

the extremes of incomprehensibility. The Commission is nevertheless concerned that its arguments should be comprehensible and its recommendations unambiguous—aims best achieved by a clear prose style, unencumbered by jargon and circumlocution. If it is readable, even to the uninitiated, this target will have been reached. No work of literature has ever been written by a committee, and anyone who has experienced the attempts of a group to compose a flowing paragraph knows its impossibility. The style of the final report therefore depends particularly on the secretary, but members are free to offer amendments or even large-scale rewrites if they feel so disposed. This is mostly done in correspondence, and the tactful secretary incorporates these offerings judiciously. Arresting phrases or a few incisive sentences are often interpolated in this way.

The end in sight

Unless there is a determined minority, acrimony recedes by subsequent meetings, for the end is in sight. Giggles sometimes

break out and the Commission's own family jokes are heard more often; murmurs of a final party or dinner after the signing ceremony are heard. As the drafts improve in style, compromises over the sticky parts are reached, and at last a final draft is agreed. A very senior civil servant reads it and talks to the Commission about obscurities and difficulties. Then the smooth machinery of Whitehall takes over. The chairman utters grave warnings against "leaks," intended or inadvertent. A date for release has to be considered and press conferences are arranged. The date must not clash with any expected public event, for the Commission does not want its press impact to be diminished. Each member has to state precisely the form in which his name, style, and titles are to be published. While members begin to relax, the chairman tenses. A government reception is given on the day of signing, with cocktail party courtesy from important figures, longing to know what has been said but forbidden to ask. On the day before publication the members each receive a printed copy of the volume, but without its blue cover to indicate its still unofficial status. Finally, publication day dawns, with eager perusal of press response, and then—a slow decline into obscurity.

Occasional Review

The natural history of chronic airflow obstruction

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Summary

A prospective epidemiological study of the early stages of the development of chronic obstructive pulmonary disease was performed on London working men. The findings showed that forced expiratory volume in one second (FEV₁) falls gradually over a lifetime, but in most non-smokers and many smokers clinically significant airflow obstruction never develops. In susceptible people, however, smoking causes irreversible obstructive changes. If a susceptible smoker stops smoking he will not recover his lung function, but the average further rates of loss of FEV₁ will revert to normal. Therefore, severe or fatal obstructive lung disease could be prevented by screening smokers' lung function in early middle age if those with reduced function could be induced to stop smoking. Infective processes and chronic mucus hypersecretion do not cause chronic airflow obstruction to progress more rapidly. There are thus two largely unrelated

disease processes, chronic airflow obstruction and the hypersecretory disorder (including infective processes).

Introduction

Chronic bronchitis and emphysema are often referred to together as the "British disease" because they are such a common cause of death and disability in Britain. Since their cardinal feature is irreversible obstruction to bronchial airflow, they are often referred to jointly as chronic obstructive pulmonary disease. This term includes chronic obstructive bronchitis and emphysema but excludes asthma or any localised cause of airways obstruction.¹

Although the number of deaths certified as being due to these conditions has declined in the past 10 years, there were still some 25 000 in England and Wales in 1974. There were also about 1000 deaths due to respiratory heart disease plus an unknown number, perhaps as many as 10 000, certified as being due either to other forms of heart disease or to pneumonia where chronic obstructive pulmonary disease was not certified as the underlying cause of death even though it caused the fatal condition or aggravated a condition that would not otherwise have been fatal. The total mortality attributable to chronic obstructive pulmonary disease is thus about the same as the total mortality attributed to lung cancer. If it were possible to identify all deaths that would not have occurred in the absence of chronic obstructive pulmonary disease it would probably be found that the proportion misleadingly certified as being due to other underlying causes is even larger in other countries, including the USA, than in Britain.² Although the certified death rates in other countries are lower than those in Britain, they

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therefore represent only a fraction of the total mortality actually attributable to chronic obstructive pulmonary disease.

When airflow obstruction first causes breathlessness that leads a patient to consult a doctor, it is usually sufficiently severe to reduce the forced expiratory volume in one second (FEV₁) to about 1 litre, which is less than half the normal value. Thereafter the course of the condition usually progresses relentlessly over five or more years, with further loss of FEV₁, causing more and more distressing disability and, finally, death from respiratory failure. This often occurs in an episode of bronchial infection complicated by cor pulmonale.

These later phases of the disease have long been well documented³⁻⁵ and it has been found that the severity of airflow obstruction, usually measured by FEV₁, is the main determinant of prognosis.⁶⁻⁸ Since the damage to the lung appears to be irreversible at this late stage of the disease, any preventive action must be taken much earlier. The essential role of smoking has long been clear,⁹⁻¹² but stopping smoking in the terminal stage is too late,^{5, 6} and general health education has not had much effect on the people (male manual workers) who suffer the greatest risk of this disease.¹³ Perhaps it could be more effective if concentrated on potential patients at an earlier stage, but how could they be identified?

In the late 1950s¹⁴ and again more recently¹⁵ it was suggested that such people could be recognised by their having a productive cough (simple bronchitis). Pathologists suggested that mucus hypersecretion encouraged bronchial infection, which caused obstructive damage to bronchioles and alveolar tissues.¹⁶⁻¹⁸ The fatal consequences of infections in terminal patients with terminal obstruction lent plausibility to this latter view, but it remains an unproved hypothesis.

In 1960 the Medical Research Council's committee on the aetiology of chronic bronchitis became concerned with the question of how smoking interacts with other factors in causing airflow obstruction and commissioned a prospective study of respiratory symptoms and changes in ventilatory function over a period of eight years in a large group of working men, few of whom had any clinical disease. The full results of this study were recently published¹⁹ together with some new statistical considerations.²⁰ We report here a short summary of the methods and main results and conclusions of this study, some of which conflict with current orthodoxy, to stimulate debate in a wider circle than those who will read a specialist epidemiological monograph.

Methods

In 1961 a stratified random sample of men (mostly skilled manual or clerical) aged 30-59 working in West London was taken. Of an initial sample of 1136 men 792 were seen regularly enough over the next eight years to provide sufficient data for analysis. The men were seen every six months, when the following measurements were made.

Mucus hypersecretion was assessed by standard questions about chronic phlegm production and by six-monthly measurements of the volume of phlegm brought up during the first hour after waking on three separate mornings. These two independent measures enabled us to rank the men with respect to chronic expectoration more reliably than has been done in other studies, in nearly all of which single estimates based on questionnaires have been used.

Bronchial infections were assessed by standard questions about chest colds or illnesses in the previous six months during which phlegm production had increased; by recording the purulence of all phlegm specimens posted to us; and by measuring serum antibodies to *Haemophilus influenzae* on one occasion.

Airflow obstruction was estimated by measuring FEV₁. After two practice blows into a spirometer the FEV₁ readings of three subsequent blows were recorded. The maximum of these three was used, contrary to MRC recommendations,²¹ because it was definitely more reproducible than the mean (p 164¹⁹). These six-monthly FEV₁ measurements over eight years allowed us to estimate the average rate of decline of FEV₁ for each man during the study. These estimates are called "FEV₁ slopes." Unfortunately, FEV₁ slopes of individuals could not be measured accurately enough to be useful, but averages of the FEV₁ slopes of groups of a dozen or more men were accurate enough

for our analysis of causal factors. To ensure that FEV₁ loss was a valid measure of development of airflow obstruction 18 men with conditions that could cause restrictive loss of FEV₁ were excluded.

Results and comment

SMOKING AND LOSS OF FEV₁

The following conclusions are summarised in figs 1 and 2.

Firstly, we found that FEV₁ declines continuously and smoothly over an individual's life (fig 1). We believe that sudden large irreversible falls are very rare, for the 9190 measurements that we made of the changes in FEV₁ between successive six-monthly surveys were distributed exactly symmetrically about their mean, with no evidence of any "tail" due to sudden substantial losses (p 224¹⁹). The rate of loss seems to accelerate slightly with aging (p 67¹⁹).

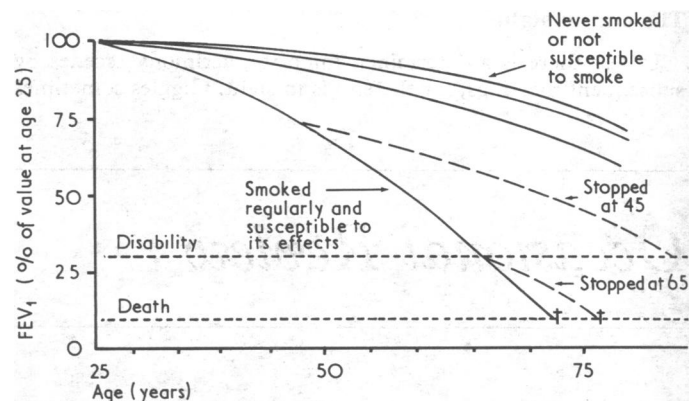


FIG 1—Risks for various men if they smoke: differences between these lines illustrate effects that smoking, and stopping smoking, can have on FEV₁ of man who is liable to develop chronic obstructive lung disease if he smokes. †=Death, the underlying cause of which is irreversible chronic obstructive lung disease, whether the immediate cause of death is respiratory failure, pneumonia, cor pulmonale, or aggravation of other heart disease by respiratory insufficiency. Although this shows rate of loss of FEV₁ for one particular susceptible smoker, other susceptible smokers will have different rates of loss, thus reaching "disability" at different ages.

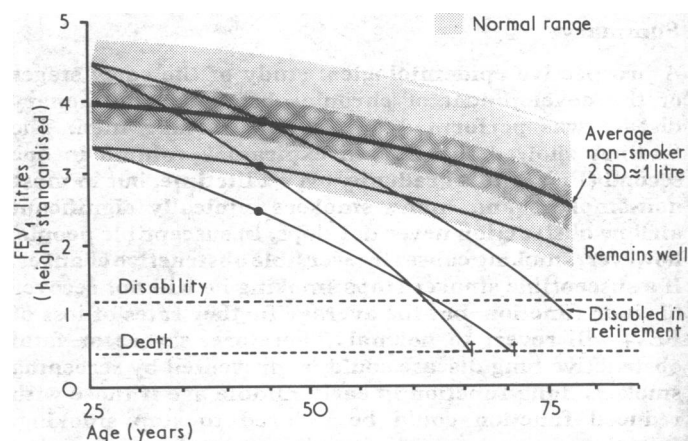


FIG 2—Identifying susceptible smokers in time to prevent death: various patterns of FEV₁ decline (—) with age that are consistent with certain observations of FEV₁ in middle age (●). Smokers who eventually die of chronic obstructive lung disease have usually already suffered appreciable FEV₁ loss in their 40s. Most smokers whose FEV₁ is already below the normal range for non-smokers by early middle age are thus at grave risk of later death from airflow obstruction unless they stop smoking immediately, while smokers whose FEV₁ is still above average in middle age will probably not get serious obstruction. If, however, FEV₁ at age 25 was originally above average for other men (of the same age and height) then FEV₁ may still lie within the normal range for middle-aged non-smokers even though considerable FEV₁ loss has occurred. It is therefore impossible to be sure of the prognosis of a smoker whose FEV₁ in middle age is just one or two standard deviations below the average for non-smokers, although many of those around two standard deviations below average will become disabled over the coming decades. Other tests may enable those at greatest risk to be detected.

Secondly, non-smokers lose FEV₁ slowly and almost never developed clinically significant airflow obstruction. None of the 103 non-smokers in our study had any evidence of even moderate obstruction (p 83¹⁹).

Thirdly, many smokers lose FEV₁ almost as slowly as non-smokers and never develop clinically severe airflow obstruction. They appear to be largely resistant to the effects of smoke on their airflow. Smokers who are more susceptible to these effects develop various degrees of airflow obstruction, which in some ultimately becomes disabling or fatal. "Susceptibility" is not an all-or-nothing attribute: rather, it appears to be a continuum, where the more susceptible a man is the sooner he will be disabled if he smokes (p 210¹⁹).

Fourthly, stopping smoking will, of course, make little difference to the FEV₁ of a non-susceptible smoker whose lungs are not being much affected by his smoking. But it may make all the difference to a susceptible smoker. A susceptible smoker who stops smoking will not recover lost FEV₁, but the subsequent rate of loss of FEV₁ will revert to normal. This finding is based on a small group of men, but it has been reported by Comstock *et al*²² and is strongly supported by both the low death rate from bronchitis and emphysema among smokers who have given up more than 10 years earlier (observed in the major prospective studies of smoking and health⁹⁻¹¹) and the minor degrees of emphysema found by pathologists in dead ex-smokers.²³⁻²⁵ It is, of course, true that severely affected patients derive little benefit from stopping^{6,7} because the damage already done to their lungs is by then severe, and merely slowing its further development will not restore adequate function. The quantitative aspects of these effects of smoking on FEV₁ are summarised in the table, where the men aged 50-59 at the start of our study were divided into those who did and those who did not have mild airflow obstruction, as indicated by a slightly low FEV₁ for their age and height. The percentages of men with such airflow obstruction were: 0% of lifelong non-smokers; 28% of ex-smokers (some of whom had probably stopped because of moderate disability); 24% of light smokers (less than 15 cigarettes per day); 46% of heavy smokers (15 or more cigarettes per day). The means of the FEV₁ slopes of non-smokers and of ex-smokers (whether obstructed or not) were similar. The non-obstructed smokers had slightly steeper slopes, and the obstructed smokers had much steeper slopes.

Among smokers who have already developed moderate obstruction, the effect of giving up in early middle age will presumably be to make their subsequent rate of loss of FEV₁ approximate to that of the obstructed ex-smokers in the table instead of that of the obstructed smokers. This twofold difference in mean FEV₁ slope may not seem very impressive, but, as indicated by the line in fig 1 marked "Stopped at 45," it can make the difference between a normal lifespan and premature death. The average effect of stopping is, of course, small since most smokers are not very susceptible and so have normal lungs that do not benefit much from stopping smoking. Those who continue smoking until they are disabled (see, for example, the line marked "Stopped at 65") will also derive little benefit. The important finding is that if those who would eventually die from airflow obstruction stop smoking in early middle age then their subsequent rates of loss of FEV₁ will on average be normal, so that most such individuals will keep well, whereas had they gone on smoking until they became short of breath it would have been too late.

Measuring FEV₁ might thus perhaps be used as a screening test to detect susceptible smokers in middle age, when the fact that the test showed them that smoking was damaging their lungs might help to persuade them to stop. (Care would have to be taken not to imply that smoking is safe for those smokers with normal lung function.)

Peak expiratory flow is even quicker and cheaper to measure and so could also be used for screening. The disadvantage of both of these tests for screening (peak flow perhaps even more than FEV₁) is their wide range of normal values. As shown in fig 2, a man whose FEV₁ is near the lower end of the normal range for non-smokers may be at high risk or may be quite free from disease. Such borderline cases could be referred for more detailed lung function tests which might help to discriminate between "low normal" and "low abnormal" FEV₁ values. Preliminary results of a study²⁶ of functional tests to diagnose small airways disease suggest that the best tests for this purpose would be the airflow rate as forced expiration nears completion—the Vmax 25—and the expiratory nitrogen slope, both of which can²⁷ be used as field screening tests.

The real effect of smoking on susceptible smokers may be underestimated by looking only at the mean FEV₁ level in all smokers (or the mean FEV₁/height³ in the total column of the table), as is usually done in prevalence surveys. There are two reasons for this. Firstly, smoking has only a small effect on not-very-susceptible smokers, but they, being in a majority, conceal the more severe effect on the most susceptible minority. Secondly, we found that smokers with symptoms tend to cut down their cigarette consumption, so that many of those who are most susceptible, and thus most severely affected, appear among the lighter smokers or the ex-smokers.

EFFECT OF MUCUS HYPERSECRETION AND BRONCHIAL INFECTION

Neither mucus hypersecretion nor bronchial infection cause chronic airflow obstruction to progress more rapidly. This was shown in two ways. Firstly, we found that after adjusting for FEV₁ level, smoking, age, and height there was no independent correlation between FEV₁ slope and indices of either mucus hypersecretion (p 94¹⁹) or bronchial infections (p 87¹⁹). This suggests that neither can play any causal part in accelerating the development of chronic airflow obstruction. Since this was a surprising finding, we sought confirmation by looking at changes of FEV₁ level in relation to changes in expectoration and to episodes of bronchial infection in individual men, and no consistent or significant effects were found. The loss of FEV₁ that an individual man suffered from one six-monthly survey to the next was on average the same if a chest cold, chest illness, or attack of sputum purulence intervened as if it did not (p 91¹⁹). We are forced to conclude that neither mucus hypersecretion nor bronchial infections, as we measured them, play any substantial part in actually causing irreversible airflow obstruction. Moreover, the chief anatomical site of chronic mucus hypersecretion (the main bronchi) is different from the (peripheral) usual chief site of fatal airflow obstruction. We therefore feel that chronic airflow obstruction and chronic hypersecretion should cease to be viewed as closely related disease entities (p 141¹⁹). Both are caused by smoking, but they are otherwise largely unrelated conditions, chronic phlegm production being much less important. The terminology that refers to both conditions as one form or another of "chronic bronchitis" is unfortunately sanctioned by usage, but may lead to confusion: those terms that unmistakably refer to either the obstructive or the hypersecretory disorder are preferable. Infective processes are related strongly only to the hypersecretory disorder. But can we really dismiss infective processes as early causes of chronic airflow obstruction? Our negative evidence is very strong, and is supported by clinical studies,²⁸⁻²⁹ while positive evidence of any effect

Mean FEV₁ 1961-9 and FEV₁ slope 1961-9 according to smoking habits among men with and without mild obstruction* who were aged 50-59 on entry to study. Data for men in late middle age are tabulated because health benefits obtained by giving up early in middle age depend on subsequent rates of loss later in middle age (ref 19; table G1)

	With mild obstruction*			Without mild obstruction*			Total		
	% of such men	Mean FEV ₁ /height ³ (cl/m ³)	FEV ₁ slope ± 1SE (ml/year)	% of such men	Mean FEV ₁ /height ³ (cl/m ³)	FEV ₁ slope ± 1SE (ml/year)	% of such men	Mean FEV ₁ /height ³ (cl/m ³)	FEV ₁ slope ± 1SE (ml/year)
Lifelong non-smokers	0			100	65	-42 ± 6	100	65	-42 ± 6
Ex-smokers, 1961-9	28	44	-37 ± 8	72	62	-30 ± 5	100	57	-32 ± 5
Light smokers (average <15 cigarettes/day)	24	41	-62 ± 5	76	62	-42 ± 3	100	57	-47 ± 3
Heavy smokers (average ≥15 cigarettes/day)	46	43	-80 ± 6	54	60	-55 ± 6	100	52	-66 ± 4
All men	29	42	-64 ± 3	71	62	-42 ± 2	100	56	-48 ± 2

*The age-standardised FEV₁/height³ was defined, in units of cl/m³, by (mean FEV₁ 1961-9)/height³ + 0.5 (age in 1965-60), and a cut-off point of 50 cl/m³ was then imposed to define "mild obstruction." This cut-off point represents very mild obstruction indeed, for in a man of 1.71 metres aged 60 it would be 2.5 litres, and even a small percentage of lifelong non-smokers would, in a larger series fall below it.

is virtually non-existent. When challenged to produce evidence to support his contrary opinion,³⁰ the editor of the *British Medical Journal* could produce no data, just published opinions^{21 31}—one of them being that of the MRC committee which organised the present study to test its opinions. If infections are an important cause of irreversible airflow obstruction, it should not be difficult to show this, but it has never been successfully done.* We suggest that those who disagree with, or want more details of, our present conclusions should consult the monograph in which our results are more fully set out and discussed.¹⁹ If it is felt that some point would be clarified by a tabulation or correlation which has not been presented in our monograph, RP can probably provide this quite easily on request, especially if full and precise details of just what is wanted are specified.

The future

Our study has emphasised the importance of smoking in causing airflow obstruction and shown how it might be possible to detect susceptible smokers in time to prevent disability, but many problems remain. What is the basis of susceptibility? It does not seem to lie in overt allergy, for we found no correlation between FEV₁ slope and either sputum eosinophilia or a history of allergic illnesses. Nor does height increase susceptibility, as might be expected from mechanical stresses in the lung, for we, and Cole,³² found that percentage losses of FEV₁ as men of different heights get older are similar. Is susceptibility in any way analogous to α_1 -antitrypsin deficiency or due to quantitative differences in leucocyte proteolytic enzymes? Can it be induced by infections in childhood that are associated with impaired lung function?

What causes of obstruction other than smoking are there? The British decline in certified death rates from bronchitis and emphysema over the past three or four decades while cigarette smoking has increased indicates (unless these changes are chiefly due to differences in death certification practice for infective disease) that some important cause or agent must have been declining in severity. Was this just air pollution? The large social class gradient of mortality, which was (unless this, too, was severely biased by nosological artefacts) present long before there was any social class gradient in smoking, suggests that there must be causes related to style of living that have not yet been identified.

Our study has disposed of some misconceptions, and provided a simpler picture of the natural history of airflow obstruction. New ideas to be tested by prospective epidemiology will now be needed to further our understanding of this common, distressing, and often fatal disorder.

The study whose main findings we have described was financed by the Medical Research Council and organised by Dr Cecily Tinker. The analysis, in Sir Richard Doll's department, involved extensive use of the Science Research Council's Atlas Computing Laboratory. We are grateful to the unions, management, and men who participated for eight years, and to many others, particularly Mr I D Hill, Mrs H Joyce, Ms G Mead, Professor G A Rose, Dr F E Speizer, and Ms M Stuart.

Requests for reprints should be addressed to Mr R Peto.

*Clinical histories given by patients with severe airflow obstruction often suggest an onset of the obstruction at the time of a particular chest illness.³ An example is one man in our study who was admitted to Hammersmith Hospital in cor pulmonale (p 236¹⁹). On admission to hospital in 1967, he reported that he had been quite fit, even rowing with an amateur rowing club, until 1964, when he had pneumonia, but that after this he had had chronic disabling shortness of breath. Our study records, however, showed that his FEV₁ had already been only 1 litre in 1961 and that in 1961 he had said that he could not keep up with other men when walking on the level because of breathlessness. His FEV₁ declined steadily from 1 litre in 1961 to 0.2 litre just before his death in 1970 in respiratory failure. There was thus no sudden change in FEV₁ level after his pneumonia in 1964. Perhaps this pneumonia increased ventilatory demand on exertion but it did not increase his airflow obstruction. It is obvious how the history given in 1967 could mislead a clinician about the effect of this man's pneumonia on the development of airflow obstruction.

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What might be the cause of pain in the testicles after intercourse?

I think this man probably has referred pain from the lower lumbar spine. Lower lumbar disc pain can be referred to the groin or lower abdomen, including the hemiscrotum of the side in question. Clearly, we are assuming here that there is no clinical abnormality in the testis, cord, or hernial orifice, and that there is no abnormality on abdominal and rectal examination. There may be a history of backache or his testicular pain may be induced by strenuous exercise of the back under other circumstances. Treatment should be on the usual lines for a lumbar disc lesion and in this case his wife should take a more active role during intercourse until such time as a clinical improvement is reached.

My sphygmomanometer has a cuff calibrated to enable one to make reductions on the observed dial reading according to the circumference of the patient's arm. I recently saw a patient who was applying for life insurance whose uncorrected diastolic reading was 100 while his corrected diastolic reading was 89. Is one justified in giving the lower reading—that is, with cuff correction—when completing a life insurance report?

I do not believe that one would be justified in giving only the "corrected" lower reading of the blood pressure after measuring the blood pressure in a patient with large arm. It would be reasonable to give the actual blood pressure reading plus the suggested correction for arm size (clearly indicating that this was an extrapolated number and not the one that was actually measured).