of the high incidence urban areas outside Greater London had lower rates of tuberculosis in 1998 than in 1993. Only two showed substantial increases (Sandwell and Kirklees; data not shown).

SITE OF DISEASE
The site of disease was known for 5625 (99%) patients. The proportion of patients with pulmonary disease (with or without extrapulmonary tuberculosis) decreased from 67% in 1988 to 63% in 1993 and 62% in 1998. The proportion of patients with extrapulmonary disease alone increased to 38% from 33% in 1988 and 37% in 1993. Nine per cent of patients had both forms of disease (mediastinal glands and pleural effusion were considered extrapulmonary tuberculosis). A smaller proportion of children (49% of 358) than adults (63% of 5262) presented with pulmonary tuberculosis, while extrapulmonary disease alone was more common in children (51% vs 37%). The proportion of patients with pulmonary tuberculosis increased with increasing age, reaching 78% in patients aged over 74 years. Detailed results for site of disease will be reported separately.

DIAGNOSIS
A positive culture was reported on specimens from 60% of all patients (66% of those with pulmonary disease; 50% with extrapulmonary disease only) compared with 59% in 1988. The proportion with a positive culture was much lower in children (0–14 years) than in adults (22% vs 62%). A smear positive result for sputum was obtained for 27% of all patients, or 43% of patients with pulmonary disease.

In 1.6% of all cases (92/5658) the laboratory support for the diagnosis was based on histological examination alone, mostly in patients with extrapulmonary disease only.

DRUG RESISTANCE
Of the 3385 patients reported with a positive culture, 3052 were matched to the Mycobnet database and drug resistance data were available for isolates from 3046 of these. Isoniazid resistance was present in the isolates from 153 patients (5.0%) compared with 2.9% in 1988, while multidrug resistance (defined as resistance to isoniazid and rifampicin, with or without resistance to any other drug) was present in 21 (0.7%) compared with 0.6% in 1988. The number of patients known to have been previously treated declined from 474 (10.2%) in 1988 to 445 (7.9%) in 1998. Isolates from these patients had higher levels of both isoniazid resistance (9.5%) and multidrug resistance (4.1%).

ESTIMATE OF HIV CO-INFECTION
In 1998 113 (3.3%) adult patients (16–54 years) with tuberculosis were co-infected with HIV compared with 62 (2.2%) in 1993. In London the proportion was higher and had also increased (5.4% in 1998; 3.3% in 1993). Most co-infected patients were from the black African or white ethnic groups. Detailed results of the overlap between tuberculosis and HIV infection will be reported separately.

Discussion
The number of reported cases of tuberculosis in England and Wales increased by 21% in the 10 years between 1988 and 1998. The increase occurred primarily in London where the number of patients with tuberculosis rose by 71% over this period. In 1998 the rates of disease were highest in the black African ethnic group, remained high but were decreasing gradually in the ISC population, had increased in the Chinese ethnic group, and were very low and continuing to decline in the white population. More than half of all patients with tuberculosis were born outside the UK and the year of entry was known for more than 75% of these. As in previous surveys, rates of disease remained particularly high in those who had recently arrived (between 1994 and 1998) from parts of the world with a high prevalence of tuberculosis. The number of patients born in the UK has continued to decline; this was reflected by continued reductions in rates both in the white population and in those of ISC origin when standardised for age, place of birth, and year of entry. Although greater than in 1993, the proportion of adult patients with tuberculosis co-infected with HIV was low overall.
The methodology used in the 1998 survey differed from previous surveys. A district coordinator was responsible for collecting survey forms in each district. Cases were ascertained throughout the year and all cases known to the district coordinators, whether formally notified or not, were eligible for inclusion in the survey. These differences may have contributed to higher ascertainment of cases compared with previous surveys. The increase in the number of patients between the 1988 and 1998 surveys (21%), however, is comparable to the increase in formally notified cases (18%) for the same period. Although it is not possible to quantify the extent of increased ascertainment in the 1998 survey, its effect is likely to have been at least partly counterbalanced by the exclusion of some formally notified patients for whom a survey form could not be obtained. This is strengthened by the fact that the number of survey forms obtained was very close to the best estimate of cases for 1998 provided by district coordinators.

In earlier surveys most analyses were of new (previously untreated) cases. As difficulties exist in distinguishing with certainty those who have not had prior treatment, in this survey (unless otherwise stated) all cases were analysed together, whether or not previously treated. For comparison, data from the 1988 and 1993 surveys were re-analysed to include all cases, regardless of prior treatment status. As these surveys collected data on all age groups for only 6 months, the data were multiplied by scaling factors to permit comparisons of annual figures. Numbers and rates in this paper may therefore be different from those published previously.

As in previous surveys, some patients who were started on treatment and notified as having tuberculosis were not subsequently de-notified when the diagnosis of a non-tuberculosis disease was made. De-notification remains important, both for local purposes (contact tracing) as well as for local and national surveillance. Mechanisms to facilitate reporting a change of diagnosis have been incorporated into enhanced tuberculosis surveillance.

In some developed countries the reversal of the decline in the number of reported patients with tuberculosis has been partly attributed to extra cases as a result of an increasing number of patients born in high prevalence countries, the HIV epidemic, poverty, and an ageing population. In Europe 11 of the 15 countries providing information to the EuroTB scheme in 1997 reported that 35% of their foreign born tuberculosis patients were born in Africa. Much of the increase in tuberculosis in England and Wales in the last 10 years was due to an increase in the number of patients who had been born abroad. Recent trends in migration into the UK may have contributed to the increased number of cases among new immigrants. The population of the black/African ethnic group approximately trebled from 1988 to 1998, while the ISC population increased by about one third with most of this increase occurring between 1988 and 1993. The populations of white, black Caribbean, and Chinese ethnic groups in England and Wales have increased by less than 6% over the same period (Labour Force Survey population data from 1988, 1993 and 1998).

The geographical pattern of tuberculosis in England and Wales reflects the distribution of some high risk population subgroups. The large number of immigrants, especially recent immigrants, in London has contributed to the substantial increases observed in the capital. In addition, the tuberculosis/HIV co-infection rate increased in the 1990s in London (PHLS CDSC, personal communication). Although the number of adult patients with tuberculosis infected with HIV in England and Wales nearly doubled between 1993 and 1998, this represented only 9% of the national increase in tuberculosis during this period. As information on the socioeconomic status of patients was not collected in this survey, no light can be shed on the effect of poverty on the occurrence of tuberculosis. Further investigation of this issue would be possible using postcodes and indices of deprivation. Despite higher rates of tuberculosis in older people, and an ageing population in Britain, the increase in patients over 65 years of age accounted for less than 10% of the increase in England and Wales since 1988 (data not shown).

As in earlier national surveys, most disease in 1998 was pulmonary in origin although the proportion of patients with a positive sputum smear in 1998 (27%) was lower than in 1993 (35%). Patients in the white ethnic group continued to contribute the largest proportion (54%) of potentially infectious cases (sputum smear positive) in the community. Prompt treatment of such patients, as well as identification and screening of their contacts, must remain an integral part of tuberculosis control to reduce transmission of infection in England and Wales.

If resources to control tuberculosis in England and Wales are to be used efficiently, they must be focused on the groups at highest risk of the disease such as new entrants to Britain from countries with a high prevalence of tuberculosis. Screening of new immigrants has been found to identify cases of active tuberculosis but the current system is weak. Strategies need to be devised to prevent the occurrence of disease in new immigrants more effectively, and resources need to be carried out to determine the factors underlying the very high rates in new immigrants and the effectiveness of intervention measures for this group. Not only should treatment services be capable of providing therapy for patients and prophylaxis for contacts, but they should also be provided in ways best suited to the needs of the population mix in the local community.

This was the last in a series of five-yearly national tuberculosis surveys beginning in 1978, which have highlighted rapid changes in the epidemiology of tuberculosis in England and Wales. The implementation of continuous enhanced surveillance of all cases of tuberculosis from 1999 will provide a detailed and accurate picture of tuberculosis in England and Wales.
rate picture of the occurrence of tuberculosis in a more timely way so that prevention and control measures can be adjusted to be more effective, while making the most efficient use of resources.

The authors would like to thank the clinicians, tuberculosis and chest clinic nurses, microbiologists and all other hospital or clinic staff who reported cases to the survey. We are grateful to all district coordinators and consultants in Communicable Disease Control for collating reports and chasing up missing data, and to the staff of the CIBS Regional Epidemiology Units for their support. We would like to thank André Charlett for statistical advice, and all other members of the survey Steering Committee for their advice and support (Corry van den Bosch, Ian Campbell, Mike Catchpole, Peter Christie, Jeremy Hawker, Owen McCarthy, Bill Smith, Neena Stewart, Tony Swan, Paul Van Buynder and Brian Watt). We are indebted to the survey committee for their advice and support (Corry van den Bosch, Ian Campbell, Mike Catchpole, Peter Christie, Jeremy Hawker, Owen McCarthy, Bill Smith, Neena Stewart, Tony Swan, Paul Van Buynder and Brian Watt). We are indebted to the survey committee for their advice and support (Corry van den Bosch, Ian Campbell, Mike Catchpole, Peter Christie, Jeremy Hawker, Owen McCarthy, Bill Smith, Neena Stewart, Tony Swan, Paul Van Buynder and Brian Watt).

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Case report

Method for manipulating peak flow measurements producing falsely raised readings

S Ross, D P Cochran

Abstract
Methods by which patients can artificially produce raised peak flow measurements have been described. We recently observed a patient manipulating the peak flow meter in a way that had not been described before. A study was therefore undertaken to determine if this technique could repeatedly produce clinically significant changes in peak flow readings. Fifteen adults, using a mini-Wright peak flow meter, made five measurements using the correct technique followed by five manipulated measurements under observation. Significant increases in peak flow measurements were observed in 14 of the 15 subjects. The mean increase in peak flow rate using the incorrect technique was 56% (range –4% to 86%). Clinicians should be aware that patients might employ this technique to manipulate measurements which could have consequences for management. (Thorax 2001;56:500–501)

Keywords: peak flow measurements; technique; false results

Peak flow measurement is used in asthma management. We are aware that peak flow measurements can be artificially increased by spitting or coughing. We recently observed a method of manipulating peak flow measurement which has not been previously described.

Table 1 Results of peak flow measurements

<table>
<thead>
<tr>
<th>Subject</th>
<th>Mean of 5 standard attempts</th>
<th>Mean of 5 manipulated attempts</th>
<th>Relative increase (%)</th>
<th>Best of 5 standard attempts</th>
<th>Best of 5 manipulated attempts</th>
<th>Relative increase (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>509</td>
<td>688</td>
<td>35</td>
<td>520</td>
<td>800*</td>
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<td>2</td>
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*These subjects produced several peak flow measurements beyond 800 (top of the scale) which were recorded as 800 for the purpose of calculating the mean recording and relative increase.

Figure 1 Position of index finger when retarding movement of peak flow indicator.

Case report
A 13 year old asthmatic boy was seen as an outpatient. He was prone to recurrent acute attacks of asthma and had four hospital admissions in the previous 3 months. The patient was asked to perform a peak flow measurement. During the manoeuvre his movements appeared slightly awkward and, when asked to explain, he admitted that he had discovered that, if he retarded the movement of the peak flow indicator with his index finger at the beginning of the manoeuvre for a fraction of a second after starting to exhale (fig 1), he could increase the reading. He also explained that, if he moved vigorously during the procedure, it appeared that he was giving maximum effort to performing the measurement and

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this made it difficult for the doctor to detect what he was doing with his finger.

A study was therefore undertaken to determine whether this technique could repeatedly produce clinically significant changes in peak flow readings.

Methods
Fifteen adults, one of whom had asthma, were asked to make five peak flow measurements in the standard way followed by a further five peak flow measurements by retarding the movement of the peak flow indicator as described in the case report, after an initial practice. All measurements were carried out using a mini-Wright peak flow meter.

Results
The results are shown in table 1. Both the mean and the best of five readings for the two techniques were recorded. It can be seen that altering the technique of peak flow measurement (as described in the case study) produced significant increases in peak flow rate in 14 of the 15 subjects. Using the mean of the five attempts, a mean increase of 28% (range –2% to 66%) was achieved. When the highest of the five attempts was taken as representative, the mean increase was 56% (range –4% to 85%).

Discussion
Peak flow recording is important in asthma management1 and these measurements are used to inform decisions about the need for referral to hospital, admission, and timing of discharge. Patients dislike being in hospital and may be tempted to manipulate peak flow measurements in order to give falsely raised readings that could influence their management. For example, patients may have a true peak flow <50% of their personal best but, by falsely increasing the peak flow measurement by 28%, management could be quite different. Clinicians need to be aware of this and of other manoeuvres that lead to falsely raised readings. We would emphasise that it is possible to interfere with the movement of the peak flow indicator with subtilty and this would not be readily noticed unless the clinician is watching closely. Asking patients to “cradle” the peak flow meter with a hand underneath the device is likely to prevent this problem.

3 Connolly CK. Falsely high PEF/low due to acceleration in the mouth. _Bmj_ 1987;294:285.

LETTERS TO THE EDITOR

CFC transition

Dr Everard wrote such a wide ranging polemic against inhaled therapy that it is difficult to know where to start. While the article contains a good deal of sense, it also contains a number of inaccuracies and misperceptions. I think the record needs to be put straight.

Firstly, Dr Everard says that inhaled insulin will be in use in 2001. However, as far as I am aware, no application for delivery of insulin as an aerosol has yet been submitted to the FDA. New devices for the delivery of insulin are under trial, but the earliest they are likely to get to the market is 2002. They are likely to be relatively complex devices costing perhaps 100 times more than the current price for a salbutamol metered dose inhaler (MDI).

Secondly, Dr Everard berates the pharmaceutical industry for its efforts in moving away from chlorofluorocarbon (CFC) MDIs. In 1996, when the Montreal protocol came into force, there was a real risk that MDIs for use in asthma/COPD would no longer be available. The industry started 10 years ago to try to reformulate MDIs but the technical challenge has been enormous and complicated by intellectual property issues. Only now, more than a decade later, are a sufficient number of CFC free MDIs coming to the market so that the transition can be completed over the next 2–4 years. This has cost a huge amount of money. But where is the evidence that they prevented new chemical entities for asthma coming to the market? If such clinically efficacious compounds were available, then I am certain that they would have been commercially exploited.

Thirdly, Dr Everard is correct in stating that the MDI in some ways is a less than ideal inhaled device. The new hydrofluorocarbon (HFC) beclomethasone product (Qvar, 3M Pharmaceuticals) has a much smaller particle size, not by design but by necessity. This results in something like 5–6 times more drug being deposited peripherally in the lung but only twice the therapeutic effect compared with the MDI it replaces. This product has given a big “wake up call” to the industry and we should all be asking which particle size really is ideal, particularly for inhaled steroids. Is it different, for example, for beclomethasone versus budesonide versus fluticasone? I am also unaware of any data that monodispersed aerosols are more efficacious or safer than aerosols with a more broad range of particle size.

Fourthly, Dr Everard mixes in with the discussion of technology a rather paternalistic view of patient compliance. If only patients would do as they are told and take their medication all would be well. Does anyone ever complete a course of antibiotics? We badly need more information about why patients do not adhere to medication regimes and what information they need about their disease and the treatments used. But isn’t this true for all therapies? Dose counters to improve compliance might be valuable for MDIs—but where is the evidence that they are cost effective, or even that doctors or patients want them?

I am firmly convinced that the MDI, along with the dry powder inhaler (DPI), is here to stay. They are remarkably cheap, instantly efficacious as bronchodilators, and have an excellent therapeutic ratio for inhaled steroids for most asthmatics. Many patients can use up to 2000 doses per year without their inhaler letting them down, and perhaps it is not surprising that they become reliant on them. Certainly they can be improved upon. Novel devices using aqueous and hydrocarbon propellants are on their way. But if Dr Everard sees any viable and cost effective alternatives, then let us hear about them.

A WOODCOCK
Department of Respiratory Medicine, Wythenshawe Hospital, Manchester M23 9LT, UK


Mark Everard’s superb leading article railing against the numerous inadequacies of inhaled therapy delivered by pMDIs is to be applauded, but ultimately is an unsatisfying tract since it does not point to any solutions applicable in the here and now. The single most important advance in the treatment of asthma at the beginning of the 21st century would be to wean ourselves—and by that I mean the UK since the process is already well advanced in continental Europe—from the use of these archaic impediments to treatment. Everard is right that the regulatory authorities bear a heavy burden of guilt by requiring devices which result in greater lung deposition to undergo much more extensive, and therefore costly, investigation than “me too” devices mimicking the appalling performance of pMDIs. I have witnessed the “detuning” of novel inhalers that he refers to in order to limbo under this bureaucratic hurdle. Removing the equivalence straight jacket for inhaler devices would, at a stroke, allow currently available devices to be used to their maximum and not merely potential.

Why are the British so hooked on the inadequate and obsolete pMDI? It is simply that...
the currently available alternative—dry powder inhalers—are perceived to be more costly. This view has been led by the Department of Health who have calculated that swapping device for device would cost them a great deal of money. This, however, is the accountant's view. Because dry powder inhalers are more efficient at drug delivery, the "Fairy Liquid argument" holds and the cost of treatment rather than the cost of the device should be used in their calculations. Of course, such back of the envelope packer arithmetic should be banished by the new god of evidence based medicine. So what is the evidence on cost? Howarth et al have performed an audit of patients who have been transferred to dry powder inhalers because they were unable to use pMDIs. The cost of treatment with inhaled steroids was actually lower with dry powder inhalers. The only randomised controlled trial using the over-priced Turbohaler came to a similar conclusion. 3 Thus, what little evidence that is available suggests that prescribing even premium priced dry powder inhalers for the delivery of inhaled steroids may be cheaper than using pMDIs. There can be no argument that there is a cost impediment with the more recent introduction of generic dry powder inhalers which are competitive even on a device for device basis.

Everard rightly points out that the issues of compliance, competence, and contrivance are closely interlinked. What is rarely taken into account in the equation is the patient perspective. We have recently analysed the results of three separate studies comparing the market leading pMDI with the Clickhaler in terms of patient preference in 118 paediatric patients using the AAD system and have unsurprisingly found that 20% of patients were unable to use pMDIs. 4 The cost of treatment with inhaled steroids was actually lower with dry powder inhalers. The only randomised controlled trial using the over-priced Turbohaler came to a similar conclusion. Thus, what little evidence that is available suggests that prescribing even premium priced dry powder inhalers for the delivery of inhaled steroids may be cheaper than using pMDIs. There can be no argument that there is a cost impediment with the more recent introduction of generic dry powder inhalers which are competitive even on a device for device basis.

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The third C—contrivance—will be tackled during a formal structured interview or on the back of the cigarette packet. Because dry powder inhalers are more efficient at drug delivery, the "Fairy Liquid argument" holds and the cost of treatment rather than the cost of the device should be used in their calculations. Of course, such back of the envelope packer arithmetic should be banished by the new god of evidence based medicine. So what is the evidence on cost? Howarth et al have performed an audit of patients who have been transferred to dry powder inhalers because they were unable to use pMDIs. The cost of treatment with inhaled steroids was actually lower with dry powder inhalers. The only randomised controlled trial using the over-priced Turbohaler came to a similar conclusion. Thus, what little evidence that is available suggests that prescribing even premium priced dry powder inhalers for the delivery of inhaled steroids may be cheaper than using pMDIs. There can be no argument that there is a cost impediment with the more recent introduction of generic dry powder inhalers which are competitive even on a device for device basis.

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treatment, but their effectiveness was limited by tachyphylaxis.

The children who were treated with SCG when it was introduced experienced less the currently available alternative—dry powder inhalers—are perceived to be more costly, exercise induced asthma and were able to participate in playground games, often for the first time. In addition, fewer colds resulted in wheezing and exacerbations were shorter. The parents and physicians of these children were delighted with this safe remedy which was effective for all but the most severe forms of the disease. The most popular formulation was a mixture of SCG with isoprorenal (Intal compound). This combined the immediate bronchodilator effect of a rapidly acting β-adrenergic drug of extremely short duration with protection from antigen challenges and exacerbation of asthma which lasted for about 6 hours.

Salmeterol was introduced in 1969, only 2 years after SCG, and had the same effect in providing both relief and protection. As soon as salbutamol, permitted 6-hourly, became the rescue bronchodilator of choice, it became difficult to demonstrate any clinically relevant benefit attributable solely to SCG in patients who were not dependent on oral steroids. Inhaled beclomethasone was introduced soon afterwards and this enabled even more asthmatics who were dependent on oral steroids to discontinue them. Beclomethasone does not have the unpleasant taste that makes SCG disagreeable to use, and suppresses more components of inflammation than SCG. Furthermore, beclomethasone rapidly reduces symptoms more effectively for the same effect on daytime FEV₁, and morning peak flow, possibly by reducing the sensation of inflammation in the airways. By the time it was recognised that long term administration of higher doses of inhaled steroids produced significant side effects in children and adults, long acting bronchodilators had been developed and proved to be useful in combination with moderate doses of inhaled steroids. If possible use of cromoglycate, with its proven safety record, was not investigated in this context. There are no long term studies which show whether SCG played any part in the improved outcomes in childhood asthma between 1970 and 1980, or whether these are entirely the consequence of inhaled steroid use.

In a lucid contemporary account, one of the first investigators to test SCG described how he and his colleagues were disappointed to be unable to show any major effect of SCG on FEV₁, in spite of a dramatic symptomatic and steroid sparing effect. At this time asthma was defined as variable or reversible airflow obstruction, and it was recognised that patients would judge the control of their condition by the frequency of regular, unexpected, or nocturnal attacks rather than by their best lung function. The value of frequent observation during the day was demonstrated by Chai et al in 1968 and accepted in late 1970s. The effects of SCG in children with previously regarded paroxysmal nocturnal dyspnoea were assessed in a network of hospitals and it was demonstrated that sodium cromoglycate is not effective for all but the most severe forms of exercise induced asthma which lasted for 3–5 years. Consequently, they were able to provide effective relief from symptoms and reduce the need for salbutamol. The most popular formulation was a mixture of SCG with isoprorenal (Intal compound). This combined the immediate bronchodilator effect of a rapidly acting β-adrenergic drug of extremely short duration with protection from antigen challenges and exacerbation of asthma which lasted for about 6 hours.

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helms@abdn.ac.uk

1 Helms PJ. Inhaled disodium cromoglycate as maintenance therapy for childhood asthma: time to consign to history? Thorax 2000;55:858–68.

AUTHORS’ REPLY Lazlo provides an historical perspective on the passing cromoglycate era in which he describes the dire state of asthma management, particularly in children, at the time that sodium cromoglycate (SCG) was introduced. He also points out that, soon after SCG was introduced, inhaled isoprenaline became available followed by the selective β₂-agonist salbutamol. It is therefore understandable that the effectiveness of sodium cromoglycate compared with placebo, not with other active treatments. As a consequence, our findings may be a guidance not only to today’s physicians but also to future generations with other therapeutic alternatives.

AUTHORS’ REPLY We appreciate Dr Laslo’s eloquent historic overview of asthma treatment and agree that a therapy should be considered within the range of available options. This range of options may change over time. However, our systematic review addressed the effectiveness of sodium cromoglycate compared with placebo, not with other active treatments. As a consequence, our findings may be a guidance not only to today’s physicians but also to future generations with other therapeutic alternatives.

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BOOK REVIEW


Margaret Branthwaite, formerly a distinguished consultant physician at the Royal Brompton hospital, retired early from her post to write a book which is therefore uniquely well qualified to write a book explaining the law as it impinges upon medical practice for doctors. She also writes with admirable conciseness and clarity and the book is a pleasure to read.

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Not many years ago most doctors regarded interaction with the law with a combination of distaste and fear, and I remember being advised by a very senior colleague to eschew contact with lawyers as far as possible—advice which I am glad I did not take. Nowadays doctors are increasingly aware of the need to have at least a passing acquaintance with the law, both in order to help their patients and to protect themselves against potential litigation for malpractice. The former consideration applies particularly in respiratory medicine where disease of occupational origin is common and patients look to their respiratory physician for advice about seeking compensation.

This book covers all the important interfaces between medicine and the law. There is a clear exposition of the legal basis for claims for damages arising from clinical practice. The criteria for a finding of negligence and the basis for the calculation of damages are explained. The legal steps involved in pursuing a claim for damages for personal injury (a term which includes occupational lung disease) are set out. The important changes to the requirements of expert witnesses introduced as the Woolf reforms last year are described.

Other areas in which the law impacts upon medicine are considered including the concept of informed consent for both adults and children. Complaints, whistle blowing, and disciplinary proceedings are discussed briefly. There is a useful explanation of the work of the Coroner’s court and the doctor’s role in its proceedings. Finally, there is a stimulating discussion of issues surrounding allegations of homicide and manslaughter against doctors and the potential criminal liability for end of life decisions.

I would strongly recommend this modestly priced book to all readers who are at all interested in the interface between the law and medicine, and to those who may be less interested but feel they should be better informed.—RR

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### NOTICE

**Respiratory Medicine**

A conference on Respiratory Medicine will be held at the Royal College of Physicians of Edinburgh on 26 October 2001. For further information contact Ms Eileen Strawn, Symposium Coordinator. Telephone 0131 225 7324. Fax 0131 220 4393. Email: e.strawn@rcpe.ac.uk. Website: www.rcpe.ac.uk.

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### CORRECTION

**TB at the end of the 20th century**

In the paper entitled “Tuberculosis at the end of the 20th century in England and Wales: results of a national survey in 1998” by A M C Rose et al which appeared on pp 173–9 of the March 2001 issue of Thorax, the data for the South East region were omitted from table 3 on page 176 of the paper. The correct version of the table is shown here. The authors apologise for this omission.

### Table 3 Number of patients with tuberculosis and rate of disease by NHS region and district health authorities in England and Wales with more than 100 tuberculosis patients and/or a rate of >25 per 100 000 in 1998

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<th>Region*</th>
<th>No of cases</th>
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<td>Trent</td>
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<td>8.53 to 8.82</td>
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<td>Brent &amp; Harrow</td>
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<td>Bradford</td>
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<td>23.7 to 25.5</td>
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<td>Leicestershire</td>
<td>172</td>
<td>18.7</td>
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*April 1999 boundaries.

**Rate per 100 000 population.