The Dust Diseases in Great Britain

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The story of dust diseases in Great Britain goes back a long way. E. L. Collis, in his classical Milroy Lectures of 1915, quoted Herbert Spencer as saying that the starting point of human progress was the "localization of industries." There was such a localization in prehistoric factories for the making of flint implements at Grime's Graves, near Brandon in Suffolk, where the flint knappers still use tools like the deer-horn picks of their prehistoric ancestors. In 1914, Collis showed that these workers have a high mortality from silicosis. He said that it is probable that "the starting point of human progress" was associated with at least one form of pneumoconiosis. It has been long known that the dust of flint, which is nearly pure SiO_2, is dangerous to health. Over 200 years ago, Thomas Benson, of Newcastle under Lyme, was granted a patent for grinding flints by a wet method. At that time it was said that a man who ground the flints dry could not live longer than two years.

Our greatest localization of industries took place during the Industrial Revolution, and this was really the starting point of our intensive knowledge about the effects of dust on the lung.

The first notable contribution was that of Pearson (1813), who, after many autopsies, decided that the black pigment in the bronchial nodes and the lungs was due to the inhalation of small particles resulting from the burning of coal, wood, and other inflammable materials. The German pathologists, on the other hand, thought that the pigment came from inside the body. Meiklejohn (1951) has described the hitherto little-known and remarkable contributions of the Scottish physicians of the 1800's on the connection between disease of the lungs and coal dust; he also tells how the incidence of anthracosis diminished with improved ventilation of the mines. The Scottish physicians decided that simple coal dust was comparatively harmless and that stone dust was the really noxious factor.

Charles Turner Thackrah, a physician in Leeds, played a major part in the study of occupational diseases (including the dust diseases) in Great Britain. In his book "The Effects of Arts, Trades, and Professions," the second edition of which was published in 1832, he crystallized the idea that the inhalation of large quantities of dusts of any kind can damage the lungs but that some dusts are more harmful than others. His conclusions were based on first-hand observations of workers in hospitals, in their homes, and in the factories.

Two other great Englishmen in the history of pneumoconiosis about the middle of the 19th century were T. B. Peacock and E. H. Greenhow. Peacock first established miners' disease as an entity and distinguished it clinically from pulmonary tuberculosis before little was known about bacteriology and nothing about x-rays. Greenhow carried out the first large field investigations into the dusty industries of England and Wales, including the heavy-metal industries, the potteries, coal, copper, and lead mining, and even agriculture. In the *Transactions of the Pathological Society of London* (1860-1866) there are to be found excellent descriptions by both of these physicians of the disease, which was later to be called silicosis by Visconti, in 1870. They even found the dust of free silica in the lungs and examined it under polarized light.

For a long time after this excellent work nothing much was done about the dust diseases, but about the beginning of the 20th

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H. M. Medical Inspector of Factories.

century a new interest began to be taken in the problem not only in England but also in other parts of the world. About the same time the tempo of life in general began to increase, and there was an urge for increased speed of production—an urge which has gradually gathered momentum. With the replacement of hand labor by the machine, dusty processes have become more dusty and methods of dust control have lagged behind output. The result has been a remarkable increase of the incidence of the dust diseases.

A noteworthy contribution in 1892 was Arlidge's book on the occupational diseases, which was only the second to be published in England.

In 1904, J. S. Haldane and his colleagues ascribed the high mortality among tin miners to the inhalation of rock dust. A little earlier, dust phthisis among slate workers was shown to be related to the dust of slate, particularly when it had a high quartz content.

E. L. Collis (1915) pinpointed the dust of free silica as the main cause of most of the dust diseases. He also drew attention to the role that dust inhalation plays in determining the mortality from lung diseases experienced by the general population. Long ago he showed the influence of air pollution on the mortality from lung diseases in general. Though Collis did this work 40 years ago, he is always up-to-date. He had, and still has, an uncanny knack of seeing to the heart of any problem. Just now in England there is controversy about chronic bronchitis and emphysema and whether they can be held to be caused by the inhalation of dust. It is true that the incidence of chronic bronchitis and emphysema is high among the general population, and the possible causes are legion. It is difficult to decide (in Arlidge's words) how much of the malady is "town-made" or "trade-made." But as Collis showed years ago, an agent which irritates the lung parenchyma can also irritate the bronchial mucous membrane. At present there is one industrial pulmonary disease of which the diagnosis is made largely on the presence of chronic bronchitis and emphysema, and that is byssinosis.

In the last 50 years in Great Britain (as elsewhere), there has been intensive research into all aspects of the dust diseases, such as causation, pathology, incidence in various industries, and prevention. For the first 25 years or so, much of the impetus came from H. M. Medical Inspectors of Factories, and prominent in the study of the chest diseases were Collis, Middleton, and Merewether. It might be said that they played almost a lone hand, with meager facilities for investigation. However, they were helped greatly by hospital physicians, general practitioners, radiologists, and pathologists, notably Hall, Robertshaw, Kettle, Gloyne, and Cooke. During this period much attention was paid to silicosis, and it, with tuberculosis, was established as being the cause of disability and death in most of the industries where there is exposure to the dust of free silica. *Pari passu*, regulations laying down dust-control measures were issued by the Factory Department for most of the major industries. Notable contributions were Collis's work, already mentioned, on the role of free silica and Middleton's field investigation (with E. L. Macklin) into the grinding industry in 1923. In the late '20's, a series of brilliant observations by Cooke, Gloyne, Burton Wood, Stuart McDonald, and M. J. Stewart on the clinical, radiological, and pathological features of a new disease, which Cooke named asbestosis, led to field investigations by Merewether and Price (1930). These firmly established that the dust of asbestos was dangerous to health, and it was the first time that the dust of combined silica, as opposed to free silica, was found to damage the lungs. The condition, as Legge points out, was first seen in 1906 by Murray at Charing Cross Hospital, but little or no notice of it was taken at that time.

An important landmark was the passing of the Workmen's Compensation (Silicosis) Act of 1918, which came into force in 1919. For the first time compensation for disablement or death was given in respect of an industrial pulmonary disease. The Act provided initial and periodical medical examination of all workers in the refractories industry

and suspension from the industry on a diagnosis of simple silicosis as well as silicosis with tuberculosis. The medical officers responsible for the examinations were the Tuberculosis Officers of the Local Authority administering sanatorium benefit under the National Health Insurance Act. The scheme was revised in 1925, and again in 1931, to provide for examinations by a medical board. This was the beginning of the whole-time Silicosis and Asbestosis Medical Board under its first (and only) Chief Medical Officer, C. L. Sutherland.

A close association began between the Medical Inspectorate of Factories and the Silicosis and Asbestosis Medical Board, both groups working under the Home Office. In general, the Medical Inspectorate made the field investigations which established the risk of silicosis (or asbestosis) in an industry or process and which led to the establishment of the various compensation schemes. But the members of the Board (stationed at Sheffield, Manchester, Stoke on Trent, and Cardiff) also carried out field investigations into various industries, such as sandstone (Sutherland and Bryson, 1929), granite (Sutherland and Bryson, 1929), and potteries (Meiklejohn, 1949). But perhaps the most significant result of the formation of the Board was that there was begun a systematic and valuable collection of case histories with x-ray films, together with occupational details and postmortem findings.

At first the Board was limited to a few industries, but gradually its scope has increased and more and more dusty industries have come under examination. Workers in the refractories industry first came under a compensation scheme in 1925, metal grinders in 1927, and sandstone workers in 1929. In 1928, the Various Industries Scheme included the processes of mining, quarrying, drilling, and blasting in silica rock, the crushing and grinding of siliceous materials, and also isolated processes in steel foundries (not iron foundries) and metal works, in potteries, and in tin mines. In 1934 the Various Industries Scheme was amended to include coal miners with silicosis; in 1935 hematite ore miners were included, and in 1939 slate miners. Workers with asbestosis were first compensated in 1931; compensation for byssinosis was introduced in 1941 and was dealt with by a specially constituted board. Pneumoconiosis, as opposed to classical silicosis, in coal miners was brought under the compensation schemes in 1943 and in coal trimmers in 1946. The Various Industries Scheme was again amended in 1946 to include molders of iron castings who used siliceous parting powders and also blasters of any type of metal castings to free them from adherent sand, even if the blasting abrasive was nonsiliceous. The National Insurance (Industrial Injuries) Act was passed in 1946 and altered the whole basis of compensation in that it became the responsibility of the Government but with contributions from employers and workers. Beryllium "poisoning" became compensable in 1949. In 1954 all foundry workers (iron, steel, and nonferrous) became entitled to compensation under the Industrial Injuries Act. A fuller account of the development of compensation in Great Britain has recently been written by Meiklejohn (1954). The Silicosis and Asbestosis Medical Board was transferred from the Home Office in 1946 to the Ministry of National Insurance (later combined with the Ministry of Pensions). The Board was divided into a series of Pneumoconiosis Panels, but they work in much the same way as the Board did, except that there is now no Chief Medical Officer.

The various compensation schemes mentioned above reflect the evolution of the study of the industrial lung diseases in Great Britain. There are comparable legal enactments and regulations designed to control the risks in each industry, but they are too numerous to mention in detail.

To "flash back" a little, in 1936 Middleton, in his Milroy Lectures, reviewed the position of the dust diseases in Great Britain. He covered all the dusty trades, including coal mining, but at that time only cases of silicosis in coal miners were recognized as being eligible for compensation. Most of the miners,

such as hardheaders, drifters, or rippers, had been exposed to stone dust, but Middleton also showed that there was a high incidence of a lung disease among coal miners, particularly in South Wales, which was not classical silicosis. Much of his evidence was drawn from the findings of the Silicosis and Asbestosis Medical Board. Following his paper, there began a series of intensive investigations by the Medical Research Council into the health risks of coal mining, which covered all aspects, such as clinical, radiographic, and environmental (Medical Research Council Special Reports I [1943], II [1943], and III [1945]). The important fact emerging from this work was that coal dust by itself could cause pneumoconiosis, and this was followed, as previously mentioned, by the extension of compensation to coal miners and coal trimmers. In 1945, the Pneumoconiosis Research Unit of the Medical Research Council was set up in Cardiff, and an impressive body of work has emerged on various aspects of coal miners' pneumoconiosis. This work is well known, and I propose, therefore, to limit my remaining observations mainly to the dust diseases resulting from work in places which come under the Factories Acts, with only incidental references to coal mining.

SILICOSIS

Silicosis has been found to be a cause of disability in the following broad groups of industries as well as in coal mining and other forms of mining, such as tin, hematite, copper, barites, and fire clay.

1. Refractories industry. This group includes the making of silica bricks, furnace dismantling and rebuilding, and retort setting and repairing.

2. Pottery industry. Workers in this group have been engaged in the manufacture of both earthenware and china with their various subdivisions and as flint millers and polishers. The substitution of ground flint by alumina for the placing of biscuit ware has resulted in a diminution of deaths from silicosis among workers in that process.

3. Stone quarrying, crushing, and dressing. This group includes workers with sandstone, millstone, gritstone, slate, granite, and other igneous rocks. The risk varies with the amount of free silica in the rock and, of course, with the protective measures adopted. Recently some limestone quarrymen have contracted silicosis, but the limestone contained a fairly high proportion of free silica.* In this group could be included rock tunnelers, in whom the onset of silicosis may be very rapid. As regards slate workers, a billiard-table maker has recently received compensation for silicosis.

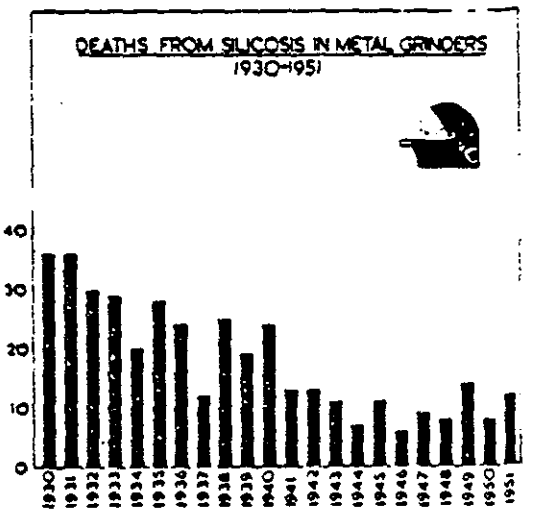

Fig. 1.—Chart showing diminishing numbers of deaths among metal grinders during the period 1930-1951, inclusive.

4. Metal grinding. The silicosis risk in the grinding of metals has greatly diminished, owing to the replacement of the sandstone grinding wheels with artificial ones composed of Carborundum, alumina, or emery. Apart from the lower "toxicity" of the dusts from these substances, the wheels are much harder than sandstone, and less dust is created. But the grinders of castings (iron, steel, and nonferrous) are still exposed to a silicosis risk on account of the presence of burned-on sand on the castings. The diminishing number of deaths among grinders is shown in the chart (Fig. 1).

* Doig, A. T.: Unpublished data.

5. Sandblasting. This job, as Merewether (1936) showed, had a high silicosis risk. The position has altered materially after sand began to be replaced by other nonsiliceous abrasives, such as steel shot, and since the use of sand as an abrasive was prohibited in 1949 by the Blasting of Castings (and other Articles) Regulations. The process of wet sandblasting of ships' hulls does not come under the Regulations (because legally a ship is not an article), but it is thought that there is also a risk of silicosis in the job. Sandblasting is still used in the open air on works of engineering construction, e. g., in order to prepare large metal surfaces for the application of paint.

6. Manufacture of abrasive soap. Middleton showed in 1936 that the manufacture of abrasive soaps carried with it a risk of acute or subacute silicosis, accompanied by tuberculosis. There was a disastrous experience at one factory in London between 1921 and 1928, when there were 13 deaths among 81 workers. This experience led to rigorous dust control in the processes, but between 1941 and 1952 there were nine deaths from silicosis among this type of worker. It is not possible to state the incidence of disease and disability, because there is a rapid turnover of labor (mostly young women) in the job. Silica flour is still being used as an abrasive, though efforts are being made to find a less harmful substitute which will be acceptable to the manufacturers and the users. There were no new cases of silicosis in this trade in 1953.

7. Foundry industry. The iron and steel foundry industry has been investigated in great detail during the past 10 years or so, and the results were published in 1950 in book form by McLaughlin and others ("Industrial Lung Diseases of Iron and Steel Foundry Workers"). Pathological studies of 64 cases, as well as clinical and x-ray examinations of some 3000 workers, showed that there was a varying risk of silicosis and mixed dust pneumoconiosis (to which reference is made below) in the various categories of foundry workers.

Steel fettlers (dressers or castings cleaners) had a severe risk, largely owing to the fact that the dust from the pneumatic hammer is not controlled. The main pathological lesion was classical silicosis, with or without tuberculosis. Among iron fettlers, the risk was not so great as among steel fettlers, and the main pathological lesion found was mixed dust pneumoconiosis, though classical silicosis did occur. Steel molders were less affected by disease and disability than iron molders, though statistically the incidence of x-ray abnormalities was greater among steel workers. More deaths occurred among iron molders, and this fact appeared to be related to the use of siliceous parting powders. The use of siliceous parting powders has now been prohibited by the Foundries (Parting Materials) Regulations, 1950. Silicosis still occurs among shot blasters of both iron and steel castings, but the risk is controlled by ventilation and personal protective devices, and the numbers of cases and deaths, in contrast with the experience among steel fettlers, are not increasing. Other categories of iron and steel foundry workers are subject to much less risk than the above groups of workers. New and stringent regulations (Iron and Steel Foundry Regulations) were issued in 1953. They require strict dust control in the dusty processes.

The question of pneumoconiosis among nonferrous foundry workers is now being examined in detail. Isolated cases of silicosis and mixed dust pneumoconiosis have occurred among both casters and dressers of nonferrous metals, but an extensive survey has not yet been carried out. Harding and McLaughlin † have described the pathological, clinical, radiological, and environmental details of six fatal cases.

Table 1 shows the number of new cases of pneumoconiosis (i. e., silicosis and mixed dust pneumoconiosis) in foundry workers diagnosed by the Pneumoconiosis Panels in 1953.

These figures can be regarded as an understatement of the real position. More than half

† Harding, H. E., and McLaughlin, A. I. G.: To be published.

TABLE 1.—*New Cases of Pneumoconiosis—1953*
Foundries

Occupation	Total
Iron molding	57
Iron dressing	21
Iron foundry knockout	1
Steel molding	4
Steel dressing	45
Nonferrous dressing	2
Welders and burners (in castings cleaning shops)	10
General foundry work	52*
Total	192

* Twenty-seven of these workers had also been exposed to coal dust for varying periods.

of the cases in iron molders came from one area where there had been an x-ray survey of three foundries. If more iron foundries had been similarly surveyed, more cases would have been found. In certain steel foundries the dressers (chippers or castings cleaners) are x-rayed each year, but a complete picture of the true position will not be obtained until all workers undergo periodical medical examination. This statement also applies to other dusty industries.

In Table 2 details are given of fatal cases of silicosis and asbestosis investigated fully by the Medical Inspectorate of Factories between 1930 and 1953. The average ages at death and the length of employment in the dusty industries are also given.

It is seen that as regards silicosis the manufacture of scouring powders and sandblasting were the most dangerous industries, with the lowest average age at death and the shortest average period of employment. Another interesting point is that tuberculosis occurred in about half of the cases of silicosis, whereas it was found in only about two-fifths of the cases of asbestosis. The average age at death and the length of exposure in the cases of asbestosis were much lower than in the silicotic group as a whole but more on the level of sandblasters and makers of scouring powders.

The connection between asbestosis and cancer of the lung is becoming clearer, and in one series of 100 autopsies on asbestosis cases there were 25 cases of cancer of the lung. Wyers (1949) has pointed out that the x-ray

TABLE 2.—*Fatal Cases of Silicosis and Asbestosis Investigated by Factory Department, 1930-1953*

	Deaths, No.	Average Age at Death, Yr.	Duration of Employment		
			Longest, Yr.	Shortest, Yr.	Average, Yr.
Silicosis					
Pottery					
Silicosis	770	41.7	62.0	1.5	26.4
Silicosis with tuberculosis	501	36.2	47.0	3.0	24.7
Sandstone					
Silicosis	207	40.9	62.0	2.0	20.0
Silicosis with tuberculosis	229	37.5	56.0	3.0	17.4
Grinding of Metals					
Silicosis	123	50.5	41.0	11.0	25.9
Silicosis with tuberculosis	211	34.0	34.0	2.5	22.0
Sandblasting					
Silicosis	72	30.4	42.0	1.7	13.4
Silicosis with tuberculosis	49	46.2	46.0	2.0	13.1
Manufacture of scouring powders					
Silicosis	14	40.8	37.0	2.3	8.3
Silicosis with tuberculosis	6	40.8	11.2	2.9	7.0
Miscellaneous					
Silicosis	293	55.4	57.0	1.5	21.3
Silicosis with tuberculosis	215	51.6	50.0	9.7	21.5
Total					
Silicosis	1,514	39.4	62.0	1.5	24.1
Silicosis with tuberculosis	1,209	34.4	67.0	0.7	21.2
Asbestosis					
Asbestosis	230	49.5	48.0	0.5	16.6
Asbestosis with tuberculosis	57	40.2	33.0	0.8	11.4

CASE HISTORIES OF 11 MALE PATIENTS WITH ASBESTOSIS

Case no.	Pt. init.	Pt. age, yr.	Pt. race	Durat. expos., yr.	Smoking history	Occupation
1	P.K.E.	58	W	20	2 pk./day 20 yr.	Hod carrier
2	J.W.	46	W	?	2 pk./day ? yr.	Ironworker
3	G.M.G.	55	N	?	?	Shipyard worker
4	H.M.G.	71	W	40	1 pk./day ? yr.	Ret. bricklayer
5	G.J.B.	61	W	30	2½ pk./day 10 yr.; stopped 8 yr. prior to death	Pipe insulator
6	H.R.H.	69	W	48	1 pk./day 20 yr.	33 yr. in asbestos mfg. & 16 yr. as asbestos insulator
7	E.J.H.	65	W	40	1 pk./day 40 yr.	53 yr. in asbestos mfg.
8	P.K.	62	W	None known	None	Carpenter
9	G.S.	68	W	?	30 yr.	Insulation worker; worked with Fiberglas many yr.
10	W.C.D.	60	W	40	1½ pk./day 46 yr.	Asbestos handler
11	C.B.	61	W	30	?	Occupat. expos. to asbestos & magnesium

From: Cordova, et al. 1962. Cancer 15:1181-487

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From: Cordova, et al. 1962. Cancer 15:1181-1187

appearances of asbestosis are undergoing some change in that nodular as opposed to ground-glass shadows are beginning to appear. He thinks it probable that this is because of the lessened exposure to dust over a longer period, owing to the rigorous application of exhaust ventilation to the dusty processes and the use of personal protective devices. Cases of asbestosis, however, are now appearing in workers who do asbestos lagging of pipes and boilers. In this process, particularly if asbestos is being sprayed, it is difficult to apply adequate protective measures, more especially because the workers are usually peripatetic.

PRESENT POSITION

How many cases of pneumoconiosis (silicosis, asbestosis, coal miners' pneumoconiosis, etc.) occur in Great Britain? It should be mentioned that at the present time it is impossible to give accurate figures of the populations at risk in the various trades and processes. Both the Registrar-General and the Ministry of Labour classify occupations in groups too broad to give specific figures of the populations in the dusty industries, and those in the possession of employers' associations and trades unions are not complete. It may be possible in several years' time to give more accurate information. All that can be done now is to state the numbers of deaths reported to the Registrar-General and to give the figures for one year of the new cases diagnosed by the Pneumoconiosis Panels.

The deaths from all forms of pneumoconiosis between 1940 and 1951, inclusive, in England and Wales are given in Table 3.

In all industries there were 8789 deaths from occupational fibrosis of the lung in the 12-year period, and it will be seen that the total yearly figures are going up. Over the same period there were 6907 deaths from non-occupational fibrosis of the lungs. About two-thirds of the total number of deaths in the occupational group occurred in coal miners, who form the largest group (about 700,000) exposed to dust inhalation. The figures rose steeply from 232 in 1940 to 937 in 1951 (in 1952 the figure dropped slightly, to 912). It may well be that part of the increase is due to more accurate diagnosis, or at least to greater interest in the pneumoconiosis problem, among coal miners. In Figure 2 is shown a comparison, based on the crude figures of Table 3, between the coal miners and factory workers. In the factory processes, the yearly number of deaths was going down until 1943, but there has been a slight rise in the later years.

TABLE 3.—*Deaths from All Types of Pneumoconiosis in England and Wales, 1940-1951*

Industry	1940	1941	1942	1943	1944	1945	1946	1947	1948	1949	1950	1951	Total
Potteries	58	46	47	41	32	41	49	54	46	63	73	62	606
Sandstone	108	82	54	77	57	65	61	55	68	51	59	71	770
Grinding of metals, etc.	41	28	26	21	24	22	32	27	26	37	22	21	325
Refractories	16	12	7	7	6	9	6	10	13	13	5	5	117
Miscellaneous	7	5	7	5	13	19	14	28	27	25	70	120	347
Coal mining	232	198	230	276	311	367	421	477	620	776	846	937	5,896
Other mining	64	40	46	43	39	30	31	20	19	64	42	31	370
Asbestos	11	17	11	8	10	11	10	15	15	17	12	15	161
Cotton (byssinosis)	..	..	6	7	1	10	3	4	5	7	11	8	66
Total	537	428	434	486	493	604	627	690	839	1,053	1,143	1,269	8,789
Non-occupational	530	461	445	488	531	529	598	616	651	554	670	782	6,907

In both groups, it should be emphasized that the deaths in each year are related to conditions which obtained in mines and factories some years previously, possibly 10 to 20 years or even longer.

New Cases in Factory Occupations.—The following Table 4 gives the numbers of new cases of pneumoconiosis diagnosed by the Pneumoconiosis Panels in 1953. These figures have been kindly supplied by the Ministry of Pensions and National Insurance, but it should be noted that they differ slightly as regards the numbers in each occupation from the figures originally supplied. The Ministry usually classifies the cases as falling into the

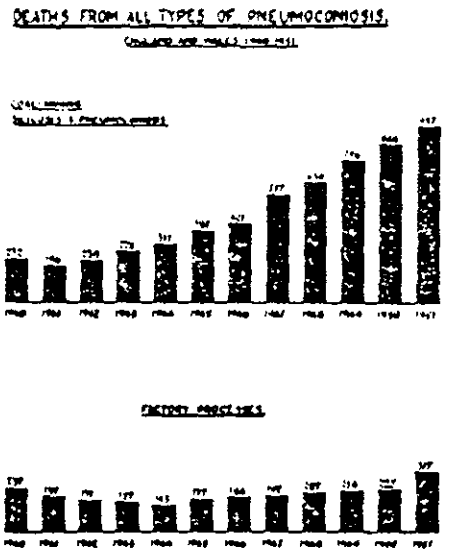


Fig. 2.—Chart showing trends of deaths from fibrosis of the lungs among coal miners and factory workers during the period 1940-1951, inclusive.

category of the last jobs done by the workers, whereas Dr. G. O. Williams, of the Factory Department, has reclassified some of the cases on the basis of all occupations done by each worker in order to determine the most likely cause of the pneumoconiosis.

There were 896 cases of pneumoconiosis diagnosed, and this figure includes 54 cases of byssinosis. The cases with an uncomplicated occupational history numbered 665, and in the remainder there was exposure for varying periods to other dusts, mainly coal dust. Additional exposure to coal dust occurred mainly in those industries situated in coal-mining areas, such as refractories, stone (sandstone and granite), pottery, slate, and foundry industries. The pottery industry contributed the greatest number of cases (353, mainly silicosis), and the foundry industry came next with 190 cases. There are roughly 25,000 pottery and about 220,000 foundry workers (iron, steel, and nonferrous); so it is probable that the pottery industry has a higher pneumoconiosis risk than foundries. Such general statements are not of much value, because the risk varies from process to process within the same industry. The substitution of ground flint by calcined alumina has reduced the silicosis risk among placers of biscuit ware in potteries, whereas the fettling of steel castings is still a dangerous job.

New Cases in Mining.—Table 5 gives the numbers of new cases of pneumoconiosis occurring in all mining operations in 1953.

These figures show clearly the tremendous problem which faces the coal-mining industry. There is no space to give in detail the varying degrees of severity of pneumoconiosis found in the 4048 cases, but the figures are given in the "Digest of Pneumoconiosis Statistics

TABLE 4.—*New Cases of Pneumoconiosis—1953*
Factory Occupations

Industry	No Other Dust Exposure	Other Dust Exposure		Total
		Coal	Other	
(1) Refractories	15	10	..	25
(2) Stone workers (sandstone, granite)	53	10	1	64
(3) Pottery	254	86	13	353
(4) Slate workers (quarrying and splitting)	62	39	..	101
(5) Foundries	154	27	9	190
(6) Metal grinding (other than foundries)	5	2	..	7
(7) Sandblasting	5	4	..	9
(8) Shot blasting	2	..	..	2
(9) Furnace dismantling, etc.	17	7	..	24
(10) Boiler scaling	4	1	..	5
(11) Abrasive wheel manufacture	3	..	..	3
(12) Asbestos	14	5	3	22
(13) Abrasive soap powders	..	..	..	..
(14) Graphite and carbon electrodes	..	..	..	..
(15) Tin smelting	1	..	..	1
(16) Cotton (byssinosis)	40	14	..	54
(17) Coal trimming	10	..	..	10
(18) Miscellaneous	21	..	..	21
Total	665	205	26	896

TABLE 3.—*New Cases of Pneumoconiosis—1953*
Mining *

Industry	Cases, No.
(1) Coal mining	4,018
(2) Tin mining	3
(3) Barytes mining	1
(4) Fire clay mining †	36
(5) Other clay mining	2
(6) Hematite mining	4
(7) Lead mining	2
(8) Tunneling	3
Total	4,069

* No cases in chert, oil shale, or stratified ironstone mining.
† Fourteen fire clay miners were also exposed to coal dust.

for 1953." issued by the Ministry of Fuel and Power (1954). It can be said, however, that more than 50% of the cases had only slight disability, and only about 3% were totally disabled.

The number of cases in hematite mining is diminishing, but the small numbers found in tunneling are usually instances of rapid and severe silicosis.

DUSTS OTHER THAN SILICA, ASBESTOS, AND COAL

About the middle 1930's the Factory Department began to turn its attention to other dusts. Middleton, in his 1936 review, referred to dusts, such as tripoli, sillimanite, kieselguhr, talc, china clay, and fullers' earth. In the same year Doig and McLaughlin published a paper on the x-ray appearances of the lungs of electric-arc welders. From this has arisen a world-wide study of the inert and radiopaque dusts, and notable American contributors have been Sander, Enzer, Pendergrass, Vorwald, and Hamlin. Perhaps the most interesting outcome of this study has been the alteration of the approach to the interpretation of x-ray films of the chest, because the x-ray features of siderosis, baritosis, and stannosis, in the absence of occupational histories and clinical examinations, can be mistaken for fibrotic changes in the lungs. In fact, an alarming x-ray picture is often found in a worker who has little or no disability. The study of siderosis was helped by the excellent work of Stewart and Faulds (1934) and later Craw (1937) on the hematite miners, in which group the lesion was found to be siderosilicosis, often accompanied by tuberculosis. Craw's later work (1947) in eliminating tuberculosis from the hematite mines and in improved dust control methods is of major importance.

Other occupations and dusts which have been studied include those of boiler scalers, graphite workers, grain dockers, and workers exposed to beryllium and its oxides, to the dust of leather mixed with other dusts, and to bagasse. Attention has also been given to exposures to the dusts of aluminum, manganese, and vanadium and to asthma occurring in workers exposed to the double salts of platinum and also to various wood dusts, such as western red cedar.

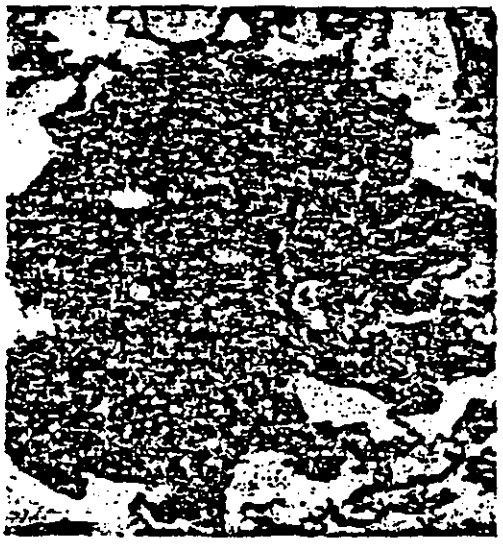
Pathology.—A great deal of work has been done on the pathology (gross, histological, and experimental) of coal miners' lungs by Gough, Harding, King, Heppleston, Wright, Gloyne, Nagelschmidt, and others. I might, however, state some conclusions about pathology which Harding and I (with our late colleague, S. Roodhouse Gloyne) have arrived at after studying the lungs of workers in many diverse industries.

Until about 10 years ago, the pathology of the dust diseases was dominated by the classical silicotic nodule (Fig. 3). In the same way, before a diagnosis could be made, the patient had to have x-ray nodulation, or the classical "snowstorm" effect. Classical silicosis usually occurs after the worker has been exposed to dust containing a high proportion of free silica. It is becoming increasingly clear, in our opinion, that even small proportions of free silica in a dust can cause a fibrosis which is composed of nodules not of the classical type (Fig. 4). The arrangement of the fibers is linear and radial, and the outline of the whole nodule is stellate. It looks like a black star. Harding, Gloyne, and I have applied the term mixed dust pneumoconiosis, or mixed dust fibrosis, to this nodule. It has been found in foundry workers, especially in cleaners of iron castings, in persons exposed to the dust of graphite containing about 10% free silica, and in boiler scalers who are ex-

Fig. 3.—Classical silicotic nodule from lung of a gold miner (30 years old); hematoxylin and eosin; × 24.

posed to a mixed dust with a low proportion of SiO_2. The *coal nodule of coal miners'* pneumoconiosis has much the same appearance as our mixed dust pneumoconiosis nodule. Coal contains varying proportions of free silica. In any case, the common denominator in those cases and occupations in which this type of nodule is found appears to be the presence of a small proportion of free silica in the dust. Where there is a high proportion, as stated above, the classical silicotic nodule is found. But in the same case, there may be both the classical nodule and the

Fig. 4.—Mixed dust pneumoconiosis nodule from a steel fettler's lung; hematoxylin and eosin: × 24.



mixed dust pneumoconiosis nodule. Indeed, there are also nodules which show a transition stage between the mixed dust nodule and the classical one (Fig. 5). It should be mentioned that the x-ray appearances of the lungs of a worker with mixed dust pneumoconiosis differ little, if at all, from those of one with *silicosis, and the disease is just as disabling* and as fatal.

Boiler Scalers' Pneumoconiosis.—Experience of pulmonary diseases in ships' boiler scalers illustrates well the etiology of mixed dust pneumoconiosis. These men are exposed to a mixed dust which varies according to the type of fuel used to heat the boilers and also according to the source of the water used

Fig. 5.—Transition nodule, from mixed dust pneumoconiosis nodule to silicotic nodule in an iron dresser's lung; hematoxylin and eosin: × 24.

in them. It contains carbonates, silicates, iron, and carbon. There is usually under 5% *of free silica, but there is often a high* proportion of iron and its oxides. Flue dust contains more iron than does the scale on the water tubes, sometimes as much as 48%.

In the first case of boiler scalers' pneumoconiosis the worker had been scaling boilers for over 40 years (Harding, Tod, and McLaughlin, 1944). In 1938 an x-ray film *of his chest showed nodulation* in the upper and outer lung fields, and in the lower zones reticulation (or micronodulation). At that time we made a tentative diagnosis of silicosis in the upper zones, with siderosis in the lower zones. Seven years later the patient

died of cancer of the lung, and histological examination of the lungs showed (apart from the cancer) the presence of classical silicotic nodules in the upper and outer zones, and in the lower zones (where there was x-ray reticulation) there were mainly deposits of carbon and iron dust in the lungs, with no fibrosis. In all other parts of the lung there were nodules of mixed dust pneumoconiosis. Up to the present time we have had autopsies on nine boiler scalers, and all showed this type of fibrosis. Only one scaler had classical silicotic nodules in addition. But five of them also died of cancer of the lung, and this may well have been partly caused by carcinogenic substances present in the soot.

The change from coal to oil as a fuel for ships' boilers has brought with it another problem. The dust from oil-fired boilers causes symptoms of bronchospasm or asthma in a high proportion of cases, and it is likely that this is due to the presence of vanadium in the oil soot. Williams (1952) has published an interesting series of cases illustrating this point. My colleagues and I are at present examining (with clinical, radiographic, and environmental details) the boiler scalers (some 300 of them) in the port of Southampton, where the boilers of ocean-going and large passenger liners are cleaned. The investigation has not been completed, but up to the present we have not found as much lung damage as was shown in a previous inquiry in the port of Hull, where the boilers of fishing trawlers are mainly scaled. So far we have found three cases of compensable pneumoconiosis among 50 workers and one case of cancer of the lung, together with a proportion of cases with lower degrees of pneumoconiosis and disability.

Talc Pneumoconiosis.—A great deal of work on talc pneumoconiosis has been done in the United States and Canada (Dreessen, 1933; Dreessen and Dalla Valle, 1935; Riddell, 1940; Siegal, Smith, and Greenburg, 1943, etc.). Merewether (1933-1934), in England, studied the x-ray and clinical features of rubber workers exposed to talc dust. But it was not until 1949 that we were able to study the histological appearances of a proved case of talc pneumoconiosis (McLaughlin, Rogers, and Dunham, 1949). A man, 51 years of age, had worked for 37 years in a rubber-tire factory, where he had been exposed to a fairly high concentration of talc dust. Moderately advanced pneumoconiosis of both lungs was found at autopsy (in addition to incompetence of the aortic valve). Throughout the lung substance were scattered small gray nodules, more numerous in the lower lobes. In some areas the nodules had coalesced to form small masses. The fibers appeared to be arranged concentrically around small vessels, giving the impression of whorling, but not like the appearance of silicosis. There were, in addition, many "curious" or talc bodies, resembling, but easily distinguishable from, asbestos bodies. Much dust, which proved to be talc, appeared in the sections, and it was thought at the time that the particles were all fibers. More recent work on the lung by Nagelschmidt ‡ has shown that the bulk of the dust is in the form of plates with only a few fibers.

At least three other autopsies on cases of talc pneumoconiosis (all from rubber works) have been made, and they show similar features to the first published case. Small surveys have been made on workers exposed to talc dust in various industries, but larger ones have been planned to take place shortly. It is clear that talc dust, though fibrogenic, is not so active as asbestos in damaging the lungs.

Leather Dressers' Pneumoconiosis.—Cooperation between the directors of chest clinics, the mass radiography units, and the Medical Inspectorate of Factories has been instrumental in bringing to light hitherto unsuspected causes of pneumoconiosis. Since the mass radiography campaign was begun in 1948 for the early detection of cases of pulmonary tuberculosis, some 13,000,000 persons have undergone examination. Many factory populations have been surveyed, and

‡ Nagelschmidt, G.: Personal communication to the author.

included among them have been factories where there is a dust risk. The workers at one leather factory were examined by Dr. Hugh Ramsay, of the Wanstead Mass Radiography Unit. He drew our attention to the fact that in one department a high proportion of the workers showed abnormal x-ray changes, whereas in other departments, where there was no dust, no abnormalities were seen. In the department with the high proportion of x-ray abnormalities, skins loaded with china clay and calcium carbonate are dressed on rapidly revolving felt wheels covered with a layer of fine Carborundum powder. The job is very dusty, though it is done under exhaust ventilation. The dust is composed of much fine leather dust, with smaller proportions of china clay (kaolin), calcium carbonate, and Carborundum. It was found that the dust contained about 5% of free silica. The x-ray films showed all stages of abnormality, varying from early reticulation (micronodulation) through nodulation to massive shadows. In a few cases there was clinical evidence of disability. One man with an x-ray film showing massive shadows had died from "asthma and pneumonia" a year before the investigation began, and there was no autopsy. In spite of the fact that up to the present time no pathological evidence has been available, it is likely that the condition will fall into the group of the mixed dust pneumoconioses. The dust of china clay, which for years has been thought to be comparatively harmless, is becoming more and more suspect. Even apart from china clay, the presence of a small proportion of free silica is prima facie evidence that the dressing of such skins is a hazardous occupation.

Beryllium Pneumonitis and Granulomatosis.—It is remarkable that British experience of beryllium pneumonitis and granulomatosis is not as extensive as that in the United States, though many workers have been exposed to the dust of fluorescent lamp powders containing beryllium oxide and also the dust and fumes from the alloys. Only one death has occurred, and the Factory Department has collected clinical, x-ray, and environmental details of five noniatal cases, one of whom is now very ill. The other four cases are either recovering, or their condition is stationary. It is also remarkable that five out of six affected workers were chemists engaged in the development of the beryllium lamp powders. At the factory where the fatal case of beryllium granulomatosis occurred, 150 of the other workers were examined clinically and radiographically and no more cases were found, though there had been considerable exposure to the dust of the lamp powders in the early days. It is undeniable that beryllium oxide is toxic, but examination of the histology of the lungs of the one fatal case has led me to hold the unorthodox view that beryllium was not the sole cause of the condition. There were two types of lesion: one of a granulomatous type, in which there were giant and epithelioid cells, and the other in which were many fibrotic nodules indistinguishable from silicosis. Under polarized light many doubly refractile particles were seen in the nodules, and an eminent crystallographer stated that these were cristobalite, one of the most active forms of free silica. This is not surprising, because one of the ingredients of the lamp powder is silica gel (25%), which in the preparation of the powder is heated up to about 1100 C. The changed methods of preparing the lamp powder have eliminated both the beryllium oxide and the free silica, so that the risks both of beryllium granulomatosis and of silicosis have also been eliminated.

"Pneumoconiosis" from Vegetable Dusts.—Apart from cotton, not a great deal of work has been done on the vegetable dusts. The illnesses noted among cotton workers include mill fever, a transitory illness which affects nearly all new workers in cotton, flax, and hemp mills and also in malt houses; "Monday fever" (or feeling); weavers' cough, and byssinosis.

Byssinosis develops after about 20 years as a natural progression from "Monday fever" and in its final stage has the characteristics of chronic bronchitis and emphysema. As Schilling (1954) says, though it was de-

scribed by Greenhow nearly 100 years ago, its etiology is still obscure. It presents an odd but characteristic history of chest tightness and breathlessness on Mondays, which gradually extends to other working days as the disease progresses. "In its later stages these symptoms are very distressing, but usually remain worse on Monday than on any other day. It causes no specific x-ray changes in the lung fields." In the main, the workers in the cotton card and blowing rooms suffer most from the disease. Strippers and grinders who clean the carding engines are especially affected. As mentioned previously, workers with byssinosis are compensated under a special scheme.

Bagassosis.—Only one factory in the country handles bagasse (or sugar cane without the sugar), and bagassosis has occurred mainly in those workers who were grinding bagasse in a dry state. The condition is an acute bronchiolitis, with high temperature, severe dyspnea, and x-ray picture of the lungs showing generalized miliary shadows. Fifteen cases of this acute disease have come to the notice of the Factory Department; some of these have been described by Castleden and Hamilton-Paterson (1942), Gillison and Taylor (1942), and Hunter and Perry (1946). The last case occurred in 1948. Since we got the firm to grind the bagasse under water, there have been no further cases.

Farmers Lung.—Farmers', or threshers', lung was first described by Campbell (1932), and after that Faweitt (1936 and 1938) did a great deal of work on the condition. It has features similar to bagassosis, but, since it occurs mainly among workers who have been handling moldy hay during a wet summer, it is regarded as being caused by a fungus. Faweitt was firmly of this opinion. Single cases of the condition are described from time to time by physicians in the agricultural districts.

Pneumoconiosis in Grain Dockers.—Dunner, Hermon, and Bagnall described in 1946 the clinical features and abnormal x-ray appearances in a group of 55 grain dockers. Thirty of these men had pulmonary tuberculosis, but 11 others had x-ray changes suggestive of the presence of pneumoconiosis. Fourteen had normal x-ray films. Analysis of the dusts from various grains showed small percentages of free silica; oat dust, for instance, had 5%. No autopsies have been carried out, but it seems that a case has been made out for an extended investigation of larger groups of grain dockers.

Graphite Pneumoconiosis.—It has been found by some observers in England (Dunner, 1945, 1948, and 1949; Gloyne, Marshall, and Hoyle, 1949, and Harding and Oliver, 1949) that workers exposed to the dust of natural graphite develop radiographic changes in the lungs and disability. The range of x-ray abnormalities closely resembles that seen in coal miners. Pathological and experimental studies (Gloyne and others, 1949, Harding and Oliver, 1949) show that the condition falls into the group of the mixed dust fibroses, the fibrotic nodules having a linear and radial pattern as opposed to the whorled fibrosis of the classical silicotic nodule. Natural graphite contains small percentages (of the order of 5%-10%) of free silica. There is as yet no evidence that pure graphite will produce a similar condition.

Manganese Pneumonitis.—A few cases of manganese poisoning affecting the nervous system occurred in the middle 1930's in workers grinding manganese dioxide for use in lamp batteries. In 1946, Lloyd Davies reported a high incidence of pneumonia in a group of workers exposed to manganese dioxide dust in the manufacture of potassium permanganate. Animal experiments by Lloyd Davies and Harding (1949) confirmed that manganese dioxide irritated the lung tissue and caused intense infiltration of the alveolar walls and alveoli. Later granulomatous changes developed in some instances. These results are in line with those described by workers in other countries.

Vanadium Pneumonitis.—Wyers (1946) recorded his observations on workers exposed to vanadium pentoxide dust, and his results

are similar to those described by Sjöberg (1949) in Sweden. The effects are a combination of systemic poisoning and irritation of the pulmonary tissue, leading in some cases to bronchospasm and pneumonia. Doig § and Williams (1952) have described the marked bronchospasm occurring in workers exposed to the soot of oil fuel, which is thought to be caused by the high percentage of vanadium in the soot.

SUMMARY

The position as regards the dust diseases in Great Britain may be summarized as follows:

1. During the past 50 years, the risk of silicosis in most of the major industries has been firmly established. Until the late 1920's, free silica was the only dust which was thought to damage the lungs. Asbestosis, caused by the dust of a combined silica mineral, came into prominence about that time. In the middle 1930's, attention was given to the effects of other dusts on the lungs, such as iron and its oxides, and of radiopaque dusts, china clay, sillimanite, kieselguhr, talc, and mixed dusts containing free silica.

During the middle 1930's also, the effects of coal dusts on the lungs were given increasing attention, and extensive surveys were carried out, culminating in the formation of the Pneumoconiosis Research Unit of the Medical Research Council in 1945. The coal miners numerically constitute the greatest problem of dust disease in Great Britain.

2. Despite the combined efforts of inspectors of factories and mines, physicians, chemists, engineers, and research workers, the deaths from pneumoconiosis have continued to rise yearly. The reasons for this increase may be found in more accurate diagnosis of the condition and in the publicity given to occupational diseases of the lungs, leading to more frequent mention of pneumoconiosis by physicians on death certificates. But a more likely cause is the urge for increased speed of production, and the introduction of machines which make a dusty process more dusty and cause a lag in dust-control methods.

3. Compensation for industrial pulmonary disease began soon after 1918, and gradually most of the processes and industries which damage the lungs are being brought under the provisions of the various Acts of Parliament. The Industrial Injuries Act of 1946 removed the responsibility for compensation from employers and insurance companies to the Government. Since then cases taken under Common Law against the employers have increased rapidly.

4. Numerous legal provisions, contained both in Acts of Parliament and in Regulations, have been brought into force. Routine inspection by inspectors of factories and mines has been instrumental in limiting the numbers of cases of disability and death brought about by inhalation of the dangerous dusts.

5. It is expected that more accurate information about the incidence of the dust diseases will be obtained in the near future, when statistical studies of the information obtained by the Pneumoconiosis Panels have been carried out, and when the report of the Registrar-General about the occupational incidence of disease at the time of the 1951 Census becomes available. At the present time no reliable information exists about the populations at risk in each industry and process.

6. Surveys of the population by mass radiography to detect early tuberculosis are bringing to light information about the effects of dust in some hitherto unsuspected occupations.

7. Many problems about dusts and the lungs are still unsolved, and some would say that we have only just begun to attack the fringes of the problem. Nevertheless, a great deal has already been accomplished.

Assistance in compiling the information contained in this paper was rendered by Dr. P. K. Walker and the members of the Pneumoconiosis Panels of the Ministry of Pensions and National Insurance: Drs. E. R. A. Merewether, A. T. Doig, and G. O. Williams, of the Factory Department.

§ Doig, A. T.: Unpublished data.

Ministry of Labour and National Service: Dr. H. E. Harding, lately of Sheffield University, and Dr. G. Nagelschmidt, of the Safety in Mines Research Laboratories, Sheffield.

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