A nurse led intermediate care package in patients who have been hospitalised with an acute exacerbation of chronic obstructive pulmonary disease

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ABSTRACT

Objectives: To determine the effects of a nurse led intermediate care programme in patients who have been hospitalised with an acute exacerbation of chronic obstructive pulmonary disease (AECOPD).

Design: Randomised controlled trial.

Setting: Community and hospital care in west London.

Participants: 122 patients with COPD.

Intervention: A care package incorporating initial pulmonary rehabilitation and self-management education, provision of a written, personalised COPD action plan, monthly telephone calls and 3 monthly home visits by a specialist nurse for a period of 2 years.

Main outcome measure: Hospital readmission rate.

Secondary outcomes: Unscheduled primary care consultations and quality of life.

Results: There were no differences in hospital admission rates or in exacerbation rates between the two groups. Self-management of exacerbations was significantly different and the intervention group were more likely to be treated with oral steroids alone or oral steroids and antibiotics, and the initiators of treatment for exacerbations were statistically more likely to be the patients themselves. 12 patients in the control group died during the 2 year period, eight as a result of COPD, compared with six patients in the intervention group, of whom one died from COPD. This is a significant difference. When the numbers were adjusted to reflect the numbers still alive at 2 years, in the intervention group patients reported a total of 171 unscheduled contacts with their general practitioner (GP) and in the control group, 280 contacts. The number needed to treat was 0.558—ie, for every one COPD patient receiving the intervention and self-management advice, there were 1.79 fewer unscheduled contacts with the GP.

Conclusions: An intermediate care package incorporating pulmonary rehabilitation, self-management education and the receipt of a written COPD action plan, together with regular nurse contact, is associated with a reduced need for unscheduled primary care consultations and a reduction in deaths due to COPD but did not affect the hospital readmission rate.

Chronic obstructive pulmonary disease (COPD) represents a global health burden and will move from 12th place to be the fifth largest cause of disability adjusted life years lost by 2020.[1] In the UK, 30 000 deaths per year are attributed to COPD, over 5% of all deaths,[2] and up to one in eight emergency hospital admissions may be due to COPD.[3] Much of the morbidity and mortality related to the condition results from exacerbations of the disease, and frequent exacerbations are associated with more rapid decline in lung function.[4] These exacerbations are much feared by patients who dislike their impact on everyday life and fear of being housebound, bedridden or hospitalised.[5][6] Previous research has focused on optimal therapeutic strategies for both the prevention[7–10] and treatment of acute exacerbations[11] and also on the effectiveness of care interventions such as "hospital at home" and early discharge schemes for those having acute exacerbations of COPD.[12][13] A systematic review of the effectiveness of innovations in nurse led management has concluded that there is little evidence to date to support such interventions, but the authors conclude that the data are too sparse to be certain, and none of the studies extended beyond a duration of 12 months.[14] There is a similar lack of evidence to show whether self management education alters outcomes in COPD.[15]

We have undertaken a randomised controlled trial to determine the effects of a nurse led intermediate care programme in the management of patients who have been hospitalised with an acute exacerbation of COPD. The care package incorporated initial pulmonary rehabilitation followed by an emphasis on self-management education and nurse follow-up for 2 years.

METHODS

A list of patients who had been admitted to Charing Cross and Hammersmith Hospitals, London, UK, between 1 January 2000 and 31 August 2004 with the main reason for admission being coded on discharge as having been due to an acute exacerbation of COPD was obtained from the hospital database. The clinical notes of these patients were reviewed by the investigators using a proforma. If thought to represent a suitable patient, the case notes were discussed and, where necessary, further information obtained. Exclusion criteria included significant comorbidity such as severe heart disease or cancer, or any condition that would preclude participation in the physical therapy component of a pulmonary rehabilitation programme. None of the patients had previously undertaken a pulmonary rehabilitation programme. Those patients thought to be suitable were sent a letter inviting them to participate in the study and those who responded were invited for initial assessment and, if still suitable and

agreeable, randomised to the intervention or usual care groups.

The study intervention involved all patients initially participating in a hospital based pulmonary rehabilitation programme consisting of two attendances per week for 4 weeks. During this visit, the patients received general education about their disease and its treatment (1 h per session) and underwent an individualised physical training programme (1 h per session). Following completion of the pulmonary rehabilitation programme, the patients received a baseline home visit by a specialist respiratory nurse, and during this first visit, the patients were given a personalised written COPD action plan (a copy of which is shown in appendix A online). This contained both lifestyle advice and advice about their usual medication, and gave specific advice about when the patient should start a course of antibiotics and when they should start a course of steroid tablets. The general practitioners of these patients were requested to provide for the patient reserve supplies of these medications.

Subsequently, those in the intervention group had monthly telephone calls from the respiratory nurses and a home visit every 3 months. During each interview and visit, the nurses undertook a structured approach to history taking and during home visits measured pulse and respiratory rate, oxygen saturation and end-tidal carbon monoxide. Spirometry was performed at baseline and after 12 and 24 months. During both telephone and home visits, they reinforced advice regarding treatments, smoking cessation if relevant, the need to continue their exercise therapy and discussed and reinforced the self-management education which had been given and offered encouragement for successful self-treatment. The patients were also given written advice about the treatment of COPD which they were asked to show to their doctor if they underwent any unscheduled healthcare.

Patients in the control group received usual care from their primary care physician, or secondary care and/or the respiratory nursing service as appropriate. No attempt was made to influence this care. Patients in both the control and intervention groups had their use of healthcare monitored by monthly telephone self-report verified by confirmation of the general practice and hospital records. General practice records alone were not used because they do not always contain a record of hospital admissions and it is not always possible to tell which consultation within primary care was scheduled and which was unscheduled. Corroboration of patient self-reported general practitioner (GP) consultations was undertaken by the GPs themselves from their records. Corroboration of patient self-reported hospital admissions was undertaken by the respiratory research nurses against local hospital records. A copy of the death certificate was requested for all patients who died.

Quality of life was assessed using the Chronic Respiratory Questionnaire[16] [17] at baseline and three further times during the subsequent 2 years.

The study was approved by the Riverside Research Ethics Committee.

Statistical analysis

The primary end point for our trial was to be hospital readmission rate with secondary outcomes being unscheduled consultations with GP and quality of life. Retrospective review of previous local hospital admission rates suggested that over a 2 year period, 579 patients might accrue a total of 1180 admissions (mean 2.04 admissions per patient). We calculated that we would need a study of 88 patients to have an 80% chance of detecting a difference (95% readmission in usual care

versus 75% in the intervention group), using a two sided alpha = 0.05. All statistical analysis was carried out using the software program SPSS (V.12.0). For all analyses, χ^2 tests, the Mann–Whitney test, independent or paired t tests were used.

RESULTS

There were 2305 admissions to Hammersmith Hospitals NHS Trust between 1 January 2000 and 31 August 2004. Many of those were multiple admissions and the total number of patients admitted with an acute exacerbation of COPD in that period was 1247. A total of 507 of these patients had died at the time we began recruiting to this study and of the 740 who were alive, 166 lived outside our area and were not therefore able to attend the pulmonary rehabilitation programme if randomised to intervention. This left 574 patients who were assessed. Two hundred and seventy-seven of these were thought to be unsuitable for the reasons shown in table 1.
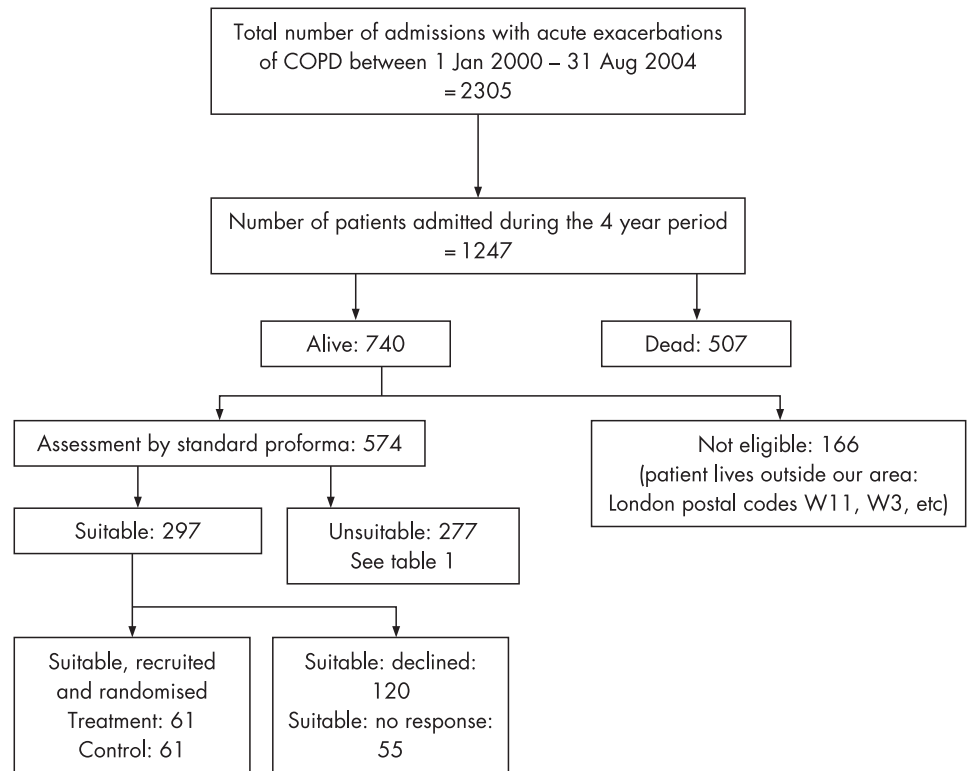
Of the 297 patients who were suitable for inclusion in the trial, 120 (40.4%) responded that they did not wish to take part in the trial, 55 (18.5%) did not reply and 122 (41%) patients were suitable, and were recruited and randomised by the use of random numbers to the intervention or control group. These data are summarised in the consort diagram in fig 1. Demographic data and details of severity, treatment, unscheduled use of healthcare and quality of life for all patients at baseline and at the end of year 2 is shown in table 2.

From table 2 it can be seen that at baseline, the control and intervention groups were well matched for age, gender, severity of disease, previous hospitalisation rate, breathlessness scores, weight, smoking habits and use of home oxygen. Both groups had similar numbers of initial and persisting smokers and breathlessness scores worsened during the course of the study in both groups. Lung function appeared to decline to a greater degree in the control group than in the intervention group, but this difference was not statistically significant. At baseline, similar proportions of patients were receiving long acting beta agonist bronchodilators and anticholinergic agents, and the majority of patients in both the intervention group and in the control group were taking inhaled steroids at entry to the trial. At the end of the trial, patients in the intervention group were statistically more likely to be on a long acting inhaled beta agonist or a short or long acting anticholinergic agent in the intervention group, compared with the control group.

At the beginning of the trial, 5% of the intervention group and 11.5% of the control group had a reserve supply of antibiotics; corresponding values for steroid tablets were 5% and 7%, respectively. After the intervention, over 95% had both types of reserve treatments available, compared with the control group, where 17% had antibiotics available at the end of 1 year and 25% at the end of 2 years. Sixteen per cent of the control

Table 1 Reasons for patients being regarded as unsuitable to be included in the trial (n = 277)

▶ Alternative diagnosis, no evidence of COPD: 122 (44.1%)
These were mainly because perusal of the notes gave clear evidence of another major pulmonary condition, such as asthma, diffuse parenchymal lung disease or bronchiectasis

▶ Psychosocial problems and significant cognitive impairment: 68 (28.5%)
(eg, dementia, blindness, alcohol abuse, psychiatric illness, homelessness or in prison)

▶ Severe limiting comorbidity involving other major organ systems: 50 (18.1%)
(mainly cardiac but also cancer and alcohol related liver disease, etc)

▶ Factors limiting mobility and locomotion: 37 (13.3%)
(housebound, wheelchair bound, previous cerebrovascular accident, severe arthritis)

Figure 1 Flowchart of patient recruitment to the study.

Total number of admissions with acute exacerbations of COPD between 1 Jan 2000 – 31 Aug 2004 = 2305

Number of patients admitted during the 4 year period = 1247

Alive: 740

Dead: 507

Assessment by standard proforma: 574

Not eligible: 166 (patient lives outside our area: London postal codes W11, W3, etc)

Suitable: 297

Unsuitable: 277 See table 1

Suitable, recruited and randomised Treatment: 61 Control: 61

Suitable: declined: 120 Suitable: no response: 55

group had a reserve supply of steroid tablets at the end of 2 years. There were no differences in exacerbation rates between the two groups (an exacerbation being defined as an unscheduled need for healthcare, or need for steroid tablets, or antibiotics for worsening of their COPD), nor was there a difference in the total number of exacerbations per group over the 2 year period. There was also no difference in the total number of hospital admissions or in the number of patients having an admission to hospital during the 2 year period. Because this could have been influenced by differences in death rates, this was also analysed as days alive and out of hospital in the two groups, but there was no statistically significant difference. Self-management of exacerbations was however significantly different and patients in the intervention group were more likely to have exacerbations treated with oral steroids alone or oral steroids and antibiotics than the control group, who were more likely to be treated with antibiotics alone. The initiator of treatment for exacerbations was statistically more likely to be the patient themselves in the intervention group and the GP in the control group.

Twelve patients in the control group died during the 2 year period, eight as a result of COPD, compared with six patients dying in the intervention group, of which one died from COPD. The number dying from COPD in the control group was significantly higher both as a proportion of the total deaths and as a proportion of all patients in the trial compared with those in the intervention group. The non-COPD deaths included four deaths due to pneumonia (three in the control group and one in the intervention group), one due to metastatic lung cancer, one due to cerebral haemorrhage, two due to coronary artery disease and one due to septic shock. All but two patients died in hospital. The stated cause of death was confirmed by acquisition of copies of the death certificates in all but one case. One patient died on a cruise ship off the coast of West Africa and death was reported by relatives to have been due to

worsening of their lung condition over several days following exposure to adverse environmental conditions. When the numbers in the intervention and control groups were adjusted to reflect the numbers still alive, it can be seen that at 2 years, 55 of 61 patients in the intervention group were still alive, and these patients had reported a total of 171 unscheduled contacts with their GP. In the control group, 49 of the 61 patients were still alive, and they had had 280 contacts with their GP. The number needed to treat, therefore, was 0.558—ie, for every one COPD patient receiving self-management education, there was 1.79 less unscheduled contacts with the GP. The health economic costs of the pulmonary rehabilitation programme are shown in table 3, and suggest that the costs of the intervention were only very slightly greater per patient than the savings on unscheduled primary healthcare (£153 compared with £142).

Within the Chronic Respiratory Questionnaire there was a statistical and clinically significant worsening in the mean dyspnoea score in both the intervention and control groups from baseline to end of year 2, and the mean mastery score improved in the intervention group but this change was not clinically significant.

To ensure that any differences did not reflect differences in routine healthcare received, data in table 4 show that equal numbers of patients received primary care alone in both the intervention and control groups but some in the control group also received care from the local respiratory nursing service. They may have been offered care which contained some similarities to that offered by the research respiratory nurses.

DISCUSSION

The proportion of patients who had been admitted to hospital with an exacerbation of COPD who were eligible for and consented to take part in this trial was lower than anticipated. This was because of both polymorbidity and non-consent, and

Table 2 Data for patients in the intervention and control groups at entry to the trial, and for those alive, at the end of year 2

Variables	Treatment (n = 61) Baseline (n = 61) (% (n))	Year 2 (n = 55) (% (n))	Control (n = 61) Baseline (n = 61) (% (n))	Year 2 (n = 49) (% (n))	Percentage difference‡	p Value
Males (%)	49.2 (30/61)		49.2 (30/61)		0	NS
Age (y) (mean (SD))	69.9 (9.6)		69.68 (10.4)		0.22	0.474†
BMI (median (range))	25.0 (14–38)		24 (16–40)		1	
Smoking status						
Current smoker	29.5 (18/61)	29.1 (16/55)	19.7 (12/61)	22.4 (11/49)	6.7	0.426
MRC dyspnoea score (median (range))	3 (1–5)	4 (1–5)	3 (1–5)	4 (1–5)		
Home O_2						
Yes (%)	19.7 (12/61)	25.4 (14/55)	21.3 (13/61)	26.5 (13/49)	−1.1	0.901
Cylinder	33.3 (4/12)	28.6 (4/14)	46.1 (6/13)	46.1 (6/13)	−17.5	
Concentrator	66.6 (8/12)	71.4 (10/14)	53.8 (7/13)	53.8 (7/13)	17.6	
FEV_1 (mean (SD))	1.04 (0.44)	0.95 (0.42)	1.17 (0.49)	1.06 (0.44)	−0.16	0.186*
FVC (mean (SD))	2.12 (0.74)	1.98 (0.65)	2.2 (0.77)	2.1 (0.79)	−0.12	0.387*
FEV_1% pred (mean (SD))	42.9 (15.5)	41.1 (17.1)	48.9 (18.69)	45.7 (17.48)	−4.6	0.201*
Change in FEV_1 ml (mean (SD))		−94.9 (162.1)		−162.7 (249.9)		0.111*
Prior hospital admissions (before randomisation)						
Total No of admissions	73		64		9	0.186*
Admission in the 2 y prior to recruitment (% (n))	80.3 (49/61)		68.8 (42/61)		11.5	0.145
No of admissions/patient/2 y (median (range))	1.0 (0–6)		1.0 (0–6)			
LABA prescription						
Yes (%)	67.2 (41/61)	92.7 (51/55)	63.9 (39/61)	71.4 (35/49)	21.3	0.004
LABA inhaler alone	29.3 (12/41)	0.00 (0/51)	33.3 (13/39)	8.6 (3/35)	8.6	
LABA and ICS combination inhaler	70.7 (29/41)	100 (51/51)	66.7 (26/39)	91.4 (32/35)	8.6	
ICS prescription						
Yes (%)	88.5 (54/61)	94.5 (52/55)	80.3 (49/61)	100.0 (49/49)	−5.5	0.097
Inhaled ICS alone	46.3 (25/54)	1.9 (1/52)	46.9 (23/49)	24.5 (12/49)	−22.6	
ICS and LABA combination inhaler	53.7 (29/54)	98.0 (51/52)	53.1 (26/49)	65.3 (32/49)	32.7	
Anticholinergic prescription						
Yes (%)	73.7 (45/61)	89.1 (49/55)	63.9 (39/61)	65.3 (32/49)	23.8	0.004
Short acting	53.3 (24/45)	6.1 (3/49)	58.9 (23/39)	31.2 (10/32)	−25.1	
Long acting	46.6 (21/45)	93.9 (46/49)	41.0 (16/39)	68.7 (22/32)	25.2	
Deaths in 2 y study period						
Total (%)		9.8 (6/61)		19.7 (12/61)	−13.1	0.126
COPD		16.7 (1/6)		66.6 (8/12)	−41.6	0.015
Non-COPD		83.3 (5/6)		33.3 (4/12)	41.7	
Hospital admissions						
Total No of admissions in study period		70		52	3.8	0.421*
Admission in study period ((% (n))		52.7 (29/55)		48.9 (24/49)		0.361
No of admissions per patient (median (range))		0 (0–13)		0 (0–5)		
Days alive and out of hospital/2 y		41400/44530		39 578/44 530		0.705*
Total		(93.0%)		(88.9%)		
Median (range)		724 (244–730)		730 (19–730)		
Self-management of COPD						
Reserve oral antibiotics prescribed	4.9 (3/61)	92.7 (51/55)	11.5 (7/61)	24.5 (12/49)	68.2	<0.001
Reserve oral steroids prescribed	4.9 (3/61)	92.7 (51/55)	6.5 (4/61)	16.3 (8/49)	76.4	<0.001
No of patients who reported an exacerbation		86.9 (53/61)		86.9 (53/61)	0	
Treated with oral antibiotics only		36.2 (161/445)		63.8 (233/364)	−27.6	<0.001
Treated with oral steroids only		19.8 (88/445)		7.7 (28/364)	12.1	<0.001
Treated with oral antibiotics and steroids		44.0 (196/445)		28.3 (103/364)	15.7	<0.001
Who initiated treatment for exacerbation?						
Self		43.1 (192/445)		10.4 (38/364)	32.7	<0.001
Research nurse		6.3 (28/445)		0 (0/364)	6.3	<0.001
GP		31.5 (140/445)		68.7 (250/364)	−37.2	<0.001
A&E doctor		0.9 (4/445)		2.5 (9/364)	−1.6	0.077
Outpatients clinic doctor		2.9 (13/445)		5.2 (19/364)	−2.3	0.095
Inpatient hospital doctor		15.3 (68/445)		13.5 (49/364)	1.8	0.464
Total No of unscheduled GP visits/contact		171		280	−109	<0.05*
Telephonic consultation		9.9 (17/171)		3.5 (10/280)	6.4	0.006
Practice attendance		67.8 (116/171)		88.2 (247/280)	−20.4	<0.001
Home visit		22.3 (38/171)		8.2 (23/280)	14.1	<0.001
Disease Specific QoL Questionnaire: CRQ	47 (fully completed)	47 (fully completed)	40 (fully completed)	40 (fully completed)		

Continued

Table 2 Continued

Variables	Treatment (n = 61)		Control (n = 61)		Percentage difference‡	p Value
	Baseline (n = 61) (% (n))	Year 2 (n = 55) (% (n))	Baseline (n = 61) (% (n))	Year 2 (n = 49) (% (n))		
Dyspnoea	3.55 (1.12)	2.83 (1.21)¶	3.49 (1.26)	2.65 (1.23)¶		¶p<0.05 (paired t-test) between baseline and 2 y
Fatigue	3.62 (1.26)	3.68 (1.35)	3.59 (1.51)	3.24 (1.11)		
Physical domain						
Emotional function	4.58 (1.22)	4.74 (1.43)	4.39 (1.37)	4.03 (1.30)		
Mastery	4.71 (1.26)	5.14 (1.33)¶	4.71 (1.46)	4.44 (1.45)		
Emotional domain						
Total score	16.46 (4.05)	16.41 (3.94)	16.18 (4.56)	14.37 (3.97)¶		

*Mann Whitney, †Student t test.

‡Percentage difference at 2 years between intervention and control (treatment − control).

§Control versus treatment end of year 2, all χ^2 statistical analysis except where stated.

BMI, body mass index; COPD, chronic obstructive pulmonary disease; CRQ, Chronic Respiratory Questionnaire; FEV$_1$, forced expiratory volume in 1 s; FVC, forced vital capacity; GP, general practitioner; ICS, inhaled corticosteroids; LABA, long acting beta-2 agonist.

Table 3 Health economic costs of the pulmonary rehabilitation programme

	Treatment (n = 61)	Control (n = 61)
GP unscheduled contacts	171	280
Cost of GP consultations (Curtis 2006)*	£5301	£8680
Cost of self management education (1 hr group sessions n = 8 and 1 home visit (approx 70 min))	81 nurse hours = £2511	No self management education/ home visits given
Travel costs for home visits (Netten 2006)	£37.21	£0
Routine follow-up calls for patients (by nurses)	547 calls, 48 hours (total length)	No follow-up calls
	Cost £28.82	Cost £0
Nurse costs for telephone calls	£1488	
Total	Nurse costs (£2511 + £1488) + telephone calls (£28.82) + unscheduled healthcare costs (£5301)	
	£9328.82	£8680.00
Approx cost of intervention (per patient)	£66	
Mean cost of unscheduled healthcare for all in the intervention group	(171×£31)/61 = £86.90	(280×£31)/61 = £142.30
Total cost (per patient)	£153	£142.30

*Curtis L, Netter A, Unit costs of health and social care 2006. Kent: Personal Social Services Research Unit, University of Kent at Canterbury, 2006. ISSN 1368-230X.

the latter was due more to the intervention, including pulmonary rehabilitation, than to it being a research trial.[18] As a result, recruitment was lower than expected but neither the total number of readmissions nor the number of patients having an admission during the 2 year follow-up period differed between the intervention and control groups. This result is thus consistent with a previous review[14] but extends our knowledge to show that the same applies if the intervention continues for 2 years. The secondary outcome of rate of

unscheduled GP consultations was significantly less in the intervention group.

It thus looks as though the self-management education part of the intervention, given in group format during the pulmonary rehabilitation programme and individually by the nurse subsequently, led to intervention patients being more likely to have received supplies of antibiotics and steroids and more likely to start these themselves to treat an exacerbation than the control group.

Unexpectedly, we showed a significant reduction in deaths due to COPD in the intervention group. We acknowledge the difficulties around the accuracy of death certificates. However, this reduction in death rate could be explained by a reduction in the severity of exacerbations as a result of self-treatment with antibiotics and steroid tablets and it is probable that the ready availability of reserve medications leads to their prompter use, and earlier treatment of exacerbations has been shown to be associated with improved outcomes.[19] A less likely explanation for a reduced death rate would be differences in routine inhaler therapy in the intervention group compared with the control group. While controversial, there have been suggestions that the use of inhaled steroids[20–24] might be associated with a reduction

Table 4 Care received by those in the intervention and control groups

	Intervention group	Control group
Primary care	31/61	36/61
1′ care doctors only	31	32
1′ care doctors and RNS	0	4
Secondary care	30/61	25/61
1′ and 2′ care doctors only	30	16
1′ and 2′ care doctors and RNS	0	9

RNS, Respiratory Nursing Service.

in mortality. However, essentially, all patients in both the intervention and control arms in our trial were receiving inhaled steroids, but there were differences in the use of anticholinergic agents and long acting beta agonists in our intervention arm and this may have contributed to increased survival.[8 9 24] However, most of those who died were also on these medications. The likelihood that the reduction in mortality instead reflected prompt self-treatment would be consistent with review of 11 randomised controlled trials comparing antibiotics with placebo for acute worsening of COPD, which showed that regardless of antibiotic used, antibiotic therapy significantly reduced mortality.[25] Such a reduction in mortality as a result of steroid tablet use has not been demonstrated.[26] Why we demonstrated a significant reduction in unscheduled need for primary healthcare and a reduction in mortality, but no reduction in hospital admission rates, is less clear. Self-administration of antibiotics (and steroid tablets) may have reduced the severity of exacerbations sufficiently to reduce mortality without influencing the need or otherwise for admission to hospital, which is in any way likely to be influenced by social factors such as isolation, depression and available support in the home.

There are clearly still many unknowns with regard to what constitutes optimal care for those with COPD. We included pulmonary rehabilitation within our intermediate care package because it contains elements of exercise therapy, group support and group education, and has been shown to beneficially affect outcomes such as quality of life, exercise ability and breathlessness scores. It has not however been shown conclusively to affect hospitalisation rate, mortality or primary care consultation rates.[27] The improvements in the latter two outcomes in our study are therefore unlikely to reflect incorporation of pulmonary rehabilitation within the intervention package. Regular follow-up by nurses in this study has again failed to demonstrate a benefit in terms of reduction in hospital admission rates and this result is almost identical to a shorter study from this hospital 20 years ago[28] However, we have demonstrated that an intervention which includes group advice regarding self-management education, followed by the issuing to each patient of a personalised action plan leads to an alteration in self-management behaviour and reduced need for unscheduled primary care. Previously, the case for self-management education in COPD was not proven.[15] Studies reported subsequent to that review, and subsequent to us starting this study, have shown conflicting results. One group[29] showed no differences in quality of life scores or walking distance and no reduction in exacerbation rate following self-management education, while another demonstrated a significant reduction in hospital admissions, emergency department visits and physicians visits.[30] In the latter study, however, and indeed in our own, it is difficult to be certain which of several parts of a complex package achieved which benefit. The relationship between possession of antibiotics and oral steroids, their self-administration and the reduced need for unscheduled primary care in our study does appear to be causally related. One explanation for differences in previous studies may lie in the way in which the personalised self-management advice was given. In asthma, where there is overwhelming evidence in favour of self-management education,[31 32] written action plans are a key component. In the systematic review of self-management in COPD,[15] only two studies involved patients receiving a written action plan, and study of the action plans used in those two studies shows that one was a typical asthma action plan[33] and did not, for example, include any advice to the patient

about antibiotics. The other study[34] did include an action plan which included advice as to when to start antibiotics and this did show an alteration in patient usage of antibiotics and steroids but the study was not powered to show a change in outcome.

A subsequent Cochrane review[35] has suggested that use of action plans can alter self-management behaviour in those with COPD but did not lead to any hard outcomes. Our study does show that self-management education can be associated with change in patient behaviour and a reduction in the need for urgent primary care, and this plus follow-up by nurses and possibly optimisation of inhaled therapy can be associated with reduced mortality due to COPD.

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Lung alert

IP-10 as a biomarker for rhinoviral infections in asthma

Asthma exacerbations are most commonly triggered by viruses, particularly rhinovirus. There is currently no biomarker that can be used to predict that a virus has triggered an exacerbation.

This group studied bronchial epithelial cells (BECs) obtained by bronchoscopy from 10 healthy controls and 10 patients with asthma who had never received inhaled corticosteroids. The epithelial cells were cultured and exposed to rhinovirus 16. The supernatants were measured for various cytokines including interferon-γ-induced protein 10 (IP-10) by FACs analysis.

There was a significant increase in IP-10, RANTES, interleukin (IL)-6, IL-8 and tumour necrosis factor α from baseline, which peaked at 48 h after infection. There was no significant difference between patients with asthma and controls. Pretreatment of the BECs with dexamethasone did not significantly reduce the release of IP-10. Rhinovirus replication was significantly greater in BECs of subjects with asthma than controls, and there was a positive correlation between IP-10 release and viral concentrations.

The authors went on to investigate whether acute virus-induced asthma could be differentiated form non-infective acute asthma. These patients were not steroid naïve. They found that patients with acute virus-induced asthma had significantly increased IP-10 levels compared with those with non-viral acute asthma. Viral infections were also found to be associated with lower forced expiratory volume in 1 s. Individuals with acute rhinoviral infection specifically had significantly increased IP-10 levels, and a level of 168–1916 pg/ml increased the likelihood of rhinoviral infection more than twofold.

It appears that rhinoviral infection initiates an inflammatory response with marked release of IP-10, and this correlates with rhinovirus replication. The authors conclude that IP-10 may therefore be a useful clinical marker to identify rhinovirus-induced asthma and may be a potential therapeutic target for the future.

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