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ADDRESSES AND ORIGINAL ARTICLES

INDUSTRIAL PULMONARY DISEASE DUE TO THE INHALATION OF DUST

WITH SPECIAL REFERENCE TO SILICOSIS *

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IN the first lecture the characteristics of the disease known as silicosis were outlined, and it was pointed out that the essential lesion is the fibrous nodule. Certain of the occupations in which the disease occurs were referred to, and it was found they involved exposure to dust of free silica, as flint or quartz. In some occupations free silica was the only important constituent of the dust; in others it was mixed with other dusts, and in the case of granite, which contains large and variable proportions of silicates, there appeared a tendency to the formation of a linear or diffuse type of fibrosis. This type of fibrosis was seen in the radiogram of a sagger-maker, in the pottery industry. The man was 65 years of age and had been employed in sagger-making for 53 years, and he would be exposed to dust of fireclay at his work. A radiogram taken five years later showed some increase of the shadows but no evidence of nodulation.

We may now pass on to the consideration of some industrial processes in which the workers are exposed to dust of silicates, that is, chemically combined silica. From the point of view of health by far the most important of the silicates is asbestos.

Asbestos

Asbestos is a commercial term applied to a group of minerals possessing the common property of finely fibrous structure. The most important varieties are chrysotile, a silicate of magnesium with little iron, crocidolite and amosite, silicates of iron with smaller amounts of magnesium. In typical samples of asbestos the fibres can be separated to an extreme degree of fineness, while retaining considerable length; this property renders the material capable of being carded, spun into yarn, and woven into cloth.

The asbestos is usually received in this country, chiefly from Canada, Russia, Italy, Rhodesia, and the Cape Province, in the crude form consisting of the crushed rock, and the subsequent processes in its manufacture include disintegrating and opening the fibre, carding, spinning, and weaving the textile materials, or grinding and mixing with other substances in the manufacture of insulating slabs and compounds, cement sheets, and a great number of other articles used in many industries.

The dust given off from asbestos in processes of manufacture consists of fragments of fibres and small particles of rounded or angular shape. The following counts were made by Watson from samples taken in a factory where three varieties of asbestos were being used (see Table).

The median sizes of *particles* in five counts varied between 0·46 μ and 0·8 μ diameter, and the percentage distribution of sizes was 32 to 54 per cent. less than 0·5 μ and 96·6 to 99·9 per cent. less than 5 μ diameter.

The median length of the *fibres* in five counts was between 2·65 μ and 3·5 μ, and the percentage distribution of lengths of fibres was 8 to 16 less than 1 μ, and 87 to 92 less than 10 μ.

The distribution of diameter, size, or thickness of *fibres* in one sample was: median diameter 0·56 μ, 5 per cent. less than 0·2 μ, 43 per cent. less than 0·5 μ 98 per cent. less than 2 μ.

The modern development of the industry has a comparatively short history. The history of the disease asbestosis is correspondingly short. The first recorded case was described by Dr. Montague Murray.[75] It was a man employed 13 or 14 years in the industry, the first 10 years in the card-room, and he was the last survivor of 10 workers in that room. That was in 1899. The diagnosis was verified by Dr. Murray after post-mortem examination. Sections of the lungs made about 1925, from this case, show "asbestosis bodies." Fahr [76] described a case of asbestosis in 1914. Merewether [77] records the few cases published in the literature between that time and 1924, when Cooke [78] described a case which has been followed by an incidence of several in each successive year.

—	Process.	Asbestos.	Position.	Particles. (In 1 c.cm.)	Fibres. (In 1 c.cm.)	Total.*
1	Carding.	White.	B.L.	235	180	415 (12)
2	"	"	G.A.	120	95	215 (6)
3	Doubling.	Blue.	G.A.	105	95	200 (6)
4	Spinning.	"	G.A.	115	120	335 (9)
5	Weaving (cloth).	White.	B.L.	240	85	325 (9)
6	"	"	G.A.	55	40	95 (3)
7	Weaving (brake-lining).	"	B.L.	140	70	210 (6)
8	"	"	G.A.	130	55	185 (5)
9	Winding.	Blue.	B.L.	200	240	440 (13)
10	Plaiting.	White.	B.L.	120	50	170 (5)
11	"	"	G.A.	120	85	205 (6)
12	Mattress making.	Blue.	B.L.	405	820	1225 (35)
13	"	Various.	G.A.	35	25	60 (2)
14	Bagging.	Amosite.	G.A.	210	210	420 (12)
15	Disintegrating.	"	G.A.	190	260	450 (13)
16	Making sections.	"	B.L.	690	1630	2320 (66)
17	"	"	G.A.	150	170	320 (9)
18	Pipe making.	Blue.	B.L.	90	190	280 (8)
19	"	Amosite.	B.L.	110	215	325 (9)
20	"	"	G.A.	80	135	215 (6)

B.L. = breathing level of worker; G.A. = general air of the room.
* The figures in parentheses represent the number of millions of particles in a cubic foot of air. (See Lecture I., p. 2.)

During the last ten years an extensive literature has been produced on this subject, and it will suffice for the present purpose to mention briefly some of the features in which this disease differs from silicosis.

Clinically, the signs of asbestosis are slight in the early stages. The striking symptom is dyspnœa of gradually increasing intensity as the disease progresses. The radiographic appearance is typically that of a fine type of fibrosis of the lungs occupying the whole of both lung fields, and more dense at the bases. The individual shadows are minute, lobulated, superimposed but not tending to form massive coalescence, and the lung fields present a veiled or ground-glass appearance. A characteristic feature is the "shaggy" outline of the heart shadow. The nodular shadows seen in silicosis are not present in uncomplicated asbestosis.

Post mortem the appearance of the lungs is quite unlike that in silicosis. Pleural adhesions are usually extensive and dense, but the surface of the pleura shows no nodulation and nodules are not seen or felt on the cut surface of the lung. The consistence of the whole lung is increased, and large areas may show fibrous condensation, which is tough rather than hard as in silicosis. Parts of the lung least affected by the fibrotic change are often congested

* The Milroy Lectures for 1936 delivered before the Royal College of Physicians of London on Feb. 27th and March 3rd. The first lecture appeared in THE LANCET last week.

or pneumonic. Emphysema is notably localised in areas and bullous in form, usually at the anterior and lower margins, and at the extreme apices a honeycomb-like patch of emphysematous bullæ may be seen. A partial pneumothorax is present in some cases.

Histological examination of the lung reveals the most characteristic features of the disease. There is generalised fibrosis and the alveolar walls show diffuse thickening.[79] Everywhere in the pulmonary tissue are to be seen fibres of apparently unaltered asbestos, isolated and in clumps, and the "asbestosis bodies" so well described by Gloyne [80] and Beger.[81]

Asbestosis bodies have been found in the sputum by Stewart and Haddow,[82] Simson and Strachan,[83] Page,[84] and others and by exploratory lung puncture (first suggested by S. A. Henry). The presence of asbestosis bodies in the fæces was described by Gloyne.[85]

In the five-year period, 1930–34, a total of 50 deaths were certified in which asbestosis is mentioned as a cause of death. In addition 4 deaths must be added in which asbestosis was known to be the cause of death, although it is not mentioned on the certificates. These 54 cases occurred in the five consecutive years in the following numbers: 8, 9, 7, 15, and 15. There is therefore no evidence of diminution in numbers.

Of the 54 deaths 31 were females and 23 were males. The average age of the females was 32·6 years and of the males 46·7 years. The seasonal incidence was irregular, being highest in January and October, with 8 each; next in order are February and June with 6 each; March, August, and December with 5 each; May with 4; April, July, and September with 2 each; and November with 1. In 13 cases asbestosis was given as the only cause of death: 8 females, 5 males. In 24 cases tuberculosis was also present (44·4 per cent.): 16 females and 8 males. Broncho-pneumonia was present in 6 cases (11 per cent.): 4 females and 2 males; cardiac disease or cardiac failure in 4 cases: 1 female and 3 males; cancer of the lung in 3 cases: 2 females and 1 male; lobar pneumonia in 2: both males; and bronchitis and nephritis, 1 each: both males. In 43 deaths from asbestosis in the four years from June, 1931, to June, 1935, the periods of employment in the industry ranged from 10 months in distintegrating, to 23 years on weaving; the average duration of employment for the 43 fatal cases was just under 10½ years.

The relationship between asbestosis and tuberculosis has been discussed by Merewether [86] and has been commented on by numerous writers. The general experience is that tuberculosis is by no means an infrequent accompaniment of asbestosis, while the opinion is general that it is less frequent than with silicosis. In the present series of 54 fatal cases of asbestosis, tuberculosis was present in 24 (44·4 per cent.). In a series of 67 fatal cases collected by the Factory Department [87] the number with tuberculosis is 26 (38·8 per cent.). In 100 cases collected by Burton Wood and Gloyne [88] there were 21 (21 per cent.) with tuberculosis. Only 26 of their series were fatal, and, in view of the higher proportion found with tuberculosis in the fatal groups, it may be that active tuberculosis is more frequent as a terminal complication than as a concomitant disease during earlier stages.

The difference in the reaction of the tissues to silica and asbestos has been demonstrated by experiment. Guinea-pig experiments made by Gloyne [89] with asbestos showed that injection of the dust was not followed by an acute inflammatory reaction; a similar observation was made by Policard.[90] Gardner and Cummings [91] found that asbestos was concentrated in the respiratory bronchioles and their lateral alveoli, and fibrosis was first manifested after 500 days' exposure. Quartz, on the other hand, penetrated to the terminal alveoli of the lung, was rapidly concentrated by migrating phagocytes in the pulmonary and mediastinal lymphoid tissues, and provoked an early and rapid multiplication of fibroblasts. The German workers quoted by Gloyne [92] suggested that the silicate is broken down into silica in the tissues. Beger and Giese took a similar view and explained the slower action of silicates compared with free silica in that way. Koppenhöfer [93] points out that the magnesium and iron are dissolved out and are replaced by alkalis which combine with the silica. The silica then undergoes solution. Fowweather [94] records the result of chemical analysis of the lungs in a fatal case of asbestosis, in which the amount of ash, in excess of what would be present in normal lungs, was wholly accounted for as silica. He drew the tentative conclusion that the asbestos had undergone decomposition in the lung with deposition of SiO_2 and removal of the metallic oxides.

Sillimanite

This is anhydrous aluminium silicate, with the formula Al_2SiO_5. It occurs as rock of great density, imported from India, and it is used as a refractory material. The broken surface shows masses of prismatic crystals in the form of fibres, which do not appear to be capable of division into extremely fine fibres like asbestos, but they tend to fracture and, when crushed, to be reduced to powder. The processes of manufacture include crushing, grinding, and sieving the rock, usually after it has been calcined. and considerable quantities of dust are given off. The results of exposure to the dust of sillimanite are of special interest, because of the fibrous form of the mineral, in which it resembles asbestos. Few persons are employed in the industry at present.

At a factory where 15 men are employed, 13 of the workers were examined clinically and radiologically, and a complete occupational history was taken, with the assistance of my colleague Dr. Henry, and the coöperation of the employers. None of the workers had any previous exposure in a dusty occupation. The ages ranged from 24 to 50 years, and the duration of employment in the industry from 1½ to 16 years. From examination of the 13 radiograms it was evident that no striking changes had occurred in the lungs of the men examined. In 4, employed respectively 16, 11, 7, and 5 years, there was some slight change which might be due to dust. This was not supported by clinical evidence, except that in the first there was slight bronchial catarrh. In 3 others, slight changes found might have been due to antecedent disease. In all the others hilum shadows were increased in extent or density, and in 4 of them there were also increase of the linear shadows and of the pulmonary reticulum.

The numbers are too small to support any final conclusion, but within the limits of the material of the investigation it does not appear that dust of sillimanite is highly injurious. It cannot be said, however, that injury to health would not be caused by longer exposure, or that it might not become apparent after a lapse of time from the period of exposure.

China-clay

China-clay, or kaolin, is hydrated silicate of aluminium, $H_4Al_2Si_2O_9$. It is formed by the decomposition of felspar, by the removal from it of silicate of potassium through the action of water containing carbon dioxide. The largest production of china-clay in the world is in Cornwall, where it occurs in deep pits from which it is washed with water under pressure. There is exposure to dust in shovelling the clay after it has been dried in the kilns.

China-clay is an important ingredient in china and earthenware pottery, and it is used in the manufacture of paper, cotton-cloth, paint, soap, and a large number of other commodities.

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There are no satisfactory data regarding the effects of china-clay on the human lungs, but amongst both workers and management, engaged in the industry in Cornwall, there is a general belief that no injury to health follows exposure to the dust and that respiratory disease is not unusually prevalent.

Dr. Chown, late medical superintendent of the sanatorium at Tehidy, kindly let me see X ray films of 4 clay-workers. Two of them showed tuberculosis only. A radiogram from a third man, aged 37, in whom there were no conclusive signs of tuberculosis, showed only increase of hilum, linear, and reticular shadows. The fourth radiogram of a man, aged 49, with no definite signs of tuberculosis, showed the whole of both lung fields covered with extremely fine mottling, most marked in the middle zones and at the right base. The film suggests a pneumoconiosis, not of silicotic typ... Later, Dr. Chown sent me a radiogram of another clay-worker, aged 40, employed for 19 years in the "dry" or kiln, where he would be exposed to dust of china-clay. He had tuberculosis with tubercle bacilli in the sputum. The radiogram shows evidence of mottling of silicotic type.

These radiological appearances recall the results of animal experiments obtained by Kettle,[95] in which china-clay alone produced only cellular reaction with the formation of plaques of dust and cells in the alveoli, while china-clay with dead tubercle bacilli caused the formation of fibrotic tissue in the form of the silicotic nodule.

Siegmund [96] has suggested that a tuberculous process may influence the changes which occur in mineral substances in the tissues.

Talc and French Chalk

In its compact form the mineral talc is known as steatite or soapstone. It is a hydrated magnesium silicate $H_2Mg_3Si_4O_{12}$. In powder form it is used as a filler for paper, soaps, and paints, and for leather dressing, and as a lubricant. It is also used in making electric switchboards and for heat insulation of steam-pipes. A fine granular variety is known as French chalk.

Merewether [97] examined 11 workers who were exposed to dust of French chalk in the tyre department of an india-rubber works, for periods varying from 10 to 32 years. He found that while the radiographic appearances suggested the presence of a developed, diffuse, interstitial fibrosis of the lungs with under 10 years' exposure, the clinical examination and other factors indicated that even after 30 years' exposure, only a peribronchial increase of fibrous tissue had resulted.

The suggestion is that the radiographic appearances seen really reflect the actual dust in the lungs, together with any associated congestion, rather than the presence of a diffuse fibrosis. The few examinations made so far did not disclose any appreciable disablement after exposures ranging from 9 to 32 years.

Examination of 13 additional workers, with exposures ranging from 4½ months to 40 years, supported the previous conclusion.[98]

Zanelli [99] described the case of a woman of 27 who had been employed for five years in the tyre department of a rubber factory where she was exposed to clouds of dust. The radiographic pictures showed an early tuberculous infiltration in the right subclavicular region, decrease in transparency of right compared with left due to increase in reticular shadows and nodular formations uniformly distributed through the right lung. In the left lung field the reticular shadows were accentuated, but there were practically no "nodules."

This case seems to show that the interaction of tuberculous infection with the silicate dust produced a radiological appearance of "nodules" of infective type in the right lung. It resembles the reaction with tubercle and china-clay.

Thorel [100] described in 1896 a case of talc pneumoconiosis which he designated *Speicksteinlunge*. Sand described the case of a woman who had worked for several years in an atmosphere of talc dust. *Post mortem* the lung showed the presence of much talc dust but without great reaction. This case was reported by Devoto and Cesabianchi.[101] Cunningham [102] examined a man employed for nine years grinding talc, and found evidence of pneumoconiosis. Dreessen [103] described the results of an investigation into working conditions in a tremolite talc-mine and mill and the effects on the health of the workers exposed to the dust. Those employed over five years (the longest was 16 years) and exposed to very high concentrations of dust showed first stage pneumoconiosis. The appearances were those of a fine, diffuse bilateral fibrosis. The changes did not cause disability. Dreessen and Dalla Valle [104] carried out a further survey of two talc-mining and manufacturing plants in Murray County, Georgia. Clinical and radiological examinations showed that 16 mill-workers out of 33 exposed to high concentration of dust and 6 of 13 miners had pneumoconiosis.

In 4 of the most seriously affected cases evidence of pulmonary infection was present.

Mica

A group of widely distributed rock-forming minerals, two of which, muscovite and phlogopite, are important commercially. Muscovite is a silicate of aluminium with potassium. It is used, on account of its transparency and its resistance to fire, for windows of stoves and lanterns. Ground mica is used in the manufacture of paper, wall-paper, and paint and as a lubricant and absorbent. Phlogopite is magnesium-aluminium silicate. It is almost exclusively used for electrical purposes. Dust is produced especially in the processes of sawing, grinding, and finishing.

Ferguson [105] examined 12 workers exposed to mica dust. In only 5 was the exposure as much as five years and these complained of cough and dyspnœa, the symptoms being slight except in one man, aged 59, who had been employed 32 years. He had well-marked pulmonary fibrosis and emphysema, and another, employed eight years, had quite definite fibrosis. Three men were examined radiologically. The radiogram of one man, employed 32 years, showed fibrosis, largely peribronchial in type, chiefly in the middle zones, with increased hilum shadows and, at one or two points, nodule formation. The other two radiograms showed increased hilum shadows with increased linear striation and fine, diffuse shadows in the middle zones. Ferguson, with a reservation regarding the small numbers, concluded that dust of mica may be capable of causing pulmonary fibrosis.

Sericite

A mineral of widespread occurrence, sericite is of interest in a study of the ætiology of silicosis because of its frequent association with quartz in rocks, exposure to the dust of which has caused the disease. It is a hydrous silicate of aluminium and potassium ($H_2KAl_3(SiO_4)_3$) and is produced in the decomposition of potash felspar. It occurs in two forms—silky, talc-like plates and minute needles. The occurrence of sericite in the gold-bearing banket of the Witwatersrand was described by Watkins-Pitchford and Moir.[106] They thought it "almost certain that a large proportion of the bright white images seen by polarized light in a silicotic-lung section of *Rand origin* are produced by sericite." Mavrogordato [107] included "intractable silicates, e.g., sericite," as a possible factor in the occurrence of silicosis.

Badham [108] suggested that the fine type of fibrosis of Western Australian miners is probably related to

sericite which is found in quantities greater than quartz in some of the mines.

It was not, however, until W. R. Jones published his paper in 1933 on Silicotic Lungs: The Minerals they Contain,[109] that sericite became familiar to everyone interested in the ætiology of silicosis. By petrological methods, he found the mineral, sericite, in rocks and materials which had given rise to silicosis, and in the lungs of 29 men who had died from the disease. Although quartz was also present it was subordinate in amount to sericite, especially in number of particles.

In the South Wales coal-mines and the gold-bearing quartz-conglomerate of South Africa, abundant sericite was shown to be present. The experience at the Broken Hill mines in New South Wales was cited to show that rocks with a low quartz content, but with fibrous silicate minerals, produced cases of silicosis. The physical property of sericite fibres, by which they remain suspended in the atmosphere, was demonstrated to show the opportunities for inhalation of the mineral.

The only industries in this country in which it appears that workers are exposed to dust of sericite without uncombined silica are those connected with the production of china-clay and manufactures in which it is used. The evidence at present available is insufficient to allow of a definite statement regarding the occurrence of pulmonary disease amongst these workers, as no systematic examinations have been carried out. The opinion of employers and workers in the china-clay industry is against the view that a high incidence of such disease is prevalent, and medical evidence is negative apart from the cases already mentioned.

Certain pulmonary changes have been produced by silicates, and, apart from asbestos, individual cases of fatal disease have occurred in which the dust of silicates seemed to be the cause. The disease, however, did not show the radiological or pathological signs which are characteristic of silicosis. This was the experience in South Africa in workers exposed, in a quarry, to dust of norite,[110] and in New South Wales with an orthoclase basalt,[111] and in the Great Cobar mine. Dr. Badham also described a fine type of fibrosis amongst the miners of Broken Hill, and of Western Australia and Tasmania.

Identity of Harmful Dusts

Kettle [112] utilised the interstitial method for determining the tissue reaction to various kinds of dust, and the influence on the local lesion of infection with tuberculosis. In these experiments "active" and "inert" dusts were distinguished. Colloidal amorphous silica caused necrosis; with crystalline silica necrosis was delayed, and the characteristic lesion was produced in 2 or 3 weeks.

Dust from a South African gold-mine caused lesions which differed scarcely, if at all, from those caused by amorphous silica. Shale from Altofts and Great Mountain collieries gave the characteristic reaction of silica, though modified by the high admixture of inert material. Kaolin had all the attributes of a soluble form of silica, while the lesion produced by asbestos mimicked that of crystalline silica but the central coagulum was more pronounced. The lesion produced by carborundum was remarkable for its quiescence, and non-siliceous dusts characteristically showed quiescence. Following inoculation with tubercle bacilli the "active" dusts were: all compounds of silicon except carborundum—i.e., colloidal, amorphous, and crystalline silica, gold-mine dust, shale, kaolin, and asbestos.

A method was introduced by Kettle [113] for administration to experimental animals by the tracheal route of dust suspended in liquid. By this method [114] no substance other than silicon derivatives was found which caused more than a moderate degree of fibrosis of the lung, certainly not enough to interfere with its function, and no other constituent of ordinary dust which could influence unfavourably a pulmonary infection. All forms of crystalline silica appeared to be capable of producing the disease, and the closest reproduction of simple silicosis in man, so far obtained, occurred in guinea-pigs some 500 days after the introduction of a single dose of finely powdered flint into their lungs.

According to Gardner [115] the reaction to dust may be divided into five general phases: (1) early diffuse parenchymatous disease; (2) linear perilymphatic proliferation; (3) beading of the trunks; (4) enlargement of the mediastinal lymph nodes; and (5) late chronic proliferation and nodule formation in the finer connective tissues of the pulmonary parenchyma. Different phases of this picture are accentuated by various types of pure dust. Silica produces all of them. Pure fibrous asbestos produces generalised fibrosis involving primarily the finer connective tissues of the pulmonary framework. He [116] found that silicon carbide (carborundum) excited a moderate degree of parenchymatous reaction and some linear fibrosis. The other common industrial dusts generally set up a reaction that is characteristically linear, with more or less diffuse changes in the parenchyma but no nodule formation.

Beger [117] expressed the view that the action of dust is due to free silica, whether it contains quartz or silicates. He explains the findings of W. R. Jones by the slow solution of sericite, which he does not consider free from danger, though only the dissolved silica is the cause of the disease.

Siegmund [118 119] produced positive results by the intravenous injection of quartz. Typical silicotic lesions were produced in the liver, spleen, and other organs by dusts containing quartz, but not with quartz-free dust. Silicates produced the changes only after ten months; the delayed action is explained as due to dissolving out of silica. The fibrosis increased with a decrease in size of the quartz particles. Experiments with colloidal silica produced the greatest change.

Giese [120] had obtained similar results by injecting quartz into the blood stream, and negative results were obtained with dusts free from silica.

Stüber,[121] on the suggestion of Dr. C. K. Drinker, used the lymphatic glands as a rapid means of determining the toxicity of dusts. The cellular response of the lymph nodes to dusts of flint (99 per cent. free silica) and Barre granite (26·5 per cent. free silica) was peculiar in the appearance of "silica-cells," while some silicates, including asbestos, did not have this effect. Using this method, Drinker, Field, and Drinker [122] found that after 10 or 12 days the changes produced by two samples of sericite were similar to those of finely ground flint.

Haynes,[123] as a result of inhalation experiments on guinea-pigs, concluded that the most deadly of all dusts was precipitated silica. Less dangerous, but all producing fibrosis, were the following, arranged in order of decreasing toxicity: flint, slate, aluminium hydroxide, precipitated chalk, magnesium carbonate, and carborundum. The harmful effects of soluble silica may be neutralised by simultaneous administration of basic dusts such as aluminium hydroxide or magnesium carbonate, though the latter are harmful when inhaled alone.

Robson was struck by the fact that the incidence of silicosis was higher amongst the underground

miners in certain gold-mines in Ontario, than amongst the men employed in the crusher-houses, although the dust in the latter was greater in amount. The underground men suffered from headaches in the gassy atmosphere after blasting, and this suggested experiments to ascertain the effects of mine-gas on the æetiology of silicosis. With Irwin and King [124] experiments were carried out at the Banting Institute. Rabbits were exposed to the inhalation of irritating gases (NO_2 and SO_2); these produced degenerative lesions and predisposed to pneumonitis; dilute irritating gases combined with sericite dust produced no fibrotic changes in an exposure of 40 weeks; dilute irritating gases combined with silica dust gave rise, in periods of 13 weeks or more, to an acute pulmonary silicosis with typical silicotic nodules in the alveoli, in the lymphatic aggregations, and in the lymphatic glands.

Perrott, Babcock, Betting, and Jones [125] found oxides of nitrogen in mine drift air in concentrations averaging 250 parts per million. Gardner, Howell, and Jones [126] found sulphur dioxide in concentrations of from 100 to 900 parts per million.

In several anthracite mines in South Wales, where cases of silicosis had occurred, twenty samples of mine-air were taken at the working faces, just after blasting, and were tested for nitrogen peroxide.[127] In no case did it exceed 90 parts per million, and even this concentration could not have been present for more than 3 or 4 minutes after each shot. An exceptional figure, nearly 200 parts per million of nitrogen tetroxide, was found in another series illustrating deficient ventilation.

PERITONEAL RESPONSE TO VARIOUS DUSTS

Miller and Sayers [128] carried out a series of experiments in which a small amount of dust (0·2 g.) in suspension was injected into the peritoneal cavity of guinea-pigs. The response could be classified as absorption, proliferative, or inert reaction. In the *absorption* response the dust disappeared from the peritoneal cavity without the production of scar tissue. This was given with calcite, limestone, precipitated calcium carbonate, gypsum (calcium sulphate), and cement. It will be noted that these are all calcium compounds. In the *inert* reaction the amount of injected dust was unaltered, but there was active movement, by the action of phagocytes, over extensive areas of the peritoneum. This reaction was given by soapstone (talc or french chalk), carborundum (carbide of silicon), jewellers' rouge (ferric oxide), anthracite and bituminous coal, and ash, collected from a smoke stack effluent. In the *proliferative* reaction the nodules produced by the dust continued to increase in size up to 112 days after injection, the maximum duration of the tests, and it seemed likely that scar tissue would ultimately have resulted. Two samples of pure quartz produced this response. "Chat" (a waste product containing quartz and chert from the concentration of lead and zinc ores) gave a similar response.

While the histological variations in the three groups were not as striking as the well-marked gross responses, they were sufficiently distinctive to permit differentiation of the dusts into the same three groups.[129]

CHEMICAL ANALYSES OF LUNGS

Kettle and Archer [130] suggested that chemical analysis may prove to be the deciding factor in determining, in the absence of a typical fibrosis, whether or not tuberculosis of the lung may be regarded as due to occupation. In their experience an ordinary dried lung does not contain more than 0·2 per cent. of silica (that is, total silica).

Sladden [131] concluded that fibrosis of an important extent, clearly contributory towards death, is usually present when the silica content of the lungs exceeds 1 per cent. of the dried lung substance. When the silica exceeds 1·6 per cent. the fibrosis associated is, with practically no exception, very severe and sufficient in itself to lead to death. The 60 cases on which this conclusion was based included hard-heading workers in coal-mines, coal-hewers, coal-trimmers, stonemasons, and others.

Badham and Taylor [132] described a method for chemical analysis of lungs, and the results of analyses of the lungs of 31 persons. They showed that the pathological findings varied markedly and without any constant relationship to the silica content of the lungs. In 4 cases without exposure to dust, the lungs contained an average of 0·123 per cent. of silica in the dried tissue.

McNally [133] reached the conclusion that the silica content of the normal lung was 0·113 per cent. of dried tissue. In a series of eight lungs from workers exposed to dust the percentage of silica varied from 0·24 in a machinist to 2·60 in a granite-cutter, while a stone-quarryman's lung gave 0·50, a stone-cutter's 1·40, and a zinc-miner's 1·09. Fowweather [134] found in the lungs of 5 normal adults between the ages of 23 and 65, a silica content from 0·12 to 0·20 per cent., with an average of 0·163 per cent. He found no obvious connexion between the amount of silica in the lungs and the amount of fibrosis.

A series of 44 analyses of lungs was carried out by Dr. P. L. Sutherland for the Committee on Industrial Pulmonary Disease. There were 21 stonemasons in this group, and in these the average content of total silica in the dried lung tissue was 1·28 per cent. In a group of 13 sandstone-quarrymen the average silica content of the dried lungs was 1·72 per cent. The highest silica figure in this series, 7·04 per cent., was found in the lungs of a miner, aged 61, who had worked on ganister and coal, in the same mine, for 49 years. Both lungs were the seat of carcinoma, and small silicotic nodules and some consolidation were present.

Recent work by Belt, Irwin, and King [135] shows that the proportion of silica varies in different portions of the lungs of persons who have not been exposed to a dust hazard. They found that in normal adults' lungs the "silica" content averaged about 151 mg. per cent.; and 217 mg. per cent. in persons over 52 years of age. The tracheo-bronchial glands of most adults contained from twice to 20 times as much silica as the corresponding lungs.

It is generally found that the proportion of total silica in a normal adult lung is between 0·1 and 0·2 per cent. of the weight of dried lung. There appears to be no constant relationship between the amount of fibrosis and the proportion of silica in altered lungs. The presence in such lungs of large amounts of other dusts appears to be accompanied by retention of silica.

During the time after the exposure to dust has ceased and before death, the amounts, relative proportions, and even the character of the minerals become altered by the action of the tissues.[136]

Tideswell [137] examined a series of silicotic lungs with a view to determining the form in which the silica occurred and particularly to attempt to estimate the hydrated silica. He found that approximately 5 per cent. of the total silica in the lung examined was present in a readily soluble form.

King and Dolan [138] have shown that silica reaching the blood stream, either by absorption from the gut or by solution in the lung, is rapidly excreted in the urine. Soluble silica injected into the blood is quickly eliminated in the urine. There appears to be a low kidney threshold for silica. No significant differences were found in the silica level in the blood of normal and silicotic persons.

Removal of silica from the lungs by continued activity of phagocytes is recognised, and the presence in the sputum of dust-laden cells long after exposure to the source of dust has been frequently observed. Belt [139] observed that an œdematous state of the tissue favoured the activity and migration of dust-laden cells several months after the exposure to dust inhalation had ceased.

Conclusions

Certain conclusions regarding the ætiology of silicosis may be drawn from the consideration of the occupations in which the disease occurs.

(1) Where silicosis has been caused, that is, the nodular form of pulmonary fibrosis, exposure to the inhalation of silica in the uncombined state appears to have been present.

(2) With a dust-cloud of moderate concentration, the disease develops slowly and the nodules of fibrous tissue appear at certain situations in the lungs. This occurs in grinders of metal, workers on sandstone, and in pottery workers.

(3) When the dust contains a very high proportion of free silica, and is in a fine state of division and the concentration of particles in the atmosphere is high, the disease tends to develop rapidly. In such cases the fibrosis retains the nodular form and develops at the usual sites, but it appears also in other parts of the structure of the lungs. This has been seen in flint-grinding, sand-blasting, and abrasive-soap manufacture.

(4) When the dust of free silica is inhaled mixed with certain other kinds of dust in important amount, the arrangement and distribution of the resulting fibrous tissue may be modified. Such modifications have been found in workers in coal-mines and hæmatite-mines.

(5) When the inhaled dust consists of silica combined with bases, as silicates, some degree of change in the pulmonary tissue appears to result. In this respect asbestos dust is unique amongst silicates in the prevalence and severity of the disease which it causes. The physical form of asbestos differs from that of all other industrial dusts.

(6) The fibrosis produced in the lungs by the action of silicates differs from that produced by free silica, and the types can be distinguished by radiological and histological means.

(7) In certain states of the lungs, for example in the presence of tuberculosis, the action of silicates may be modified.

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GOOLE HOSPITAL.—Lady Eaglesome is laying the foundation-stone of the extensions of the Goole Bartholomew Hospital on Saturday. The extensions, which will cost about £11,000, will include a new ward, a modern operating theatre, and a laundry. Some £2500 is already in hand.