

Bronchial gland dimensions in coalminers: influence of smoking and dust exposure

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ABSTRACT Bronchi from 94 coalminers were examined for enlargement of the mucous glands. The measurements used were the mean and maximum ratios of gland thickness to wall thickness, the mean acinar diameter, and the ratio of the gland area to the area of the non-cartilaginous portion of the bronchial wall. The relationships between these measurements and the subjects' smoking habits and their past exposure to dust in the coal mines were analysed. Smoking was found to be significantly related to mean and maximum gland-to-wall ratios. In addition, coalmine dust exposure was found to be significantly related to maximum gland:wall ratio. No significant relationships were found between gland dimensions and pneumoconiosis or dust retained in the lung. These results lend support to the view that irritants encountered in an occupational environment may play an important part in the development of hypersecretion of mucus.

Measurement of the dimensions of the bronchial mucous gland was pioneered by Reid,¹ who found that the proportion of mucous glands in the bronchial wall was increased in patients with chronic bronchitis. Subsequent work has indicated that this association between enlargement of mucous glands and bronchitis is less clearcut than was initially suggested. The relation between chronic bronchitis and smoking is well known and smoking has been shown to be associated with enlargement of the bronchial glands.²⁻⁵

Rae *et al*⁶ showed for the first time an increase in the prevalence of bronchitis among a group of coalface workers as their cumulative exposure to respirable dust increased. Studying gold miners, Wiles and Faure⁷ found a relation between the dust inhaled and both chronic bronchitis and airways obstruction.

In a necropsy study of lungs from Australian coalminers, Leigh *et al*⁸ found no association between the proportion of mucous glands in the bronchial wall and either smoking or time spent in various working environments. Other studies on coalminers have not been concerned with the role of occupational factors in the development of gland enlargement. Ryder *et al*⁹ examined differences between miners with and without radiographic signs of coalworkers' pneumoconiosis and found none, while Glick *et al*¹⁰ showed that gland enlargement was related to clinical symptoms of bronchitis in a mining population but took the analysis no further.

The aim of the present work was to elucidate the relation

between exposure to coalmine dust and mucous gland enlargement.

Methods

We examined the bronchi from 94 deceased coalworkers. This group was selected, from 500 men included in a necropsy study of coalworkers' lungs,^{11 12} on the basis of a consistently reported smoking history. The main source of material was the pneumoconiosis medical panels of the Department of Health and Social Security. The men had all participated during life in the National Coal Board's pneumoconiosis field research, the objectives and research methods of which are well documented.^{13 14}

Smoking histories were obtained from the pneumoconiosis medical panels' notes. Cases were excluded if the reported smoking habits had varied over the years; for non-smokers in particular we adhered rigorously to this principle of consistency. The cases were divided into four groups according to smoking habit, as follows: non-smokers, ex-smokers (five years or more), light smokers (≤ 10 cigarettes/day), and medium and heavy smokers (> 10 cigarettes/day) (table 1). As only two men had admitted to smoking more than 20 a day, we did not have a category of heavy smokers. Although the miners examined do not represent a true cross-section of British coalworkers they do cover the range of pulmonary diseases associated with coalmining.

The mean age of the 94 men used in this study was 67.2 years, 80% of them having been over 60.

From the measurements of dust concentration and relevant manpower records collected during the pneumo-

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Table 1 Numbers of men in each smoking group

Smoking group	No of men	Code for group
Non-smokers	17	0
Ex-smokers	23	1
Current smoker, ≤ 10 cigarettes/day	29	2
Current smoker, > 10 cigarettes/day	25	3

coniosis field research we calculated the cumulative exposure to coalmine dust for each man. We included only exposure to "respirable" dust, as described by Hamilton and Walton;¹⁵ a method for calculating this has been described.^{14, 16} Cumulative dust exposure is expressed as gram-hours per cubic metre of sampled air (gh/m³).

Measurements of the lung dust content were made routinely on the 500 men included in the necropsy studies of coalworkers' lungs.^{11, 12} A description of the extraction and analysis of lung dust is given by Davis *et al.*¹⁷ The amount of dust retained in the lungs was expressed (a) as the total weight of dust retained by the lung and (b) as the weight of dust in 100 g of blood-free, dust-free tissue.

The lungs from the 94 miners were examined for evidence of coalworkers' pneumoconiosis. Three broad groups (M, F, and PMF) were defined on morphological grounds from naked-eye observations.¹² The M (macule) group comprised lungs with dust deposits, but no macroscopic evidence of fibrosis. Radiographs in these cases rarely showed evidence of pneumoconiosis. Lungs in the F or fibrotic-nodule group had one or more "nodules" with a solid, palpable centre of at least 1 mm but less than 10 mm in diameter. If the lungs contained one or more fibrotic lesions 10 mm or more in diameter the case was assigned to the progressive massive fibrosis (PMF) group.

HISTOLOGICAL EXAMINATION OF BRONCHI

Where possible, blocks were taken from the following six sites in the upper bronchial tree for histological examination: left and right main bronchi, left and right upper lobe bronchi, left lower lobe bronchus, and right bronchus intermedius.

In all, 496 sections of bronchi were examined, of which 209 were complete cuffs. Histological sections were cut at 8 μm, stained for elastic by Weigart's method, and counterstained for muscle and collagen by van Gieson's method. The following measurements were made (the first two on all 496 sections and the others where complete cuffs of bronchi were available):

(1) Ratio of gland thickness to wall thickness. This was measured according to the method of Reid.¹ Up to six measurements were made at positions where the epithelial basement membrane ran roughly parallel to the underlying cartilage. Two values of the ratio were used in the analyses—a mean value and a maximum value calculated as the mean of the highest ratios recorded at each site.

(2) Mean diameter of glandular acini. These were measured at an objective magnification of 40 diameters. A maximum of 12 readings per section were taken. The largest acini were measured as advocated by De Haller and Reid.¹⁸ Where the section under examination was a complete circumference of bronchial wall or nearly so, then three acini were measured in each of the four quadrants of the cuff. If the section was incomplete it was scanned from one end to the other, the largest 12 acini being measured on the traverse.

(3) Gland area.

(4) Cartilage area, cartilage being judged to extend to the inner aspect of the perichondrium.

(5) Total bronchial wall area. The bronchial wall was delineated by the mucosal basement membrane of the luminal surface and by the obvious junction between connective tissue associated with the bronchus and the surrounding tissue.

Measurements 1–3 were made with the aid of a drawing tube attachment to the microscope, which enabled the image of a pen to be seen on the histological section. The appropriate lengths and areas were recorded on paper and subsequently measured.

Measurements of cartilage area and total bronchial wall area were performed on tracings taken from the projected image of the bronchus. The areas were computed with a point count grid.

The gland area was expressed as a percentage of the total wall area less cartilage:

$$\text{Gland index} = \frac{\text{gland area} \times 100}{\text{total wall area} - \text{cartilage area}}$$

ANALYSIS OF DATA

The method of analysis used to investigate the relationships was linear regression. The first models fitted had three exponent variables. The first two variables, smoking group and dust exposure, remained the same and the third was changed. Smoking group and dust exposure were not associated with each other ($r = 0.04$). The third variable was either total weight of lung dust, weight of dust in 100 g of blood-free and dust-free tissue, age, or pathological group. Age was included as it could be argued that any effect supposedly due to dust exposure or lung dust content might simply be accounted for by age. Pathological group was included as Davis *et al.*¹¹ showed that the total weight of dust retained in the lung increased through the pneumoconiosis groups (M→F→PMF). Thus any relationship with lung dust might also hold good for the pathological group. For these analyses ex-smokers were excluded as the models did not accommodate changes in smoking habit.

From table 2 dust exposure is seen to be strongly correlated with total lung dust and lung dust in 100 g of tissue and the two expressions of lung dust to be highly correlated with each other. For this reason these variables

Table 2 Correlation coefficients between explanatory variables

	Smoking group	Total lung dust	Lung dust in 100 g tissue	Age	Pathological group
Exposure	-0.03	0.47*	0.40*	0.09	0.17
Smoking group	1.00	-0.13	-0.15	0.08	-0.16
Total lung dust		1.00	0.85	0.19	0.38

* $p < 0.01$.

were tested separately when we were looking for associations between them and the measures of bronchial wall dimension.

Results

The coefficients of the regression equations are shown in tables 3 and 4. Smoking was found to be significantly associated with both mean and maximum gland:wall thickness ratios and gland acinus diameter ($p < 0.05$, 0.01 , 0.01 respectively). Dust exposure was significantly associated ($p < 0.05$) with maximum gland:wall ratio and age with acinar diameter. Gland index was not associated with any of the explanatory variables. We could not show

Table 3 Values for the components of the regression equations: mean and maximum gland:wall ratios (GWR), mean acinar diameter (mean AD), and gland index versus smoking group, dust exposure, pathological group, and age (number of cases 71, ex-smokers excluded)

(a) Pathological group included as a variable

Bronchial measurement	Regression coefficients				
	Constant	Smoking group	Dust exposure	Pathological group	r
Mean GWR	34.84	1.53 [†]	0.0077	0.63	0.33 [‡]
Max GWR	39.52	2.10 [‡]	0.0135 [†]	1.21	0.41 [‡]
Mean AD	54.67	2.62 [‡]	0.0043	1.00	0.46 [‡]
Gland index §	20.95	0.17	0.0058	-0.92	0.15

(b) Age included as a variable

Bronchial measurement	Regression coefficients				
	Constant	Smoking logical	Dust exposure	Age	r
Mean GWR	34.67	1.49 [†]	0.0079	0.03	0.32 [‡]
Max GWR	38.44	2.01 [‡]	0.0140 [†]	0.06	0.40 [‡]
Mean AD	42.18	2.46 [‡]	0.0037	0.23 [†]	0.50 [‡]
Gland index §	26.86	0.21	0.0058	0.10	0.17

(c) Only two variables included

Bronchial measurement	Regression coefficients			
	Constant	Smoking group	Dust exposure	r
Mean GWR	36.26	1.50 [†]	0.0080	0.32 [‡]
Max GWR	42.23	2.03 [‡]	0.0143 [†]	0.40 [‡]
Mean AD	56.92	2.57 [‡]	0.0048	0.45 [‡]
Gland index §	18.92	0.25	0.0050	0.11

[†] $p < 0.05$.[‡] $p < 0.01$.

§ Measured on complete sections only—61 cases included.

Table 4 Values for the components of the regression equations: mean and maximum gland:wall ratios (GWR) and gland index versus smoking group, total lung dust, and dust in 100 g of blood-free, dust-free tissue (number of cases 71, ex-smokers excluded)

(a) Total lung dust included as a variable

Bronchial measurement	Regression coefficients			
	Constant	Smoking group	Total lung dust	r
Mean GWR	38.2 [†]	1.45 [†]	0.03	0.28 [‡]
Maximum GWR	46.0	2.06 [‡]	0.03	0.33 [‡]
Gland index §	19.2	0.37	0.05	0.12

(b) Lung dust in 100 g of tissue included as a variable

Bronchial measurement	Regression coefficients			
	Constant	Smoking group	Lung dust in 100 g of tissue	r
Mean GWR	38.2	1.46 [†]	0.04	0.28 [‡]
Maximum GWR	46.4	2.05 [‡]	0.01	0.33 [‡]
Gland index §	18.7	0.42	0.11	0.16

[†] $p < 0.05$.[‡] $p < 0.01$.

§ Measured on complete sections only—61 cases included.

any difference between smoking groups in their associations between the anatomical measurements and dust exposure. Simple linear regression analysis showed no association between measurements performed on the major bronchi and the coal, ash, and quartz components of the lung dust content.

Average values for the maximum gland:wall ratio for cases divided by smoking group and levels of dust exposure are shown in table 5. The values were generally higher in the smoking groups than the non-smokers. In the light smokers the values were higher for those with higher dust exposures and the difference between high-exposure and low-exposure groups nears significance at conventional levels ($p = 0.051$).

The results indicate that: (a) smoking contributes significantly to the enlargement of glands as measured by the mean and maximum gland:wall ratios and to the enlargement of the tubules (acini) of the glands; (b) dust exposure contributes to the enlargement of mucous glands as measured by maximum gland:wall ratio; (c) dust retained in the lung (total or percentage of tissue), age, and pneumoconiosis (pathological) group are not associated with overall dimensions of the mucous gland dimensions; (d) gland area as a percentage of non-cartilaginous wall area is not significantly related to dust exposure or smoking.

Discussion

Previous studies have found an association between enlargement of bronchial glands and smoking^{2,4,5} and in this respect the present study does not differ. The association between the maximum ratio of gland thickness to wall thickness and coalmine dust exposure is, so

Table 5 Average values of maximum gland:wall ratio for cases divided by smoking group and dust exposure (numbers in parentheses indicate cases in subgroup)

Smoking group	Dust exposure (gh/m ³ *)			All exposures
	<200	200 <400	≥400	
Non-smokers	45.2 (5)	45.9 (9)	48.1 (3)	46.1 (17)
SD	4.0	(8.8)	15.0	8.5
Smokers				
(≤ 10 cigarettes/day)	48.0 (8)	50.1 (17)	56.6 (4)	50.4 (29)
SD	7.9	6.8	4.0	7.1
Smokers				
(> 10 cigarettes/day)	50.4 (7)	52.6 (16)	52.7 (2)	52.0 (25)
SD	9.3	5.2	1.4	6.3
Ex-smokers	46.3 (8)	48.5 (12)	42.4 (3)	46.9 (23)
SD	6.4	8.2	5.3	7.3
All cases	47.6 (28)	49.8 (54)	50.3 (12)	49.2 (94)
SD	7.2	7.2	9.2	7.5

*Gram-hours per cubic metre of sampled air.

far as we are aware, a new observation. It is a result that supports the idea that irritants encountered in an industrial environment may be important in the development of chronic hypersecretory bronchitis, as suggested by several epidemiological studies.^{6 19-21}

The lack of association with the other indices of gland enlargement may be explained by the fact that for most of the people examined in the study exposure to coalmine dust had ended more than five years before death and for one-third 10 or more years before. These men could be regarded as ex-dust-breathers. The number of men still in employment at the time of death was 24 and this group was considered too small to analyse the results separately.

There is, however, some experimental evidence suggesting that the bronchial mucous glands revert to their original size after the stimulus to secrete is removed. In work where the stimulus was of an acute nature the gland enlargement seemed to be due to the swelling of the mucous cells by their secretions.²² In chronic bronchitis the mucous gland enlargement is largely due to an increase in cell numbers²³ and what happens to the glands after the removal of an irritant is unknown, though some diminution in volume is likely. The lack of association between the area gland index and smoking and dust exposure may in part be due to a reduction in gland volume after regular inhalation of dust ceased. By contrast, the association between the maximum gland: wall ratio and these factors may be due to some residual, localised gland enlargement. These findings are otherwise difficult to explain. If sufficient data from men who were still employed at the collieries at their time of death can be collected then it will be interesting to compare them with those from the current study.

A second limitation may be that for only 44 of the men were there measured exposure data covering 20% or more of their working life, their previous exposure having been estimated.¹⁴ Exposure measurements for the pneumoconiosis field research did not start until the mid 1950s, by

which time the men in the study had been working for an average of 30 years. Earlier exposure data were not available for the study group but the exposure levels were likely to have been higher as dust suppression was not widespread before 1952.²⁴

A further factor may be that the data on dust exposure we used are a measure of the "respirable" dust. This is only a fraction of the dust to which a man has been exposed as it excludes particles greater than 7.1 μm diameter and half of those over 5 μm. Most of the larger particles are removed from the air by the mouth or nose, the rest being deposited in the trachea and large bronchi. Animal experiments have shown that, in addition to local irritation, mucus secretion from the tracheobronchial glands can be stimulated by irritation of the nasopharynx.²⁵ If this is the case in man then a stronger relationship may exist between "bronchitis" and the fraction of airborne dust that is inhaled and subsequently impinges on the upper airways.

No relationships between mucous gland dimensions and lung dust or pneumoconiosis could be shown. With regard to pneumoconiosis, the present results are in accordance with those of Ryder *et al.*⁹ As lung dust content and pneumoconiosis are likely to be related to respirable dust exposure and mucous gland enlargement to inhalable dust the lack of association is not unexpected.

Although other studies have found that the overall dimensions of the bronchial glands tend to be enlarged in elderly people^{26 27} no such effect was noted in this study. The apparent inconsistency between the association of age with the diameter of the larger acini and the absence of such an association with the proportion of gland in the bronchial wall is explained by the observation that gland enlargement is due to the proliferation of the gland tubules and not to increase in the size of those already present.²³ Acinar diameter and the ratio between gland thickness and wall thickness (or gland index) are therefore measurements of different aspects of the bronchial mucous glands.

As the work reported here has been concerned only with the large bronchi, we can make little comment on the idea that "industrial" bronchitis is confined to the upper airways, as suggested by Morgan.²⁸ It does, however, support the view that irritants encountered in an occupational environment do play an important part in producing hypersecretion in the proximal bronchi.

This work formed part of a thesis accepted for the degree of M Phil by the University of Edinburgh. We are grateful to Mr JS Chapman and Mr H Tully and their colleagues for help with the work, and to Mrs Paula Collings for statistical guidance. We acknowledge gratefully the financial support of the National Coal Board and the Commission of the European Communities.

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strong state. It is not therefore satisfactory to use a gentler technique for its removal.

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Notices

Annual meeting of the European Academy of Allergology and Clinical Immunology

The annual meeting of the European Academy of Allergology and Clinical Immunology 1984 will be held in Brussels on 16-19 May. The themes will be the immunological aspects of asthma, occupational allergy, and immunomodulation, with a postgraduate course on the treatment of allergic diseases. Further information from Professor R Pauwels, Department of Respiratory Diseases, De Pintelaan 185, B 9000 Ghent, Belgium.

Applied respiratory physiology

A course in applied respiratory physiology will be held at the Royal Postgraduate Medical School, Hammersmith Hospital, from 26 to 30 March 1984. The topics will include mass spectrometry, body plethysmography, non-invasive monitoring, nocturnal hypoxaemia, control of breathing, regional lung function, airflow obstruction, diffusion and clinical pulmonary function testing. Visiting speakers will include Dr NR Anthonison (Canada). Application forms and further details are available from: School Office (SSC), Royal Postgraduate Medical School, Hammersmith Hospital, London W12 0HS.

Ninth International Cystic Fibrosis Congress

The medical/scientific programme of the Ninth International Cystic Fibrosis Congress will be held in Brighton from 12 to 15 June 1984. Papers on any aspect of cystic fibrosis may be submitted (closing date for abstracts 31 January 1984). The principal plenary sessions will include presentations on: prenatal diagnosis and carrier detection, pathophysiology (characteristics of the cystic fibrosis cell,

calcium metabolism, glycoproteins of cystic fibrosis mucus), the changing management of cystic fibrosis (aerosol antibiotics, the role of exercise, supplemental nutrition), and the basic defect. There will be an allied health professionals programme that will include parallel sessions on social aspects of cystic fibrosis, dietetics and nutrition, and physiotherapy. Further details are obtainable from the Congress Office, Cystic Fibrosis Research Trust, Alexandra House, 5 Blyth Road, Bromley, Kent BR1 3RS.

Corrections

Bronchial gland dimensions in coalminers: influence of smoking and dust exposure

Drs Douglas, Lamb and Ruckley in their paper (*Thorax* 1982;**37**:760-4) refer to work by Dr WKC Morgan and colleagues (*Br J Ind Med* 1978;**35**:285-91). It has become clear that Dr Morgan's work has been misquoted: Dr Morgan did not state that industrial bronchitis is confined to the upper airways. The authors regret this error.

Microbiological investigation of farmer's lung disease in Somerset 1963-83

In the abstract by JP Anderson and FB Greatorex (September 1983, p 709), "acute" in the 6th line from the end should be "chronic"—that is, "In chronic farmer's lung disease. . . ."