



Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active.

Acute bronchitis in the community: clinical features, infective factors, changes in pulmonary function and bronchial reactivity to histamine

D. A. R. BOLDY, S. J. SKIDMORE* AND J. G. AYRES

Department of Respiratory Medicine, East Birmingham Hospital, Bordesley Green East, Birmingham B9 5ST, and *Regional Virus Laboratory, East Birmingham Hospital, Bordesley Green East, Birmingham B9 5ST, U.K.

A descriptive study of acute bronchitis in patients without pre-existing pulmonary disease was undertaken in the community during the winter months of 1986-87. Forty-two episodes were investigated in 40 individuals. The cardinal symptom was the acute onset of cough (100%), usually productive (90%). Wheezing was noted by 62% of patients, but heard on auscultation in only 31%. A potential pathogen was isolated in 29% of cases with a virus (eight cases) being identified more frequently than either *Mycoplasma pneumoniae* (three cases) or a bacterium (three cases). The acute illness was associated with significant reductions in forced expired volume in 1 second ($P < 0.02$) and peak expiratory flow ($P < 0.001$) but not forced vital capacity compared to 6 weeks later. Ten of the 27 (37%) patients who had a histamine challenge test performed at 6 weeks had a PD_{20} of $< 7.8 \mu\text{mol}$ histamine. Thirty-nine episodes (93%) were treated with antibiotics by the general practitioner, the clinical course being unremarkable apart from one patient who developed a lingular pneumonia despite antibiotic therapy. Further studies are required to assess whether acute bronchitis causes an acute increase in bronchial hyperresponsiveness and whether either antibiotics or inhaled bronchodilators or anti-inflammatory therapy has a useful role in the management of this predominantly viral illness.

Introduction

Acute bronchitis is a condition diagnosed commonly in general practice, particularly in the winter months, reaching rates of approximately 150 cases per 100 000 population per week (1). It is presumed that most episodes of acute bronchitis are viral in origin, although previous work, with one exception (2) has studied children (3-5) or patients with underlying pulmonary disease (6-8) and has shown only modest rates of virus identification and very low rates of bacterial isolation. There is also the diagnostic problem of identifying whether an individual patient has simple acute bronchitis: in many patients, particularly children, the diagnosis of asthma is missed (9). It is believed that the acute effect of such an illness on pulmonary function is minimal and does not contribute to the development of chronic airflow obstruction (10). However, upper respiratory tract infections may produce changes in pulmonary function as indicated by a reduction in gas transfer (11), increase in closing volumes in smokers (12), and a fall in dynamic compliance (13). In addition, uncomplicated influenza has been shown to produce a transient fall in forced expired volume in 1 second and arterial oxygen concentration (14). The

Received 24 November 1989 and accepted 11 May 1990.

effect of viral infections on bronchial hyperreactivity is uncertain (15-19) and epidemiological studies in the community give conflicting results with respect to respiratory tract infections and bronchial hyperreactivity (20,21). In view of the continuing apparent difficulty in separating acute bronchitis from asthma, this descriptive study was undertaken to examine the clinical, microbiological and pulmonary function aspects of the illness being diagnosed as acute bronchitis in patients with no pre-existing pulmonary disease.

Methods

Patients diagnosed as having acute bronchitis by the general practitioner (GP) were studied. No criteria for the diagnosis of acute bronchitis were given to the GPs, except that patients with previously diagnosed pulmonary disease were excluded from the study. The study was undertaken in a local general practice with four partners and a practice population of 7700. Sequential patients were recruited into the study between September 1986 and March 1987 and were studied on presentation and at 2 and 6 weeks. On presentation, demographic data were collected and symptoms were recorded using questions from the MRC/

PHLS study (22). Three methods were used to determine potential pathogens.

NASOPHARYNGEAL WASHINGS (NPW)

NPW were obtained for viral culture. A size 10 physiotherapy suction catheter was cut 5" from the distal end and a mucus trap inserted between the two cut ends of the catheter. The proximal end of the catheter was attached to a mechanical pump (Vacuaide Model 721, DeVilbiss Health Care). With the patient lying on his/her back and the neck extended slightly, the distal end of the catheter was introduced through the nares into the posterior naso-pharynx. Suction was commenced as 5 ml of phosphate buffered saline was introduced into the opposite nares to aid flow back to the mucus trap and to keep the specimen moist until transfer to the laboratory. The washings were inoculated onto four cell lines: monkey kidney, human embryo lung, HEP-2 and MRC-5. The cultures were incubated, rolling at 33°C for a minimum of 2 weeks.

VIRAL SEROLOGY

Serum was taken on presentation and 2 weeks later for paired complement fixation tests for antibodies to Influenza types A and B, respiratory syncytial virus (RSV), adenovirus and *Mycoplasma pneumoniae*. A four fold rise in antibody titre was considered indicative of a recent infection. Some patients had high titres of *M. pneumoniae* antibodies at both visits and identification of recent infection was sought by demonstration of IgM antibody in the initial samples (23).

SPUTUM CULTURE

Sputum samples were obtained from patients who were able to produce a specimen during the initial consultation and were sent for routine bacteriological culture.

Venous blood was taken for full blood count and α IFN on presentation only, and serum IgE at both presentation and 6 weeks. α IFN was measured using a radioimmunoassay method (Boots Celltech); levels $>1 \text{ IU ml}^{-1}$ were regarded as suggestive of infection (24). Specimens for IgE were separated, the sera frozen at -20°C and measured in batches at the end of the study by the PRIST technique (Pharmacia).

On presentation, skin prick testing was performed to cat fur, *Dermatophagoides pteronyssinus*, grass pollens and a saline control (Bencard, Brentford). Weal diameters were measured at 15 min as the mean of two transverse diameters, 90° to each other, one of which was the maximum measurable weal diameter. Any response $>3 \text{ mm}$ diameter with a negative control was regarded as positive.

PULMONARY FUNCTION TESTS

Spirometry and peak expiratory flow were performed on presentation and at 2 weeks and 6 weeks. Forced expired volume in 1 second (FEV_1) and the forced vital capacity (FVC) were recorded using the same dry bellows spirometer (Vitalograph) throughout the study; three attempts reproducible to within 200 ml were made and the best FEV_1 and FVC recorded. Peak expiratory flow (PEF) was measured using a Wright mini peak flow meter and the best of three attempts recorded. At the first two visits, the response to inhaled bronchodilators was assessed by repeating all measurements 15 min after inhalation of 1.5 mg terbutaline through a spacer device (Nebuhaler, Astra Ltd).

HISTAMINE CHALLENGE TESTING

At the third visit (6 weeks), histamine challenge test (HCT) was performed using the Yan method (25), until either a 20% fall in the post saline FEV_1 was recorded or the maximum dose of histamine ($7.8 \mu\text{mol}$) was administered. The $\text{PD}_{20} \text{FEV}_1$ was used in analysis. A positive HCT was regarded as one in which the $\text{PD}_{20} \text{FEV}_1$ was $<7.8 \mu\text{mol}$ (26). HCTs were not performed in patients who were unable to produce FEV_1 values reproducible to within 200 ml prior to HCT. Patients were asked to refrain from taking any bronchodilator medication from the evening before, and requested not to smoke in the 2 h before the test. After completion of the test, 1.5 mg of terbutaline was administered and spirometry repeated until the FEV_1 was $>95\%$ of the pre-test value. Those patients in whom HCT was performed at 6 weeks were asked to return 1 year later for repeat spirometry and HCT.

Predicted values for pulmonary function tests in children were calculated from Cotes (27) and for adults from ECCS (28); for patients aged 16–25 the adult reference table was used. Standardized residual (SR) values were calculated for each adult individual for each visit (29).

ANALYSIS

Serum IgE data were log transformed prior to analysis. Student's *t*-test on paired and unpaired samples was used where appropriate in analysis and the Chi-squared test and Fishers exact probability test were used for 2×2 contingency tables.

CONSENT

Written informed consent was obtained from each patient and the study was passed by the local Ethical Committee.

Table 1 Demographic details of the 42 patients investigated for acute bronchitis

	Females (n=29)	Males (n=13)	All patients (n=42)
Age in years [mean (SD)]	35.8 (15.6)	41.5 (17.3)	37.5 (16.5)
Age range in years	12-72	15-73	12-73
Current smokers patients ≥ 16 years, n=40 [n(%)]	13(46)	7(58)	20(50)
Family history of atopy n=40 [n(%)]	15(56)	3(23)	18(45)
Positive skin prick test(s), n=40 [n(%)]	8(30)	7(54)	15(37.5)

Results

Initially, patients of all ages were entered into the study at both morning and evening surgeries. However, because of incompleteness of data (no pulmonary function tests in patients under 12-years-old, and no presentation pulmonary function for evening surgery patients), the paper describes the results of patients aged 12 years or over referred for study from the morning surgery only.

During the study period, forty-two episodes were investigated in 40 such patients. Thirty patients attended both follow-up appointments, four attended one only and 6 did not attend either follow-up appointments, an overall re-attendance rate of 81%.

DEMOGRAPHIC DATA

Demographic data are shown in Table 1. Female patients predominated in a ratio of 2.2:1 and the mean (SD) age of the whole group was 37.5 (16.5) years (range from 12-73 years). Twenty episodes (48%) occurred in current cigarette smokers, including two patients referred twice, one being a current smoker at both visits and one a current smoker at her second visit only. There was a family history of atopic disease in 18 out of 40 cases (47%)

SYMPTOMS AND SIGNS

Acute onset of cough was reported by all patients, being productive in 90% of cases. (See Fig. 1 for a full listing of symptoms and signs.) Wheezing was noted by 62% of patients, coryza by 71% and sore throat by 57%. A variety of less specific symptoms were also reported, notably sweating (60%) and headache (50%). Symptoms were reported with equal frequency by males and females. Clinical signs were recorded by the GP in 18 cases (43%), wheeze being noted in 13 (31%) and crackles in ten (24%). Delay in presentation to the GP after the onset of symptoms varied from 2-35 days [median 7 days; mean (SD) 10 (9) days] (Figure 2). Twenty six patients (62%) had had symptoms for

less than 1 week before presentation (Fig. 2). Current smokers had symptoms for a mean of 10.1 days compared to 8.7 days in nonsmokers and exsmokers (not significant). Patients between the ages of 20 and 60 were less likely to present to their GP within 5 days of the onset of the illness than younger or older patients ($P < 0.02$).

VIROLOGY AND BACTERIOLOGY

The virology and bacteriology of the patients investigated are given in Table 2. NPW was generally well tolerated. A virus was isolated in seven out of 38 (18%) patients in whom NPW was performed on presentation, 4 rhinoviruses and three Influenza A. Four of the 13 (31%) in whom NPW was performed within 5 days of the onset of symptoms, were positive compared to three of the 25 (12%) performed after 5 days, but this was not a significant difference. Serology confirmed the three Influenza A infections, and also detected one adenovirus (NPW negative). Three *Mycoplasma pneumoniae* infections were identified, two in adolescents.

Sputum specimens were obtained from 13 individuals yielding three positive cultures; *Branhamella catarrhalis* in a 26-year-old woman from whom a rhinovirus was also grown from the NPW, *Haemophilus influenzae* from a 46-year-old woman with insulin dependent diabetes mellitus and *Streptococcus pneumoniae* in a 23-year-old female nonsmoker with Influenza A on culture and serology. Overall, a potential pathogen was identified in 12 out of 42 episodes (29%) who had had NPW/sputum and/or paired serology performed. Only one case progressed to a more substantial illness; a 46-year-old woman with a rhinovirus isolated on presentation was admitted to hospital 14 days later with a lingular pneumonia, for which no causative organism was demonstrated. She made an uneventful recovery.

Serum α IFN levels were measured on presentation in all episodes. Only one patient, with an Influenza A

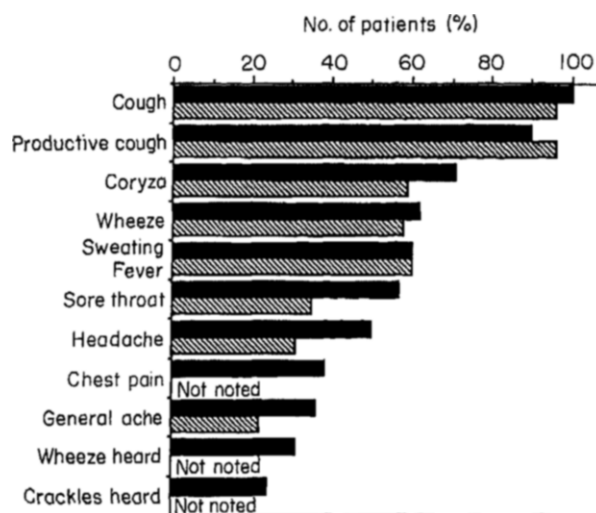


Fig. 1 Clinical features of the patients with acute bronchitis in this study (■, $n=42$) and in the MRC/PHLS study (▨, $n=198$) (2) (198 patients). All results are expressed as percentages.

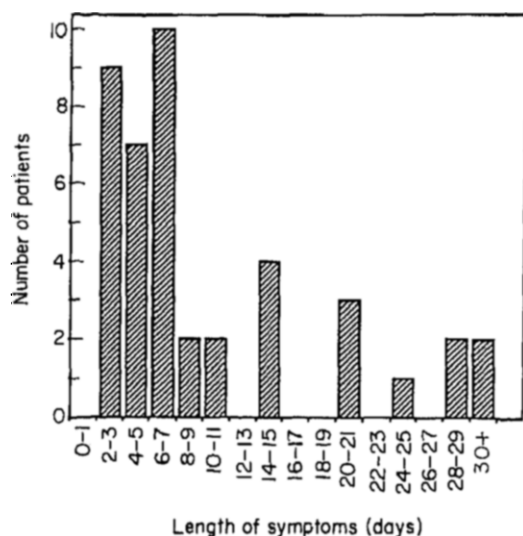


Fig. 2 Length of symptoms (in days) recorded at presentation in 42 patients investigated for acute bronchitis.

infection, had an elevated level at 4 IU ml^{-1} (Patient 5, Table 2).

OTHER INVESTIGATIONS

The total white blood cell count (WBCC) was above the reference range ($> 10.8 \times 10^9 \text{ l}^{-1}$) in eight out of 37 cases tested (22%). Six patients had mildly elevated total white blood counts (range $10.9\text{--}11.9 \times 10^9 \text{ l}^{-1}$), while two patients showed more substantial elevations

(14.0 and $19.4 \times 10^9 \text{ l}^{-1}$). However, a potential pathogen was demonstrated in only one of these patients (total WBCC $11.1 \times 10^9 \text{ l}^{-1}$), (Patient 4, Table 2).

A positive skin prick test to at least one allergen was observed in 15 out of 40 patients (37.5%). Patients under 20 years of age were significantly more likely to have a positive skin prick test result (6/6 compared to 9/34; $P=0.0026$).

One patient had grossly elevated IgE levels both on presentation [serum IgE $13\,575$ ($\log \text{ IgE } 4.133 \text{ KU l}^{-1}$)] and at 6 weeks [serum IgE $10\,385$ ($\log \text{ IgE } 4.02 \text{ KU l}^{-1}$)], and his data are not included further in the analysis. In the remaining cases there was no statistically significant difference between the value [$\log \text{ IgE}(\text{SD})$] on presentation [$1.44(0.66)$] and at 6 weeks [$1.51(0.64)$]. Current smokers had similar IgE levels to nonsmokers [$\log \text{ IgE } 1.48$ compared to 1.53].

TREATMENT

Thirty-nine of the 42 (93%) episodes were treated with a course of antibiotics, those prescribed being erythromycin (11), amoxycillin plus clavulanic acid (7), a drug trial antibiotic (7), tetracycline (5), cefaclor (4), cotrimoxazole (3), amoxycillin (2), and penicillin V (1) (one patient received two antibiotics).

PULMONARY FUNCTION TESTS

Results of the pulmonary function tests are given in Table 3. The mean FEV_1 rose from 2.71 (87% predicted) on presentation to 2.81 (92% predicted) at 2 weeks ($P<0.01$) and to 2.71 (91% predicted) at 6

Table 2 Details of the 12 cases investigated for acute bronchitis in whom a pathogen was identified

Patient	Sex (M/F)	Age (YRS)	Date of presentation	Length of symptoms	Viral cultures	Serological response	Sputum culture
1	F	16	Sept 1986	28	Negative	<i>M. pneumoniae</i> 1:32 1:128	NP*
2	M	15	Sept 1986	3	Negative	<i>M. pneumoniae</i> 1:64 1:64	NP
3	F	46	Sept 1986	5	Negative	Negative	<i>H. influenzae</i>
4	F	26	Oct 1986	3	Rhinovirus	Negative	<i>B. catarrhalis</i>
5	M	33	Nov 1986	14	Influenza A	Influenza A 1:8 1:64	Negative
6	M	19	Nov 1986	4	Influenza A	Influenza A 1:16 1:64	Negative
7	F	23	Dec 1986	4	Influenza A	Influenza A 1:8 1:64	<i>S. pneumoniae</i>
8	F	39	Jan 1987	21	Negative	<i>M. pneumoniae</i> 1:256 1:256	NP
9	F	72	Jan 1987	3	Rhinovirus	Negative	NP
10	M	56	Jan 1987	14	Negative	Adenovirus 1:16 1:64	NP
11	F	31	Feb 1987	7	Rhinovirus	Negative	NP
12	F	46	Feb 1987	7	Rhinovirus	Negative	NP

*NP=Not performed.

Table 3 Mean FEV₁, FVC and PEF values in 42 patients with acute bronchitis on presentation, 2 weeks and 6 weeks later. Results are expressed as the per cent of the predicted value, and for patients aged 16 or over ($n=40$) the mean standardized residual (SR)

	FEV ₁ (% predicted)	SR _{FEV1}	FVC (% predicted)	SR _{FVC}	PEF (% predicted)	SR _{PEF}
On presentation	86.9	-0.98	90.8	-0.73	86.5	-1.06
2 weeks	91.6***	-0.64***	94.7**	-0.49*	94.1**	-0.40***
6 weeks	91.0**	-0.53**	94.2	-0.52	95.8****	-0.31***

From presentation: * $P<0.05$, ** $P<0.02$, *** $P<0.01$, **** $P<0.001$.

weeks ($P<0.02$ compared to presentation); FVC rose from 3.31 (91%) on presentation to 3.41 (95%) at 2 weeks ($P<0.02$) and to 3.31 (94%) at 6 weeks (not significant) and PEF rose from 384 l min⁻¹ (87%) on presentation to 413 l min⁻¹ (94%) at 2 weeks ($P<0.02$) and to 415 l min⁻¹ (96%) at 6 weeks ($P<0.001$ compared to presentation). Patients in whom a wheeze was heard by the general practitioner on presentation had a significantly lower FEV₁ (mean 79% predicted) than patients without wheeze (mean 90% predicted) ($P<0.05$). Twelve out of 42 patients (29%) had a bronchodilator response > 15% to terbutaline at the initial visit in either the FEV₁ alone (three patients), PEF alone (7 patients), or both FEV₁ and PEF (two patients). Four further patients had a bronchodilator response at the second visit (FEV₁ in two patients, PEF in two patients), making 16 out of 42 (38%) in total.

HISTAMINE CHALLENGE TESTS

Histamine challenge tests were performed on 29 occasions in 27 patients at 6 weeks (two patients presenting twice had two HCT performed) (Table 4). Eleven of the 29 cases (38%) had a PD₂₀ of < 7.8 μmol at 6 weeks. The mean SR FEV₁ at presentation of patients with a positive HCT was significantly lower than the mean SR FEV₁ of patients with a negative HCT (-1.77 vs. -0.80, $P<0.05$) (Fig. 3). A positive HCT at 6 weeks was significantly correlated with a > 15% bronchodilator response in FEV₁ on presentation ($P=0.024$), but with no other variable. HCTs were not performed in 13 patients (nonattenders at 6 weeks, 9; unable to produce reproducible spirometric curves, 2; ischaemic heart disease, 1; refused, 1).

The 29 patients were recalled for repeat HCT a year later. Eighteen patients re-attended (59%), six of

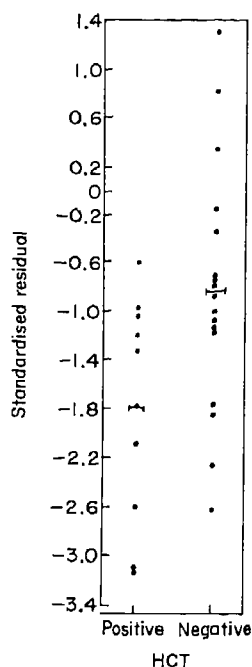


Fig. 3 Standardized residuals (SR) of the forced expired volume in 1 second at presentation in the 27 patients over 16 years of age who had histamine challenge tests (HCT) performed at 6 weeks. HCT status: patients with a positive HCT ($PD_{20} < 7.8 \mu\text{mol}$ histamine) at 6 weeks ($n=11$, mean SR = 1.77); patients with a negative HCT ($PD_{20} > 7.8 \mu\text{mol}$ histamine) at 6 weeks ($n=16$, mean SR = 0.80). The difference between the two groups is significant at the 5% level.

whom originally had a positive HCT. Three remained reactive, and one further patient who was not reactive originally, showed a $PD_{20} FEV_1$ of 5.0, 12 months later. The change in bronchial hyperreactivity between the two visits for all patients was within one doubling dilution except for one patient who had a respiratory tract infection 4 weeks prior to repeat HCT (Patient 6, Table 4) and two patients who had been started on inhaled steroids by the GP during the intervening year (Patients 4 and 9, Table 4), whose PD_{20} s had fallen.

Discussion

Based on the weekly returns data for the years 1976 to 1983, the mean annual attack rate for acute bronchitis is 87 cases per 100 000 population per week (1). Hospital inpatient enquiry data suggests that less than 0.25% of cases of acute bronchitis are admitted to hospital. Thus, acute bronchitis is a common illness managed largely by general practitioners. The weekly returns data on acute bronchitis from the Research

Unit of the Royal College of General Practitioners (RURCGP) demonstrate a consistent pattern with the highest attack rates being seen in winter months (1), in the very young and in the elderly. This age pattern differs from that observed in the present study because we excluded cases of acute bronchiolitis and exacerbations of chronic airflow obstruction (both of which are included in the RURCGP figures) and asthma, a condition often missed and mislabelled 'acute bronchitis' in general practice. In addition, we probably lost cases by allowing the option of other diagnoses such as chest infection. The advantage of this study, however, is that patients were investigated in whom the GP had made a diagnosis of what s/he regarded as acute bronchitis. It is possible, but unlikely, that some patients may have had pneumonia on presentation, since previous work of lower respiratory tract infection in the community (excluding clinical pneumonia) showed new X-ray changes in only 2.3% of patients (30).

A variety of definitions of acute bronchitis have been proposed (31–33). Since ours was a descriptive study, we chose to allow the GPs free rein to determine what they regarded as acute bronchitis. Interestingly, the results of the MRC/PHLS study—where acute bronchitis was defined as 'productive cough and rhonchi' (33)—and our study are strikingly similar (Fig. 1) suggesting that, in this general practice at least, the diagnosis of acute bronchitis is following defined lines.

A potential pathogen was identified in only 30% of cases, despite the short time between sample and inoculation of cultures (less than 3 h), a finding similar to previous studies in adults with asthma (7), acute exacerbations of chronic airflow obstruction (6,8) and children with wheezy bronchitis (3–5). Three factors contributed to this finding. Firstly, where patients were able to determine the duration of symptoms, 62% stated that symptoms had been present for 6 or more days. Since viral shedding declines quickly after the onset of infection, delay in investigation, such as was seen in our study, may be a major reason for failing to identify a viral pathogen. This is further supported by the lack of detectable α IFN in all but one of our patients, α IFN being present only transiently after the initial infection (24). Secondly, serological investigation was performed for a limited number of respiratory viruses, the major omissions being coronaviruses, which usually produce a coryzal illness (34) and parainfluenza viruses for which serology is unreliable. Thirdly, it has been suggested that sputum might be a better source for viral culture than NPW or throat swabs (4). In our study, only 31% were able to produce a sputum specimen at the time of consultation, despite having a history of productive cough. NPW was well

Table 4 Details of the 11 patients with a positive histamine challenge test ($PD_{20}FEV_1 < 7.8 \mu\text{mol}$ histamine) at 6 weeks or 1 year after the episode of acute bronchitis

Patient	Sex (M/F)	Age (years)	Pathogen identified	Smoking history	Family history of atopic disease	Positive skin prick test	SR*FEV ₁ on presentation	PD ₂₀ FEV ₁ (μmol) at 6 weeks	PD ₂₀ FEV ₁ (μmol) at 1 year
1	F	12	No	Non	Yes	Yes	NP†	0.5	0.85
2	M	57	No	Current	No	Yes	-1.76	1.15	NP
3	M	58	No	Current	No	Yes	-2.59	1.20	NP
4	M	56	No	Ex	Yes	No	-1.31	1.30	3.90
5	F	39	<i>M. pneumoniae</i>	Ex	Yes	No	-3.11	2.30	NP
6	F	45	No	Non	Yes	Yes	-1.03	2.4	0.08
7	F	28	No	Non	No	No	-0.95	3.2	NP
8	F	31	(a) No (b) Rhinovirus	Current	No	No	(a) -1.18 (b) -0.58	(a) 4.4 (b) 5.0	> 7.8
9	F	46	No	Current	Yes	Yes	-2.08	5.2	> 7.8
10	M	19	Influenza A	Non	No	Yes	-3.14	7.0	> 7.8
11	F	23	No	Non	Yes	No	-1.74	> 7.8	5.0

*SR = Standardized residual.

†NP = Not performed.

tolerated by the majority of individuals, but may have failed to demonstrate organisms present in the lower respiratory tract.

Finally, there is the possibility that some of these attacks may not have been due to any infective agent, but instead might have been misdiagnosed asthma precipitated by, for example, changes in temperature or acute bronchitis attacks associated with other environmental factors.

The *M. pneumoniae* infections and the Influenza A infections both occurred in defined outbreaks, the former occurring at the end of the recent Mycoplasma epidemic and the latter occurring in November/December. Influenza infections can result in a variety of clinical syndromes ranging from seroconversion without overt illness (35), through acute bronchitis (2), to fulminating pneumonia, but it is perhaps not well recognized as a cause of simple acute bronchitis.

Bacteria were found infrequently (in only three cases) and there were no features that distinguished these cases from the others investigated. The bacterial identification rate was lower than the studies of acute exacerbations of chronic bronchitis (6,8) which is what might be expected, in view of the colonization of the respiratory tract by bacteria in patients with chronic airways disease.

This study was descriptive in nature and so a control group was not recruited. The MRC/PHLS study (2) included a control group and showed significantly lower microbiological identification rates in these individuals than the patient groups. We believe therefore, that it is likely that the micro-organisms we identified were responsible for the illness.

In our population, there was a significant reduction in both FEV₁ and PEF, improving over 6 weeks by 4% and 9% respectively, whilst the change in FVC was less marked. These findings are similar to those of Johanssen *et al.* in patients with influenza uncomplicated by pneumonia (14). In some patients there is an element of smooth muscle constriction since nearly one third of patients bronchodilated with inhaled terbutaline on presentation.

Assessment of bronchial hyperreactivity (BHR) was not performed until 6 weeks after the initial presentation. In the past, patients with upper respiratory tract infection have had HCTs performed safely on presentation (15–19), and since we found only minor changes in pulmonary function, we feel that HCT could be performed on presentation in future studies. Ideally, premorbid HCTs would be needed to assess viral induced changes in BHR, but this would be logistically a major task, but HCTs on presentation may point to an initial even higher degree of reactivity. A positive HCT was observed in 41% of patients, despite

having excluded patients with known asthma or chronic airflow obstruction. The prevalence of BHR in this group, therefore, is higher than that observed in epidemiological studies in this country (20). A proportion of these cases may be 'missed' asthma, but the degree of bronchial hyperreactivity was slight, with only one individual in the range regarded as 'moderate' hyperreactivity by Woolcock and colleagues (26). The bronchial hyperreactivity may be transient due to the recent infection (15), but because we did not perform HCT on presentation, it is not possible to examine this point more closely. Alternatively, the patients with bronchial hyperreactivity may be more liable than normal individuals to develop lower respiratory tract involvement with a viral respiratory tract infection and may be individuals with the 'bronchial irritability syndrome' identified by Mortagy *et al.* (36). Epidemiological studies would only identify such patients if questions were asked about lower respiratory tract infections/symptoms.

In our study, 93% of patients were treated with antibiotics. The benign outcome in all but the one case of lingular pneumonia (who had been given antibiotics on presentation) may be due to the antibiotics reducing the possibility of secondary bacterial infection, or to the illness being induced by a virus or even misdiagnosed asthma. Many patients commented that the inhalation of terbutaline helped ease their cough at the initial consultation and it may prove a useful symptomatic treatment. Further studies could assess whether inhaled steroids or other inhaled anti-inflammatory agents, such as disodium cromoglycate or nedocromil sodium, may be of benefit in managing this inflammatory condition.

Acknowledgements

We would like to thank our GP colleagues, Drs Ellis, Brown, Nyholm and Thebridge for their enthusiasm and tolerance, Dr T Wreghitt at the PHLS Laboratory, Cambridge, for measuring Mycoplasma pneumoniae IgM, Mrs Judi Rudkin for secretarial help and the staff of the Regional Virus Laboratory at East Birmingham Hospital.

References

1. Ayres JG. Seasonal pattern of acute bronchitis in general practice in the United Kingdom 1976–1983. *Thorax* 1986; **41**: 106–110.
2. Poole PM, Tobin JO'H. Viral and epidemiological findings in MRC/PHLS surveys of respiratory disease in hospital and general practice. *Postgrad Med J* 1973; **49**: 778–787.
3. Horn MEC, Brain EA, Gregg I, Inglis JM, Yealland SJ, Taylor P. Respiratory viral infection and wheezy bronchitis in childhood. *Thorax* 1979; **34**: 23–28.

4. Horn MEC, Reed SE, Taylor P. Role of viruses and bacteria in acute wheezy bronchitis in childhood: a study of sputum. *Arch Dis Child* 1979; **54**: 587-592.
5. Glezen WP, Denny FW. Epidemiology of acute lower respiratory disease in children. *N Engl J Med* 1973; **288**: 498-505.
6. Lambert HP, Stern H. Infective factors in exacerbations of bronchitis and asthma. *Br Med J* 1972; **3**: 323-327.
7. Gregg I. The role of viral infection in asthma and bronchitis. In: Proudfoot AT, ed. *Symposium on viral diseases*. Edinburgh: Royal College of Physicians 1975, pp. 82-98.
8. McHardy VU, Inglis JM, Calder MA *et al*. A study of infective and other factors in exacerbations of chronic bronchitis. *Br J Dis Chest* 1980; **74**: 228-238.
9. Speight ANP, Lee DA, Hey EN. Underdiagnosis and undertreatment of asthma in childhood. *Br Med J* 1983; **286**: 1253-1256.
10. Fletcher C, Peto R, Tinker C, Speizer FE. *The Natural History of Chronic Bronchitis and Emphysema*. Oxford: Oxford University Press, 1976, pp. 85-93.
11. Cate TR, Roberts JS, Russ MA, Pierce JA. Effects of common colds on pulmonary function. *Am Rev Respir Dis* 1973; **108**: 858-865.
12. Fridy WW, Ingram RH, Hierholzer JC, Coleman MT. Airway function during mild viral respiratory illnesses. *Ann Intern Med* 1974; **80**: 150-155.
13. Picken JJ, Niewoehner DE, Chester EH. Prolonged effects of viral infections of the upper respiratory tract upon small airways. *Am J Med* 1972; **52**: 738-746.
14. Johanson WG, Pierce AK, Sanford JP. Pulmonary function in uncomplicated influenza. *Am Rev Respir Dis* 1969; **100**: 141-146.
15. Empey DW, Laitinen LA, Jacobs L, Gold WM, Nadel JA. Mechanism of bronchial hyperreactivity in normal subjects after upper respiratory tract infection. *Am Rev Respir Dis* 1976; **113**: 131-139.
16. Utell MJ, Aquilina AT, Hall WJ *et al*. Development of airway reactivity to nitrates in subjects with influenza. *Am Rev Respir Dis* 1980; **121**: 233-241.
17. Aquilina AT, Hall WJ, Douglas RG, Utell MJ. Airway reactivity in subjects with viral upper respiratory tract infections: the effects of exercise and cold air. *Am Rev Respir Dis* 1980; **122**: 3-10.
18. Jenkins CR, Breslin ABX. Upper respiratory tract infections and airway reactivity in normal and asthmatic subjects. *Am Rev Respir Dis* 1984; **130**: 879-883.
19. Halperin SA, Eggleston PA, Beasley P *et al*. Exacerbations of asthma in adults during experimental rhinovirus infections. *Am Rev Respir Dis* 1985; **132**: 976-980.
20. Burney PGJ, Britton JR, Chinn S *et al*. Descriptive epidemiology of bronchial reactivity in an adult population: results from a community study. *Thorax* 1987; **42**: 38-44.
21. Peat JK, Britton WJ, Salome CM, Woolcock AJ. Bronchial hyperresponsiveness in two populations of Australian schoolchildren. II. Relative importance of associated factors. *Clin Allergy* 1987; **17**: 283-290.
22. Miller DL. Collaborative studies of acute respiratory disease in patients seen in general practice and in children admitted to hospital. *Postgrad Med J* 1973; **49**: 749-762.
23. Wreghitt TG, Sillis M. A μ -capture ELISA for detecting *Mycoplasma pneumoniae* IgM: comparison with indirect immunofluorescence and indirect ELISA. *J Hyg Camb* 1985; **94**: 217-227.
24. Skidmore SJ, Tarlow MJ. Interferon assay as a viral diagnostic test. *J Virol Methods* 1987; **16**: 155-158.
25. Yan K, Salome C, Woolcock AJ. Rapid method for measurement of bronchial responsiveness. *Thorax* 1983; **38**: 760-765.
26. Salome CM, Peat JK, Britton WJ, Woolcock AJ. Bronchial hyperresponsiveness in two populations of Australian schoolchildren. I. Relation to respiratory symptoms and diagnosed asthma. *Clin Allergy* 1987; **17**: 271-281.
27. Cotes JE. Lung function. *Assessment and Application in Medicine*. 4th edn., Oxford: Blackwell, 1979, pp. 340.
28. European Coal and Steel Community Recommendations. *Bull Eur Physiopathol Respir* 1983; **19** (Suppl 5): 49.
29. Miller MR, Pincock AC. Predicted values: How should we use them? *Thorax* 1988; **43**: 265-267.
30. Woodhead MA, Macfarlane JT, McCracken JS, Rose DH, Finch RG. Prospective study of the aetiology and outcome of pneumonia in the community. *Lancet* 1987; **i**: 671-674.
31. American Thoracic Society. Definitions and classifications of infectious reactions of the lung. *Am Rev Respir Dis* 1970; **101**: 119.
32. Fry J. Acute Respiratory Infections. In: Fry J, Byrne PS, Johnson S, eds, *A Textbook of Medical Practice*. Lancaster: MTP Press, 1976, p.45
33. Hope-Simpson RE, Miller DL. The definition of acute respiratory illnesses in general practice. *Postgrad Med J* 1973; **49**: 763-770.
34. Hendley JO, Fishburne HB, Gwaltney JM. Coronavirus infections in working adults. *Am Rev Respir Dis* 1972; **105**: 805-811.
35. Hennessy AV, Davenport FM, Horton RJM, Napier JA, Francis T. Asian influenza: occurrence and recurrence, a community and family study. *Milit Med* 1964; **129**: 38-50.
36. Mortagy AK, Howell JBL, Waters WE. Respiratory symptoms and bronchial reactivity: identification of a syndrome and its relation to asthma. *Br Med J* 1986; **293**: 525-529.