

Pneumoconiosis

INDUSTRIAL DISEASES OF THE LUNG
CAUSED BY DUST

By

P. F. HOLT, Ph.D., D.I.C., F.R.I.C., M.I.Biol.

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PREFACE

Fifty years ago pneumoconiosis was a word which belonged exclusively to the vocabulary of the medical practitioner and it was seldom used even by him. Now pneumoconiosis is recognised as the most serious industrial disease. Legislation has been introduced to regulate the conditions in factories where operations give rise to dust and to provide for the compensation of sufferers. In some industries agreement has been reached on maximum permissible dust levels and these are enforced by law. Special ventilation methods have been designed to reduce the dust levels in industrial atmospheres, and in some works the periodic check on dust levels is an established routine, as is the regular medical examination of all personnel.

Quite apart from the research workers who are studying the disease and the dusts which cause it, an increasing number of scientists, medical specialists, physicists, chemists, engineers, and administrators have become involved in the pneumoconiosis problem. The papers in which the various aspects of the disease are discussed are widely scattered in many types of journals and it is often difficult for a worker to find information on matters outside his own field. The intention of this book is to provide sufficient material to act as an introduction and to indicate where further information can be found. Each chapter reviews one aspect of the subject with enough detail to give a true picture of the present position, but no lengthy discussion, which properly belongs to a more specialised book, has been attempted.

Throughout this book an attempt has been made to emphasise the controversial nature of much of the information. This, it is hoped, may counteract the reliance which is sometimes shown on one or other of the many theories which have been advanced. Undoubtedly, less stringent precautions against dust are sometimes taken because, on theoretical grounds, a particular dust is presumed to have a low pathogenicity. It cannot be emphasised too strongly that any dust must be regarded as a health hazard unless specific investigations have proved it otherwise, and this statement applies particularly to silica dust in all its forms. If this book facilitates the work of anyone interested in the problem of pneumoconiosis, it has served its purpose.

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CHAPTER 1

INTRODUCTION

For centuries it has been known that certain lung diseases are associated with dusty occupations. In 1866 Zenker¹ used the word "pneumonokoniosis" to describe these diseases and this word was abbreviated by Proust² in 1874 to "pneumoconiosis". In the National Insurance (Industrial Injuries) Act, 1946, Section 57 (3), pneumoconiosis is defined as "fibrosis of the lungs due to silica dust, asbestos dust or other dust and includes the condition of the lungs known as dust reticulation but does not include byssinosis". Byssinosis is a condition of the lung which results from the inhalation of cotton dust.

It is impossible to say how widespread was the incidence of pneumoconiosis until very recent times. The symptoms of pneumoconiosis and those of tuberculosis are very similar. "It must be accepted as a fact that dust induces a malady bearing a strong similitude to tubercular phthisis, and yet that the malady is not tubercular in its actual nature" (Aldridge,³ 1892). Before the discovery of the tubercle bacillus in 1882, conditions of the lung produced by dust could not be differentiated with certainty from tuberculosis; indeed differentiation between pneumoconiosis and other pathological conditions of the lung was seldom made until some fifty years later. Even in 1919 Landis⁴ introduced a paper on dust diseases by the statement "Pneumoconiosis occupies a curious position. The term is a familiar one and the nature of the disorder is described in all textbooks dealing with diseases of the lungs. In spite of this the term is rarely used by the clinician and only a little less frequently by the pathologist. One may search statistics dealing with morbidity and mortality rates and find no mention of the condition. In those instances in which the deposition of dust in the respiratory system has actually given rise to pathological changes, the illness or the cause of death is ascribed to secondary changes, such as chronic bronchitis, asthma or tuberculosis. The etiological factor is thus concealed by the use of other terms." For a long time the inhalation of any dust was considered to predispose to tuberculosis, a disease widely prevalent amongst factory operatives in the

eighteenth and nineteenth centuries. Not until 1913 did Collis⁵ point out that the nature of the respiratory disease caused by dust varies with the type of dust inhaled. In his Milroy Lecture in 1915 he stated that only free silica caused silicosis and that silicates other than asbestos were not fibrogenic.

In England attention was drawn particularly to the lung diseases of textile workers. Thackrah⁶ wrote of the damage caused by the inhalation of flax dust, particularly as it affected child workers. "Children from 7 to 15 years of age go to work at half past five in the morning and leave at seven in the evening and thus spend twelve hours a day, for five or six years, in an atmosphere of flax dust." He suggested that ventilating ducts should be cut in the factory floors and that a forced draught should be used to remove the dust from the building. "Let a light broad wheel, attached to the machinery, be made to revolve rapidly. A current of air will thus be produced, and this entering the channel will draw down the greater part of the dust, and carry it out of the building." However, Beddoes⁷ in 1799 attributed the high mortality from phthisis in the textile industry to confinement and inactivity rather than to dust.

Silicosis and asbestosis must be the oldest of industrial diseases since materials known to cause these diseases are amongst those used by earliest man. The manufacture of gunflints at Brandon, Sussex, and at Meusnes in France was associated with a terrible mortality among the workers, who used implements little different from the deerhorn picks with which flints were shaped in the palaeolithic period, so it seems likely that silicosis existed in prehistoric times.⁸ Although the pyramids were built from limestone, the dust of which is harmless, the Egyptians (3000–2000 B.C.) used granite for their doors and for building altars and obelisks.⁹ The Egyptians also mined gold which was found in quartz veins. The quartz was crushed in mortars and ground to a powder, then the gold was separated by levigation. Signs of pneumoconiosis were found in a number of Egyptian mummies.¹⁰ The fact that the Romans mined asbestos more than two thousand years ago suggests that asbestosis occurred early in history. Early references to pneumoconiosis have been collected by Orenstein¹¹ and by Hunter.¹²

Silicosis was mentioned by Hippocrates, who described the metal-miner as a man who breathes with difficulty, and Georgius Agricola in his *De re metallica* (1556) spoke of dusts which ulcerated the lungs and caused consumption. "Other mines are very dry and the constant dust enters the blood and lungs, producing the difficulty of breathing which the Greeks call asthma. When the dust is corrosive it ulcerates the lungs and produces consumption; hence it is that in the Carpathian Mountains there are women who have married

seven husbands, all of whom this dreadful disease has brought to an early grave."

Agricola advocated the ventilation of mines not only because of the effects of noxious gases and dust on the miners but also because their lights would not burn: "These lights, too, burn feebly, whilst they give out a worse emanation than men do." Brard¹³ (1788) mentioned that glow-worms were used for illumination when a light would not burn. The conditions in the mines at this time were very bad and it is not surprising that in *Bericht von Bergwercken* of 1690, there were published oaths for each class of the mining community, and prayers for all their conditions!



Fig. 1.1. Stamp Mill for crushing flints for the manufacture of Porcelain (Tomlinson: *Illustrations of Useful Arts, Manufactures and Trades*, 1867).

As industries developed and siliceous materials were used in increasing quantities, pneumoconiosis became more common. The industries involved can be judged by the names under which the disease was known in different parts of the country—mason's disease, potters' rot, grinders' asthma, miners' disease, miners' asthma and sewer disease.

During the eighteenth century a high phthisis mortality was noted

amongst workers manufacturing millstones, stone-cutters and miners,⁷ and needle pointers.¹⁴ Linnaeus stated that all grind-stone cutters died of phthisis before the age of thirty. This description of the process of needle-pointing is taken from *Illustrations of Useful Arts, Manufactures and Trades* (1867): "The third process is pointing or grinding the ends of the wires on a grit stone. Several thousand wires can be pointed at both ends in an hour. A stream of sparks accompanies the contact of the wires and the stone, and minute particles of grit and steel fill the air of the room, and, entering the workman's lungs, produce a disease called the 'grinders' asthma'."

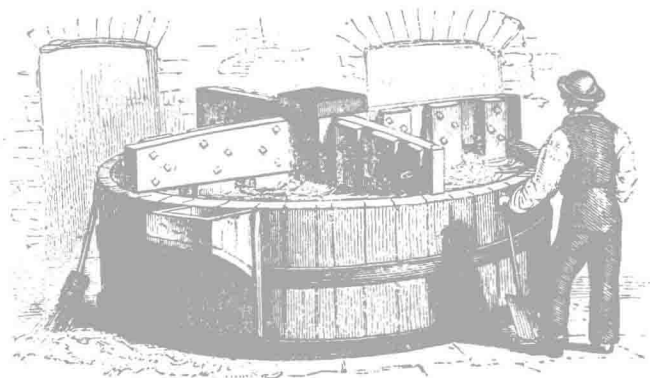


Fig. 1.2. Flint Pan used in the wet grinding of flint (Tomlinson: *Illustrations of Useful Arts, Manufactures and Trades*, 1867).

Josiah Wedgwood introduced finely powdered calcined flint into his white pottery in 1720. The high concentration of air-borne silica dust which resulted when the dry flints were ground and sieved produced lung diseases in the operatives. A wet grinding process was later adopted.¹⁵ Machines used in the crushing of calcined flints and the wet grinding of the crushed flints are shown in Figs. 1.1 and 1.2.

With the introduction of steam power in the nineteenth century there was a notable increase in silicosis, particularly in trades such as knife grinding. From the record of 61 persons engaged in this trade who died between 1825 and 1840, Holland¹⁶ found that 35 died before the age of 30.

The effects of the lethal granite dust in the Cornish copper- and tin-mines in the middle of the nineteenth century is evident from statistics given in Tables 1.1 and 1.2, quoted in the report of the

TABLE 1.1

*Extract from the Report of the Chief of the Statistical Department
in the General Register Office, 1863*

Out of 10,000 males who attain the age of 15, there were registered as dying from consumption and other lung diseases:

Age group	In England	Of Cornish miners	Difference
15	351	292	59 less
25	417	387	30 „
35	417	660	243 more
45	454	1,498	1,044 „
55	500	1,709	1,209 „
65 to 75	469	930	461 „
Total . . .	2,608	5,476	2,868 more

TABLE 1.2

*Extracts from the Report of the Chief of the Statistical Department
in the General Register Office, 1863*

Out of 10,000 males who attain the age of 15, there were registered as dying of the following diseases:

	In England all occupations	Miners in			
		Cornwall	Stafford	Durham	S. Wales
Diarrhoea and cholera . . .	547	230	1,052	1,255	1,915
Fever . . .	426	324	385	307	782
Diseased heart and dropsy . . .	928	484	612	928	333
Consumption . .	1,523	4,439	680	948	1,604
Other lung disease	1,343	1,157	1,975	1,010	1,433
Accident or violence . . .	532	782	2,782	1,312	2,158
All lung disease .	2,866	5,596	2,655	1,958	3,037
Lung disease and accident . .	3,498	6,378	5,437	3,270	5,195

Royal Commissioners appointed in 1862 to inquire into the health of workers in metalliferous mines. The figures, which refer to the years 1849-53, were given by the Chief of the Statistical Department in the General Register Office in 1863.¹⁷

There was doubt among the medical profession however as to how far "miners' disease" was due to dust.¹⁷ "If also the men inhale the smaller particles of granite and other hard and gritty earthy matters which are thrown off from the rock by their tools, such must certainly be serious sources of irritation to the delicate structure of the laryngeal and bronchial mucous membrane and to the substance of the lungs. Several of the men complained of the dryness and dustiness of the mines in which they worked, and one man said that 'the dust was far worse than anything else they had to contend with'. So far, however, as I was able to judge I should doubt whether this cause generally acts very unfavourably upon the men, or at least whether it is comparable in its effects to other injurious influences. Portions of the lung of John Francis were subjected to careful chemical analysis by Dr. Bernays, and no siliceous material was detected in them. In the French millstone makers, to whose peculiar condition I have elsewhere drawn attention, there can be no doubt that the sharp particles of siliceous matter which they inhale do find their way into the lungs and become sources of serious irritation; determining, in persons of healthy constitution, broncho-pneumonic attacks which may ultimately lead to disorganization of the lungs, or in persons predisposed to consumption, true tubercular phthisis. The Cornish miners, however, probably like the gunflint makers of Meusnes in France, suffer more from the impure air which they breathe than from the inhalation of particles of stone." The Royal Commissioners of 1862 decided that dust was not a major factor in the production of the widely prevalent phthisis, but a Departmental Committee of 1902¹⁸ investigating disease among the metal miners of Cornwall concluded that "it seems evident enough that stone dust which they inhale produces permanent injury of the lungs . . . and that this injury . . . predisposes enormously to tuberculosis of the lungs".

The interrelation of silicosis and tuberculosis, which is still not understood, was fully recognized during the last century as the quotation in the last paragraph shows. Aldridge (1892) suggested that the tubercle bacillus is always inactive unless the lung is previously damaged by an agent such as dust: "Pathologists tell us of the presence of bacilli in tubercular disease, and favour the belief that these minute bodies are the cause of it. This belief may represent a whole truth, or only a partial one; in my opinion, the latter. For I doubt if these bacilli actually develop phthisis unless there be

some antecedent change in the vitality of the affected tissue . . . assuredly the breathing of dust may be reckoned as one (contributory factor) of no slight energy."

In this country the importance of pneumoconiosis as an industrial disease in the refractories industry was officially recognized in 1918 when Parliament passed the Workman's Compensation (Silicosis) Act under the Factory and Workshop Act. Four years earlier the British Royal Commission on Metalliferous Mines and Quarries (Second Report) classified quartz, quartzite, flint and sandstone as substances which, when inhaled, cause excessive mortality from respiratory diseases and especially from phthisis. These minerals were contrasted with coal, shale, slate, iron ore, clay, limestone, plaster of Paris and cement which were not thought to be responsible for any respiratory disease.

Once the serious nature of the disease was recognized, precautions were taken by most industrial organizations throughout the world. Ventilation methods were generally improved and medical supervision was arranged. In at least one English firm a system of casual labour was adopted so that silica dust was not inhaled by any workman for more than a few months. In a few areas there was a refusal to recognize silicosis as an industrial disease. In France and Belgium, for example, a small but powerful group of mining companies and their medical advisers would not recognize the existence or the specificity of silicosis for some years. Martin¹⁹ described their opposition in *La Médecine du Travail* which journal, in 1937, devoted a whole issue to silicosis. The view forcibly stated there²⁰ was that silicosis is different from tuberculosis, that it presents a different X-ray picture and that it is found only where quartz-containing dust is inhaled.

In many industries enormous sums of money have been spent in combating the pneumoconiosis risk and in the payment of compensation. Some idea of the magnitude of the problem can be gained from the sums of money involved. The Rand gold-mines paid £12,000,000 in compensation during twenty years in the early part of the century, mostly to whites of whom about 15,000 were employed. Present-day methods for the selection of labour and the frequent medical examination of personnel who are withdrawn from the mines at the first sign of trouble, make it difficult to provide a comparable picture from more recent statistics.

The magnitude of the pneumoconiosis problem in different industries can be judged from Table 1.3. Table 1.4 gives the average age at death of persons reported to have died of pneumoconiosis contracted in several industries, and the number of years of employment in that industry.

TABLE 1.3

Number of death certificates in which is recorded fibrosis of the lungs, including all forms of pneumoconiosis as well as byssinosis

	England and Wales, 1943-1952										Eng. and Wales	Scot- land
	1943	1944	1945	1946	1947	1948	1949	1950	1951	1952	1953	1953
SILICOSIS:											PNEUMO- CONIOSIS	
1. Refractories industries	7	6	9	8	10	13	13	8	8	11	6	3
2. Pottery, manufacture of	41	32	41	49	54	48	63	73	62	70	79	—
3. Sandstone, quarrying and dressing	24	26	21	20	12	28	15	10	33	9	23	—
4. Stone masons	53	31	44	41	43	40	36	19	38	23	21	1
5. Metal grinding	11	7	11	6	9	8	14	8	12	14	15	—
6. Sandblasting	6	7	6	4	5	5	5	3	3	1	3	—
7. Steel dressing and casting	4	10	5	22	13	13	18	11	6	11	10	8
8. Stone, pebble, flint and sand crushing	1	4	—	—	3	1	3	3	1	2	4	—
9. Scouring powders, manufacture of	1	1	1	—	—	—	1	1	1	1	—	—
10. Abrasive wheel manufacture	1	1	2	1	—	—	—	—	2	—	1	—
11. Glass cutting and bevelling	—	—	—	2	—	—	—	—	—	1	1	—
12. Millstone dressing	—	—	—	—	—	—	—	1	—	1	—	—
13. Slate quarrying and dressing	6	7	6	13	20	16	21	17	32	35	36	1
14. Granite quarrying and dressing	2	—	1	—	4	1	1	3	3	9	8	7
15. Tunnel mining (sewage works, etc.)	4	4	4	5	—	3	2	—	2	1	1	2
16. Coal-mining	276	277	323	343	347	322	336	337	347	339	1,016	240
17. Gold-mining (South Africa)	4	9	4	7	5	4	4	6	2	2	2	—
18. Tin-mining	14	11	19	14	12	11	20	9	4	17	18	—
19. Iron ore (haematite)-mining	5	7	2	9	6	12	9	6	6	9	23	—
20. Lead-mining	1	1	1	1	2	—	1	—	1	1	2	1
21. Copper-mining	1	—	—	—	—	—	1	—	—	—	—	—
22. Barytes-mining	4	—	2	1	1	—	3	—	1	—	—	1
23. Clay-mining	1	—	1	—	—	—	1	1	—	1	—	—
24. Mining engineers	1	—	—	1	1	2	1	—	—	—	—	—
25. Miscellaneous	2	4	5	6	11	7	7	39	67	28	63	16
Total	470	445	508	553	558	534	575	555	631	586	1,332	280
ASBESTOSIS	8	10	11	16	15	15	17	12	18	12	14	1
PNEUMOCONIOSIS:												
Coal-mining	—	34	64	78	230	317	420	509	590	573	—	—
Other industries	—	3	11	5	12	19	14	26	58	44	—	—
BYSSINOSIS	7	1	10	3	4	8	7	11	8	13	22	—
OTHER CASES OF (NON- OCCUPATIONAL) FIBROSIS	493	531	529	568	616	651	554	679	732	677	687	16
GRAND TOTAL	978	1,024	1,133	1,223	1,435	1,544	1,587	1,792	2,037	1,905	2,055	297

The figures are based on information contained in death certificates as notified to the Registrar General.

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TABLE 1.4
Fatal cases investigated up to the end of 1953

	Number of deaths	Average age at death	Duration of employment in years		
			Longest	Shortest	Average
SILICOSIS					
Pottery:					
Silicosis	750	61.7	62.0	2.8	38.4
Silicosis with tuber- culosis	501	56.2	67.0	5.0	34.7
Sandstone:					
Silicosis	267	60.9	62.0	9.0	39.0
Silicosis with tuber- culosis	229	57.5	58.0	5.0	37.4
Grinding of Metals:					
Silicosis	123	59.5	61.0	14.0	35.9
Silicosis with tuber- culosis	211	54.0	56.0	2.8	33.0
Sandblasting:					
Silicosis	75	50.4	42.0	1.7	13.4
Silicosis with tuber- culosis	99	46.2	46.0	2.0	13.1
Manufacture of scouring powders:					
Silicosis	16	40.8	37.0	2.3	8.3
Silicosis with tuber- culosis	6	40.8	12.2	2.0	7.0
Miscellaneous:					
Silicosis	283	55.4	57.0	1.5	24.3
Silicosis with tuber- culosis	248	51.6	50.0	0.7	24.8
TOTAL:					
Silicosis	1,514	59.4	62.0	1.5	34.1
Silicosis with tuber- culosis	1,294	54.4	67.0	0.7	31.2
ASBESTOS					
Asbestosis	230	49.5	48.0	0.5	16.6
Asbestosis with tuber- culosis	87	40.2	33.0	0.8	11.4

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According to Collis and Yule²¹ the figures in the Report on the Occupational Mortality of Males (based on the 1921 census) show that, when deaths from all causes are considered, the general mortality rate among workers exposed to silica dust is considerably higher than that of the general population or that of workers in other comparable industries where the dust contains little free silica. As a basis of comparison for different groups, the number of persons of the general population in a particular age group which give 1,000 deaths is found. The same number of persons in other groups is then studied, the number of deaths giving the Comparative Mortality Figure (C.M.F.). Certain errors may occur; for example, sandstone workers may move to limestone quarries and "non-silica" dusts may contain a proportion of silica, but the figures show a sufficient contrast to suggest that these errors can be neglected.

Collis and Yule selected a group of workers who handled materials giving rise to silica dust (silica group) and another group who were employed in similar occupations, but who handled materials producing little free silica. Table 1.5 shows that the youngest section in both groups showed mortality figures below that of the general

TABLE 1.5

Comparative Mortality Figures (all diseases)
(Collis and Yule, *J. industr. Hyg.*, 1933, 15, 395)

Age group	Silica group	Non-silica group
20-24	965	872
25-34	1,310	879
35-44	1,870	928
45-54	2,200	979
55-65	2,222	1,016

Comparative figure for standard population at each age is 1,000.

population. The section of age 55-65 in the non-silica group had a slightly higher C.M.F. than the corresponding age group of the general population, but this age group of the silica group showed a C.M.F. more than double that of the general population. Table 1.6 compares the death-rate from respiratory tuberculosis in the silica group, in the non-silica group, and in a group of agricultural labourers with that of the standard population. The C.M.F. is far higher for the silica group. The C.M.F. for a few comparable trades in the silica and non-silica groups are given in Table 1.7. It is again

apparent that the mortality is considerably higher in any trade if silica dust is formed.

TABLE 1.6

Comparative Mortality Figures (Respiratory tuberculosis only)
(Collis and Yule, *J. industr. Hyg.*, 1933, 15, 395)

Age group	Silica group	Non-silica group	Agricultural Labourers
20-24	1,244	661	777
25-34	1,638	700	800
35-44	3,232	1,057	475
45-54	5,426	1,447	484
55-	6,643	1,318	467

Comparative figure for standard population at each age is 1,000.

TABLE 1.7

Comparative Mortality Figures (Silica and non-silica groups)
(Collis and Yule, *J. industr. Hyg.*, 1933, 15, 395)

	C.M.F.
SILICA GROUP	
Tin and copper mine—underground workers, not superintending staff	4,335
Potters' mill workers; slip makers; potters	1,642
Earthenware, china, etc.; kiln and oven men, and kiln setters and placers	1,830
Metal grinders	1,977
Sandstone miners and quarriers	1,644
Sandstone masons, cutters and dressers	2,068
NON-SILICA GROUP	
Brick and plain tile makers, moulders, etc.; furnace and crucible pot makers	926
Brick, tile, etc.; kiln and oven men	878
Iron ore mine—underground workers, not superintending staff (Staffordshire and North Riding, Yorkshire)	792
Limestone miners and quarriers	918
Limestone masons, cutters and dressers	1,197

Comparative figure for standard population is 1,000.

With the changing pattern of industry, many new raw materials have been introduced in recent years. Some of these are known to be