PNEUMOCONIOSIS IN CARBON ELECTRODE MAKERS

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In the absence of adequate preventive measures the manufacture of carbon electrodes is attended by a considerable dust hazard. The present paper is based on a study of the clinical, radiological, and pathological changes resulting from inhalation of this dust, which is derived from crushed coke and anthracite.

An account is given of the findings in a clinical survey of 15 men who had been employed for at least 10 years in manufacturing carbon electrodes. Four of these men were suffering from complicated and five from simple pneumoconiosis.

In addition, the findings in three necropsied cases (two complicated and one simple) are recorded in detail. Bacteriological examination of the lungs and analysis of the lung dust was carried out in the two cases of complicated pneumoconiosis.

It is shown that carbon electrode makers may develop simple pneumoconiosis with focal emphysema and that this may be complicated by the development of massive fibrotic lesions. Both the simple and the complicated pneumoconiosis are indistinguishable from the corresponding conditions in other coalworkers.

Quartz was almost entirely absent from the lung dust of the two necropsied cases with massive fibrosis and in one of these cases virulent tubercle bacilli were shown. The significance of these findings is discussed in relation to the aetiology of progressive massive fibrosis. While it is evident that they are incompatible with the "silica" theory they provide some limited support for the "tuberculosis" theory.

The manufacture of carbon electrodes involves the production of much dust which must inevitably be inhaled unless efficient dust control is achieved; the dust consists mainly of coal and coke particles. It is the purpose of this paper to present clinical, radiological, and pathological evidence that prolonged exposure to such dust causes the development of pneumoconiosis indistinguishable from that of coalworkers in both its simple and its complicated varieties. Two necropsied cases with complicated pneumoconiosis afforded the opportunity to compare the characteristics of the massive fibrotic lesions and their contained dust with those of the corresponding lesions in cases of complicated pneumoconiosis of other coalworkers. The findings are discussed in relation to the possible aetiology of this condition.

The Process

In the process of carbon electrode manufacture at the factory with which we are concerned the raw material is principally a mixture of coke and anthracite. After pulverization these are mixed with

a binding agent, usually pitch, and then pressed into the desired shapes. The processes involve crushing, milling, and sieving the coke, anthracite, and scrap electrode material, mixing with hot pitch as the binding agent, shaping in presses, baking the shapes in furnaces, and lastly, fettling their surfaces with hand or pneumatic tools. At almost all stages of the process very large quantities of dust are produced, with which the measures of dust suppression in use until 1945 were quite unable to cope efficiently. At the end of the war, long delayed plans for rebuilding and improving the plant were put into effect and by 1948 rather more than half of the works had been modernized by installing new plant to enclose the dusty processes, to transport materials mechanically, and to provide efficient local exhaust ventilation where required. Since then further improvements have been carried out to plant and premises and, in addition, an excellent scheme of medical supervision of the workers has been instituted, which includes initial and periodical medical examinations. Periodical checks on environmental conditions by dust counting are also carried out.

Clinical Survey

In 1948, the possibility of a pneumoconiosis risk in the process was suggested to one of us (A.T.D.) by Dr. J. M. M. Jamieson (now in Kenya). Working conditions were investigated, and clinical and radiological examinations were carried out on 15 workers. These 15 men comprised all those on one shift who had been employed for 10 years or more on the process. Nine cases of pneumoconiosis were found; five of these cases were in the category of simple pneumoconiosis and the remaining four cases were of the complicated variety. The detailed findings are given in Table 1. The radiological classification adopted conforms to that evolved by the M.R.C. Pneumoconiosis Research Unit in Cardiff (Fletcher, Mann, Davies, Cochrane, Gilson, and Hugh-Jones, 1949).

TABLE 1

PARTICULARS OF 15 CARBON ELECTRODE WORKERS INCLUDED IN THE CLINICAL SURVEY

Case	Age (years)	Exposure (years)	Radiograph of Chest
1 (S.H.)	52	28	Normal
2 (G.B.)	34	16	Normal
3 (W.R.)	35	10½	Normal
4 (N.L.)	60	25	Increased vascular markings
5 (J.M.)	57	28	Increased vascular markings
6 (D.M.)	59	37	Emphysema
7 (A.M.)	60	34½	Simple pneumoconiosis, Cat. 2
8 (P.D.)	46	16	Simple pneumoconiosis, Cat. 2
9 (D.W.)	34	13	Simple pneumoconiosis, Cat. 2
10 (S.M.)	54	32	Simple pneumoconiosis, Cat. 2
11 (J.D.)	64	28½	Simple pneumoconiosis, Cat. 2
12 (H.C.)	62	39	Complicated pneumoconiosis, 2A 1/0
13 (R.K.)	64	18	Complicated pneumoconiosis, 3A 2/1
14 (J.M.)	65	30	Complicated pneumoconiosis with emphysema, 3C 4/1
15 (G.G.)	61	19	Complicated pneumoconiosis, 3B 3/2

Age and Duration of Exposure.—The ages of the 15 workers ranged from 34 to 65 years (mean 53·8) and experience in the process from 10½ to 39 years (mean 25·6). The mean duration of employment of the men whose chest radiographs were classed as normal was 18·2 years; of those with simple pneumoconiosis, 24·8 years; and of those with complicated pneumoconiosis, 26·5 years. As the factory is situated in a non-industrial area, previous occupations were mainly agricultural, horticultural, and soldiering. Only two men had been engaged in previous occupations with a dust hazard, one as a slate dresser for seven years (Case 6, found not to have pneumoconiosis) and the other as a foundry labourer for one year (Case 7, with simple pneumoconiosis). The pneumoconiotic lesions found can therefore be attributed entirely to the dust exposure in electrode making.

Clinical.—Two of the four men with complicated pneumoconiosis gave a history of previous respiratory illness, recent severe bronchitis in one case

(Case 15) and pneumonia and pleurisy 23 years previously in another (Case 12). A third man in this group (Case 13) said that he had been suspected of having pleurisy five years before. Two of these men (Cases 12 and 13) had slight or occasional symptoms consisting of cough, dyspnoea, and sputum; the other two had more severe symptoms, and Case 14 had been given a light job because of dyspnoea. This man showed hyperresonance on percussion, with a diminished area of cardiac dullness and pleural friction at the right base. In Case 12, sonorous and sibilant rhonchi were heard, but in the other two cases there were only basal crepitations. Chest expansion was notably reduced in all four men, the mean being 1·4 in. (3·5 cm.).

None of the five men with simple pneumoconiosis had a history of previous respiratory illness and two of them (Cases 7 and 9) were quite free of chest symptoms. One man (Case 11) was considerably disabled by dyspnoea, but this was probably mainly due to auricular fibrillation; the other two men had only mild symptoms. No abnormalities as regards percussion and auscultation were encountered in three cases (Cases 7, 9, and 11) and in the other two there was little apart from basal crepitations. The mean chest expansion in this group was 1·6 in. (4·5 cm.).

In the group of six men without pneumoconiosis, none gave a history of previous respiratory illness; three had slight occasional cough, two had occasional sputum, and one was doubtful about the presence of slight dyspnoea. Two of this group (Cases 1 and 5) had basal crepitations and, in addition, one of them had signs of emphysema. The mean chest expansion was 2·2 in. (5·6 cm.).

Radiological.—The appearances of the radiographs were within normal limits in three patients (Cases 1, 2, and 3), showed increased vascular markings in two (Cases 4 and 5), and suggested the presence of emphysema in one (Case 6). The five cases showing the changes of simple pneumoconiosis were Cases 7, 8, 9, 10, and 11, and the four showing complicated pneumoconiosis were Cases 12, 13, 14, and 15.

Details of Three Necropsied Cases

One of the men (Case 15) examined in the course of the clinical survey and found to be suffering from complicated pneumoconiosis has since died. A necropsy was performed on this man and on two other carbon electrode makers from the same factory who were not included in the survey; one of these (Case 16) was a case of complicated pneumoconiosis and the other (Case 17) a case of simple pneumoconiosis. A full account of the clinical and pathological findings in all three cases is given below

and the results of an analysis of the lung dust in the two with massive fibrosis are also recorded.*

Case 15.—This man, aged 70 when he died in August, 1958, had been engaged in the manufacture of carbon electrodes for 19 years. From 1946 he was incapacitated by his chest condition for some 20 months, resuming lighter work in 1948. In July, 1952, he was considered to be totally disabled by pneumoconiosis, his chief complaint at this time being shortness of breath on exertion which was apparent during examination. He also suffered from an intermittent cough accompanied by sputum. Nutrition was average, chest expansion was poor. On auscultation the breath sounds were high pitched, with prolongation of the expiratory murmur. Air entry was rather poor and a few rhonchi were heard over the lower zones of the chest posteriorly. Radiological examination of the chest showed the appearances of complicated pneumoconiosis, category 3B 3/2 (Fig. 1).

Post-mortem Examination.—The subject was a strongly built, well-nourished, elderly man with considerable pitting oedema of the legs and feet. Dissection showed that the pleural sacs were partially obliterated posteriorly by fibrous adhesions. The trachea and major bronchi appeared normal. The lungs were abnormally heavy and bulky and failed to collapse when the thorax was opened. Large emphysematous bullae were present at the apices and along the anterior margins of the lungs. Posteriorly the lungs showed coarse cicatrization in relation to underlying fibrous masses. Although heavily dust-impregnated the hilar and tracheo-bronchial lymph nodes were only slightly enlarged. The right lung (780 g.) was sliced before fixation, disclosing numerous small, soft, dust-impregnated foci, each attended by a severe degree of focal emphysema. In addition, larger palpable foci up to 1.0 cm. in diameter were sparsely distributed throughout all the lobes. A black fibrotic mass about 6.0 cm. in diameter was present in the parahilar zone of the upper lobe and nearby, in the superior segment of the lower lobe, there was a similar mass measuring 5.0 × 4.0 cm. on hemisection. Both these lesions were fairly well circumscribed and had a slightly compressible rubbery consistency; neither showed central necrosis. The left lung (740 g.) was fixed in the position of inflation by running formol saline into the air passages. Slicing of the lung showed appearances very similar to those described in the right lung: numerous soft dust foci with severe focal emphysema, scattered small palpable foci, and two large fibrotic masses were noted. One of the massive lesions was situated at the apex of the upper lobe and the other at the apex of the lower lobe; both were solid, rubbery, and discrete, measuring about 5.0 cm. in diameter. These lesions are illustrated in the accompanying reproduction of a thin sagittal section of the whole left lung (Fig. 2). In both lungs the pulmonary

FIG. 1.—Case 15. Carbon electrode maker for 19 years. Chest radiograph showing complicated pneumoconiosis, category 3B 3/2.

arteries were extremely atheromatous and ante-mortem thrombus was present in many of the larger divisions, extending more peripherally in the left lower lobe where there was a large red infarct in the lateral basal segment. There was no gross evidence of tuberculosis or of pneumonic consolidation in either lung.

The heart was markedly enlarged due to hypertrophy of both ventricles, particularly the right. The coronary arteries were sclerotic and showed widespread atheromatous degeneration resulting in almost complete occlusion of the anterior descending branch of the left coronary artery. There was myocardial fibrosis but no evidence of infarction. Apart from changes due to chronic venous congestion in the liver and slight surface granularity of the kidneys, other organs showed no notable abnormality.

Histology.—Microscopical examination of the massive dust-impregnated lesions in both lungs showed that they had a fairly uniform structure. This consisted of a connective tissue meshwork with its interstices filled by small aggregates of black dust particles. Because of the density of these aggregates it was impossible to determine whether or not the dust lay free or was contained within macrophages. The arrangement and uniform size of the aggregates suggested that the dust was mainly intracellular (Fig. 3). The bands of connective tissue constituting the meshwork were generally of a loose nature but in places they were densely collagenous and thicker. In addition, these massive lesions were intersected by a few prominent collagenous septa bearing large blood vessels. Some of the pulmonary artery branches in these septa were collapsed and occluded. The appearance of the smaller fibrotic nodules in the lungs was essentially similar to that of the massive lesions, the difference being

*Case 13, another of the men in the clinical survey with complicated pneumoconiosis, has also died. We are indebted to Dr. T. Skeoch of Middlesbrough, who carried out a post-mortem examination, for an account of the findings and for the opportunity to examine histological sections of the lungs. The gross and microscopical appearances of the lungs were indistinguishable from coalworkers' pneumoconiosis with massive fibrotic lesions. Unfortunately the lungs were not retained for dust analysis.

FIG. 2

FIG. 2.—Case 15. Thin parasagittal section of whole left lung showing massive fibrotic lesion in posterior part of the lower lobe. Elsewhere there are numerous small dust foci with severe focal emphysema. Emphysematous bullae are present antero-superiorly.

FIG. 3.—Case 15. This illustrates the structure of the massive fibrotic lesions, consisting of a collagenous meshwork with the interstices filled by aggregates of dust particles. $\times 180$ (Masson).

FIG. 3

FIG. 4.—Case 15. Characteristic small dust deposit with well developed focal emphysema. $\times 40$ (haematoxylin and eosin).

merely one of size. In the spongy parts of the lungs there were numerous, small, stellate accumulations of dust particles, often investing small branches of the pulmonary arteries and sending fine extensions into the interalveolar septa. As in the case of the massive lesions, it appeared that the dust was mostly contained within macrophages and these were enmeshed in a fine connective tissue network. It was confirmed that there was a severe degree of focal emphysema in relation to these small dust aggregates (Fig. 4). The hilar lymph nodes were also heavily impregnated with dust and showed areas of fibrosis. Examination of the various sections in polarized light showed very few doubly refractile dust particles. Evidence of chronic bronchitis was noted and some of the bronchial arteries showed considerable medial hypertrophy.

Bacteriology.—Material from one of the massive lesions in the right lung was examined for the presence of tubercle bacilli. These could not be demonstrated in direct smears nor by guinea-pig inoculation.

Case 16.—This patient died in November, 1956, at the age of 73 years. He had been employed in the manufacture of carbon electrodes for 18 years between 1930 and 1948. Previously he had been a blacksmith for 11

years, and had then served in the Regular Army for 12 years. He had enjoyed good health until he developed pneumonia in 1948 when he finally ceased work; before that date he had had a cough for some time. In 1952 he complained of severe cough at night with expectoration which was sometimes black. Dyspnoea severely limited his walking exercise and he suffered from chest pain, lassitude, and giddiness. Examination showed him to be of spare build though he had formerly been stout. The chest was flattened and expansion was poor. Some impairment of the percussion note was present, especially in the upper zones, and breath sounds were weak with prolongation of the expiratory murmur. Scattered rhonchi were heard throughout both lungs and crepitations at the bases. The chest radiograph showed fine discrete shadows throughout both lung fields with increased lung markings extending slightly into the peripheral zone; large, dense, woolly opacities were present on both sides in the perihilar areas; the classification was 2B 3/2 (Fig. 5). In January, 1953, he was certified as being totally disabled on account of pneumoconiosis.

Post-mortem Examination.—The body was extremely wasted and emaciated and there was severe pitting

FIG. 5.—Case 16. Carbon electrode maker for 18 years. Chest radiograph showing fine discrete shadows and perihilar massive fibrosis, category 2B 3/2.

oedema of the feet and ankles. The left pleural sac was almost completely obliterated by dense fibrous adhesions, but in the right pleural sac there were only some apical fibrous adhesions. The trachea and bronchi contained a large quantity of black, turbid, watery fluid. The right lung was voluminous and heavy (1,370 g.) and was fixed in the inflated position by perfusing the air passages with formal saline. Slicing of the lung then disclosed a large, irregular, black, fibrous mass, approximately 8.0 cm. in diameter, occupying the central, parahilar zone of the upper lobe but also involving the adjacent parts of the middle and lower lobes. In the centre of this massive fibrotic lesion there was an irregular cavity measuring up to 4.0 cm. in diameter filled with black, turbid fluid. The remainder of the right lung was studded with numerous small, soft, dust-impregnated foci attended by a severe degree of focal emphysema. A few fibrous nodules, up to 2.0 cm. in diameter, were also distributed sparsely throughout the lung. Some bronchiectasis was present in relation to the fibrotic lesions. These various features are demonstrated in the accompanying photograph of a sagittal thin section of the right lung (Fig. 6). The left lung was also large and heavy (970 g.) and was cut unfixed so that material might be obtained for bacteriological examination. In the anterior segment of the upper lobe there was a large irregular cavity, containing black, turbid fluid, and measuring 7.0 × 5.0 cm. on hemisection. The wall of this cavity was composed of dense, black, fibrous tissue, and was up to 2.0 cm. in thickness. A similar thick-walled cavity was present in the superior segment of the lower lobe. Other parts of the left lung were studded with numerous soft and some small palpable dust foci, the soft foci being associated with severe focal emphysema. There was general ectasia of the larger bronchi in both lobes but this was most prominent in the

upper lobe. The hilar and mediastinal lymph nodes were moderately enlarged, black, and fibrotic. The heart (200 g.) showed the appearances of brown atrophy and the coronary arteries were moderately atheromatous. Other organs showed no significant abnormality.

Histology.—Microscopical examination of the massive fibrotic lesions in both lungs showed that their structure was basically similar to that of the corresponding lesions in Case 15. They consisted, for the most part, of a close meshwork of collagenous connective tissue in which the interstices were filled by innumerable small (intracellular?) aggregates of black particulate material giving a densely impregnated appearance. In these massive lesions occasional small foci of coagulative necrosis were present (Fig. 7), separated from the investing dust-impregnated collagenous tissue by narrow zones of granulation tissue infiltrated by macrophages. It was possible to discern in the central necrotic areas the outline of pre-existing emphysematous spaces filled with necrotic cells and it was notable that these were quite free of dust particles. Although the usual histological stigmata of tuberculosis were absent, the necrotic material had an appearance reminiscent of caseation, and the presence of moderately numerous acid- and alcohol-fast bacilli confirmed the tuberculous nature of the lesions. The walls of the bronchi in and around the massive lesions were generally fibrotic and the mucosal lining was often replaced by granulation tissue densely infiltrated by plasma cells and macrophages. Many bronchial arteries showed severe degrees of obliterative endarteritis (Fig. 8).

In those parts of the lungs which were not involved in the massive fibrotic lesions there were numerous small focal dust deposits, usually with a crenated or scalloped outline and generally similar to those seen in Case 15. These smaller foci often appeared to be perivascular or peribronchiolar and focal emphysema was a prominent feature in relation to most of them. Throughout both lungs there was, in addition to the above findings, widespread acute bronchopneumonic consolidation. Examination of the dust-impregnated areas by means of polarized light showed that there were very few doubly refractile particles. In the hilar lymph nodes there was extensive dust impregnation and fibrosis similar to that seen in the lungs, but no evidence of caseous necrosis.

Bacteriology.—Tubercle bacilli were cultured from the wall of one of the cavities in the left lung and were shown by guinea-pig inoculation to be virulent.

Case 17.—This man, aged 61, had been employed as a carbon electrode process worker from 1922 until 1939, having previously served in the Army and engaged in agricultural work. He had a further period of service in the Armed Forces from 1939 until 1944, being discharged on account of "epilepsy". He then undertook light labouring duties in an engineering workshop until he finally gave up work because of illness in 1954. A cough which he had had for some time became severe and was accompanied by sputum, occasionally with haemoptysis. Dyspnoea became noticeable and he complained of pain across the chest, giddiness, headache, and lassitude. Clinical examination in January, 1953, showed him to be of fair nutrition and without cyanosis, finger clubbing, or oedema. There were no abnormal signs in the chest,

FIG. 6

FIG. 6.—Case 16. Parasagittal thin section of right lung showing a massive fibrotic lesion in the parahilar region with a large central cavity. The remainder of the lung is studded with innumerable small dust foci attended by a severe degree of focal emphysema.

FIG. 7.—Case 16. A typical example of the sort of necrotic foci which were present in the fibrotic masses. $\times 38$ (reticulin).

FIG. 7

FIG. 8.—Case 16. Severe obliterative endarteritis of arteries in wall of a chronically inflamed dilated bronchus within a massive fibrotic nodule. $\times 80$ (Masson).

FIG. 9.—Case 17. Focal dust deposit with aggregates of dust particles set in a meshwork consisting mainly of reticulin fibres. The dust within the brochiole is clearly intraphagocytic. $\times 160$ (haematoxylin and eosin).

except broncho-vesicular breathing in the upper zones and the presence of extrasystoles. The chest radiograph showed discrete nodulation evenly distributed throughout both lung fields and extending to the periphery. There was diminished translucency over the left upper zone with a dense opacity above the upper pole of the left hilum. A diagnosis of simple pneumoconiosis accompanied by bronchial carcinoma was made. Treatment by deep radiotherapy and administration of nitrogen mustard was carried out, but his condition progressively deteriorated and he died in June, 1956.

Post-mortem Examination.—The upper part of the left pleural sac was obliterated by dense fibrous adhesions. In the left lung there was a large spherical cancerous mass, 7.0 cm. in diameter, apparently arising from the main bronchus to the left upper lobe. The tumour had invaded the parahilar region of both the upper and lower lobes, and metastatic deposits of carcinoma were present in several enlarged mediastinal lymph nodes. Throughout the remaining parts of both lungs there were scattered moderately numerous soft focal aggregates of black dust. These foci measured up to a few millimetres in diameter and were associated with a slight degree of focal emphysema. A metastatic carcinomatous deposit was present in the left adrenal gland. Other organs showed no notable abnormality.

Histology.—The tumour arising from the left upper lobe bronchus was a small-celled anaplastic carcinoma of oat-cell type. The small dust foci consisted of aggregates of small black particles lying in an inconspicuous meshwork of irregularly disposed reticulin and collagen fibres; associated with the black dust there were occasional doubly refractile particles. In all of the foci there was a preponderance of dust over connective tissue (Fig. 9). Focal emphysema was present in relation to many of the dust foci but was not of great severity.

Lung Dust Analysis

An analysis was made of the lung dust in Cases 15 and 16. The methods were essentially the same as those used for coal-miners' lungs by King, Maguire, and Nagelschmidt (1956), except that portions of the right and left lung were analysed separately for carbonaceous matter which is recorded as "coal". Briefly, "coal" was obtained by dissolution of the dried lung tissue with alcoholic KOH and its amount determined by ashing. Mineral matter was estimated as the acid-insoluble portion of the lung ash prepared at 380°C. Carbon and hydrogen determinations were performed on the coal residues which were also examined by x-ray diffraction and the size of the coal particles was determined by electron and optical microscopy. The mineral residues were examined by x-ray diffraction and partial chemical analysis.

According to its carbon/hydrogen ratio the "coal" was found to be a mixture of coke and anthracite. The mineral matter consisted of corundum and mullite with traces of mica and quartz (numerical data are given in Table 2). The size distribution of the dust from Case 16 is shown in Table 3 and illustrated in Figs. 10a and 10b, while x-ray diffraction diagrams of the mineral matter are given in Fig. 11.

TABLE 2
LUNG DUST ANALYSES OF CASES 15 AND 16

	Case 15		Case 16	
	Left	Right	Left	Right
% Dried Lung				
Total dust	32.6	35.4	20.1	23.4
"Coal"	32.3	35.2	19.7	23.0
Mineral residue	0.2		0.4	
Quartz	<0.01		<0.02	
Total SiO ₂	0.05		0.13	
Total Al ₂ O ₃	ND		0.10	
Estimated Amounts in Both Lungs (g.)				
Total dust	102.0		74.9	
"Coal"	101.3		73.5	
Mineral residue	0.7		1.4	
Quartz	<0.03		<0.07	

ND = not determined.

TABLE 3
SIZE DISTRIBUTION OF LUNG DUST FOR CASE 16
BASED ON AN ELECTRON MICROSCOPE COUNT OF 882 PARTICLES

Size (microns)	Number (%)	Mass (%)
0.23	5.0	0.026
0.32	12.2	0.1
0.45	10.4	0.3
0.64	14.3	1.2
0.9	21.2	4.8
1.3	17.3	11.25
1.8	13.7	25.6
2.6	5.1	26.9
3.6	0.5	6.76
5.1	0.2	9.6
7.2 to 10.2	0.1	13.5

Discussion

Carbon electrode workers have been scheduled under the National Insurance (Industrial Injuries) Act since 1951 and it is clear from the findings in the clinical survey detailed above that, in the past at any rate, there has been a considerable risk of the development of pneumoconiosis in this employment. Nine cases of pneumoconiosis were found in the group of 15 men surveyed, and in four of these it was of the complicated variety. It is probable that the incidence of dust disease of the lungs in carbon electrode makers, in the factory with which we are concerned, will fall considerably and eventually disappear as a result of the more effective preventive measures which have now been instituted.

Of the three cases which came to necropsy the third (Case 17) is of interest in so far as the lungs showed simple pneumoconiosis with focal emphysema, resembling in every way the corresponding condition in coalworkers. Obviously it would be unprofitable to discuss the question of a relationship between pneumoconiosis and concomitant bronchial carcinoma on the basis of one such case. The findings in Cases 15 and 16 demonstrate that carbon electrode makers are subject to a form of pneumoconiosis closely resembling in its gross and

FIG. 10.—Electron micrographs of lung dust of Case 16: (a) at lower magnification, and (b) at higher magnification.

Fig. 11.—X-ray diffraction diagrams of (A) mullite, (B) lung residue (acid-washed ash) of Case 16, and (C) corundum.

in its histological appearances the condition of progressive massive fibrosis in coalworkers. In this respect alone these cases are of some importance, but their main significance lies in the light which they throw on the aetiology of massive fibrosis in coalworkers.

The aetiology of massive fibrosis in coalworkers is an unsolved problem and the several opposing views in the literature have been succinctly summarized by Cochrane, Miall, Clarke, Jarman, Jonathan, and Moore (1956). The two main theories are spoken of as the "silica" and "tuberculosis" theories. According to the "silica" theory the confluence and collagenization of coal dust macules, due somehow to the action of silica contained in them, can result in massive fibrosis. The "tuberculosis" theory suggests that the fibrotic masses represent tuberculous lesions modified by the presence of coal dust. In this country the "tuberculosis" theory undoubtedly has the great majority of adherents and has been strongly supported by Gough and the Cardiff "school" (Gough, 1947; Heppleston, 1951; James, 1954). On the other hand, Di Biasi (1949) and many European pathologists adhere to the "silica" theory.

As the lung dusts of coal-miners and even of coal trimmers always contain some quartz, it has been difficult to disprove the "silica" theory. A comparison can be drawn with the occurrence of massive

fibrotic lesions in the lungs of kaolin workers (Hale, Gough, King, and Nagelschmidt, 1956) and of talc workers (Hunt, 1956). But although quartz was absent in these cases, kaolin and talc are plate-shaped silicates very dissimilar to coal dust particles. A search amongst carbon black workers showed early stages of simple pneumoconiosis (Gärtner and Brauss, 1951), but there were no instances of massive fibrosis. Likewise Meiklejohn (1958) found no cases of massive fibrosis among carbon black workers engaged in the manufacture or handling of "nearly pure carbon". In a group of workers handling "mixed carbon", which contained a small amount of mineral impurity, there was one who showed radiological evidence of complicated pneumoconiosis (category 2A). Vaccarezza (1958) has recently described a case of massive pneumoconiosis in a man who had been engaged in shovelling charcoal for several years. The evidence was again purely radiological.

The results of lung dust analyses in our Cases 15 and 16 with massive pneumoconiosis are noteworthy in relation to the validity or otherwise of the "silica" theory. The amounts of carbonaceous matter (coal and coke) found in the lungs were large, although similar figures have been observed in massive fibrosis of South Wales coal-miners, screen workers, and trimmers (King and Gilchrist, 1945; King, *et al.*, 1956). The range of coal percentages of the dried lung in such cases has been from 6 to 29.5. As compared with these, the proportion of mineral matter to coal was considerably lower in the present cases: quartz was almost completely absent and the silica found by chemical analysis was mainly accounted for by mullite and mica which, in the amounts found, are very unlikely to constitute a dust hazard to the lungs. In Table 4 our results are compared with those of lung dust analyses in cases of massive fibrosis of coalworkers from South Wales. The present cases can be explained as a variety of silicosis only if it is further assumed that the quartz which caused it had disappeared from the lungs by solution or lymph transport. We consider that this is very unlikely.

TABLE 4
LUNG DUST ANALYSES OF MASSIVE FIBROSIS OF COALWORKERS WITH MORE THAN 20% COAL (PERCENTAGE DRIED LUNG) COMPARED WITH PRESENT TWO ELECTRODE MAKERS (CASES 15 AND 16)

Occupation	Percentage Dry Lung				Reference
	Coal	Non-coal	Quartz	Total Silica	
Fireman	29.5	1.91	0.50	1.22	King, Maguire, and Nagelschmidt (1956)
Screen worker	26.6	1.78	0.27	0.95	King and Nagelschmidt (1945)
Anthracite collier	26.0	3.05	0.45	1.62	King and Nagelschmidt (1945)
Anthracite collier	23.8	1.53	0.31	0.86	King and Nagelschmidt (1945)
Anthracite collier	23.6	1.20	0.18	0.64	King and Nagelschmidt (1945)
Trimmer	20.8	0.83	0.12	0.44	King and Nagelschmidt (1945)
Carbon electrode maker	21.5	0.4	<0.02	0.13	Present paper (Case 16)
Carbon electrode maker	33.8	0.2	>0.01	0.05	Present paper (Case 15)

A case of massive fibrosis in a graphite grinder, reported by Rüttner, Bovet, and Aufdermaur (1952), is very similar to our Cases 15 and 16: there were large fibrous masses and nodular lesions in the lungs with no gross or microscopical evidence of tuberculosis, and analysis of the lung dust showed the presence of graphite and carborundum but no quartz. No figures were given for the percentage dust content of the lungs nor for the total amount of dust present. Also relevant to our work are the findings of Koelsch (1952), who described advanced stages of pneumoconiosis in carbon electrode makers handling carbon or anthracite with only a trace of silica, mostly in the form of silicates. Diagnosis in these cases was radiological and there was no reference to pathological examination. We believe that our Cases 15 and 16 demonstrate better than any cases hitherto recorded that the presence in the lungs of large amounts of dust which are virtually free from quartz can cause a form of dust disease entirely similar to coalworkers' pneumoconiosis, with small focal and massive fibrotic lesions. The lung dust in our cases was a mixture of coke and coal and the fact that Case 16 may have inhaled some coal dust during his employment as a blacksmith does not alter the significance of the findings.

In Case 16 small foci of acute tuberculous caseation were seen histologically within the areas of massive fibrosis. Although none of these foci was associated with a tuberculous type of tissue reaction their tuberculous nature was confirmed by the demonstration of tubercle bacilli within them. By culture and animal inoculation it was proved that these tubercle bacilli were fully virulent. From their acute nature it is evident that these frank tuberculous lesions were of terminal occurrence, but it does not necessarily follow that they were the result of a secondary exogenous infection. It may

well be that they represented an exacerbation, due presumably to lowered resistance, of a pre-existing infection. If this was so, the finding of tubercle bacilli in the lungs of our Case 16 is additional evidence in favour of the "tuberculosis" theory of the causation of massive fibrosis, especially in view of the virtual absence of quartz. One of us (A.T.D.), however, holds the view that a major factor in the production of massive fibrosis in coalworkers is the occurrence of atelectasis of the alveoli between discrete lesions, thereby forming a dense, airless mass which, due to changes in the vessel walls, is ill supplied with blood and in which infection and necrosis readily occur (Doig, 1956).

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