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DISEASES OF THE LUNG RESULTING FROM OCCUPATIONAL DUSTS OTHER THAN SILICA*

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It is only in recent years that considerable interest has been given to the dusts to which men are exposed at their work, even though silicosis is known to have occurred in prehistoric times. Many dusts are now recognized as dangerous, and in the extreme it may even be doubted whether any dust can be regarded as harmless. It is rational at least to suppose that the lung cannot become a physiological dust trap and yet retain its elasticity. It seems possible that any dust, no matter how innocuous in small concentrations, would in large enough quantity eventually overwhelm the defences of the lung and accumulate in such amounts as to impair function; such a form of lung disease would be the result of causes of a mechanical nature—the physical presence of large amounts of inert foreign material. The term "benign pneumoconiosis" has been given to this type of disease in order to contrast it with diseases resulting from the inhalation of siliceous matter. But besides this group of conditions occupational dust may give rise to inflammatory lesions, allergic responses, and neoplastic changes.

Inflammatory Changes

Inflammatory changes may be caused by inorganic metals, such as manganese, beryllium, vanadium, and osmium, giving rise to a chemical pneumonitis; and by organic matter, such as decaying hay and grain, bagasse, cotton fibre, and similar substances where the aetiology is somewhat obscure, though fungi are frequently blamed.

Manganese

Manganese occurs in nature as the oxide in the ores pyrolusite, braunite, hausmanite, and manganite, which are found in Russia, India, the Gold Coast of Africa, Brazil, the United States, and Cuba. Manganese appears to be an essential substance in plant and animal nutrition. Its most important alloys are spiegeleisen and ferro-manganese, but silico-manganese and silico-spiegel are used in certain grades of steel. Manganese bronze is used for marine construction and mining machinery where corrosion is serious. The

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metal is also alloyed with aluminium, tin, arsenic, antimony, bismuth, and boron. Manganese oxide is used for bleaching glass, manganese chloride is used in dyeing, the sulphate in calico printing, and the persulphate as an oxidizing agent in making organic products; while manganates and permanganates are used for preserving wood, for bleaching textile fibres, and for disinfecting and oxidizing purposes. Chronic manganese poisoning was first described in 1837 by Couper, and since that time some 353 cases have been reported. The commonest pathological changes are those which occur in the central nervous system and result in the Parkinsonian syndrome. Accounts of the condition have mostly come from Europe, but cases have been reported in England by Charles (1927) and Owen and Cohen (1934), while recently a full review has been produced by Fairhall and Neal in the United States (1943).

In 1921 Brezina first drew attention to the relationship of manganese to pneumonia; he reported that 5 out of 10 men working in a pyrolusite mill had died of pneumonia in a period of two years. Baader (1933) ascribed the high incidence of pneumonia amongst workers making dry battery cells to manganese; while Elstad (1939) observed that the erection of an electrical plant for manganese smelting in Sauda in Norway was followed by a tenfold increase in the mortality rate for pneumonia in that area. A pall of smoke containing manganese oxide with particles of less than 5 μ overhung the town. Heine (1943) admitted the high incidence of pneumonia in German factories handling pyrolusite.

Riddervold and Halvorsen (1943) described Sauda as an isolated village in western Norway situated at the farthest end of a deep fjord and surrounded by high hills. Sudden changes in climate are experienced, with 2,000 mm. (78 in.) of rain a year. Before the erection of the factory in 1915 Sauda had 1,500 inhabitants. The population slowly increased to 4,500. The factory started producing in 1923, and during that year an epidemic of lobar pneumonia affected the town. Every year since, the village has had a high mortality rate from pneumonia (Table I).

TABLE I

MORTALITY (PER 100,000) FROM ALL KINDS OF PNEUMONIA IN VARIOUS LOCALITIES (RIDDERVOLD AND HALVORSEN, 1943)

Sauda, Norway 1924–37	Oslo 1924–36	Norway 1924–36	U.S.A. 1933–37	Pittsburgh 1929–31	Long Beach, U.S.A. 1929–31
326*	106	82	82	217	42

* Lobar pneumonia only.

A survey of the bacteriology of the "Sauda" pneumonias showed that of 46 cases 13 were due to pneumococcus Type 1, 4 to Type 2, and 5 to Type 3. A second attack of pneumonia was frequent. There were 2 cases of atypical pneumonia due to pneumococcus Type 3. The age distribution of pneumonia was: 0 to 15 years, 11 cases; 15 to 30 years, 11 cases; 30 to 40 years, 3 cases;

40 to 50 years, 5 cases (2 fatal); 50 to 60 years, 9 cases (2 fatal); over 60 years, 6 cases (3 fatal). In only 6 out of the 14 cases investigated were contacts found to have the same type of pneumococcus in the throat, but 46 per cent of healthy people carried pneumococcus in the throat. Immediately before the war, Buttner (1939) reported that men working in a Rhenish pyrolusite mine suffered an average pneumonia rate of 17 per thousand with an average death rate of 6.3 per thousand, compared with a pneumonia rate of 0.54 per thousand in a control group from Leipzig.

Lloyd-Davies (1946) recorded his observations on men employed in the manufacture of potassium permanganate who were exposed to the inhalation of dust of manganese dioxide and higher oxides of manganese. The manganese content of the atmospheric dust collected by the electrostatic precipitator, expressed as manganese dioxide, varied from 41 to 66 per cent; practically all the particles were below 1 μ in size, and 80 per cent were below 0.2 μ. The group consisting of between 40 and 124 men had an incidence of pneumonia which varied from 1,500 to 6,300 per 100,000 in the period 1938 to 1945, compared with an average of 73 for the same period amongst male members of Boots' Health Insurance Society. Naso-pharyngeal catarrh and attacks of bronchitis were frequent, the latter sometimes being associated with a spasmodic element. The symptoms, however, always subsided after removal from exposure. The pneumonia which occurred in the workers was not different clinically from that which may develop in any individual, and Lloyd-Davies illustrated his paper with radiographs showing consolidation in the upper lobe which resolved in a period of nine weeks. He suggested, however, that the fever and general condition of the patient responded more slowly than usual to sulphonamides. No permanent pulmonary changes were observed in the men either on clinical or on radiographic examination.

Animal experiments in which mice were exposed to manganese dust were complicated by the obvious general toxic effect of the manganese, but the histological effects on the lungs were uniform and striking. The changes were dependent on the length of exposure, and varied from slight mononuclear infiltration to intense mononuclear interstitial infiltration with many dust-laden cells, and finally to consolidation, including haemorrhagic areas, leading to complete disorganization of the lung structure. The changes were most marked around the bronchi, and the cells lining the bronchi were swollen and had undergone a hydropic change. Lloyd-Davies thinks the manganese has a direct influence on the cells of the bronchial and alveolar epithelium. The presence of dust cells, even though few, suggests that the dust is phagocytosed, but it is probable that after absorption the phagocytic cells are killed. Either before or after absorption manganese appears to have a specific action, causing intense mononuclear proliferation and infiltration, and sufficient to cause consolidation of the lung with necrosis and haemorrhage.

Beryllium

Beryllium was discovered in 1797, but in recent years it has become increasingly important because of the properties of its alloys. Beryllium copper is corrosion resistant, non-rusting, non-sparking, and non-magnetic, and has good electrical conductivity, high strength, and astonishing fatigue-resisting powers. It is used in precision instruments, altimeters, airplane pipe lines, carburettors, and telephone switchboards, and also in aircraft production. Beryllium steel has such remarkably valuable properties that it will doubtless receive much commercial attention. Beryllium is important in the production of atomic energy. The principal ore of beryllium is beryl, and from this the beryllium oxide is extracted by processing at high temperatures with concentrated acids and fusion methods which result in the production of fumes and dust. Weber and Engelhardt (1933) first showed that these dusts produced lung damage in guinea-pigs ; while Fabrioni (1935) coined the term " berylliosis " for this pathological condition. Fairhall and his colleagues (1943), however, reported a full investigation of the toxicity of beryllium compounds in animals. They succeeded in producing experimentally both pneumonitis and dermatitis with beryllium fluoride and oxyfluoride and certain hydrolysed beryllium salts such as the chloride and sulphate.

The earliest reports of poisoning in industrial workers came from Russia, Gelman (1936 and 1938) describing a condition in which shivering and fever, similar to metal fume fever, were followed in two days by extensive broncho-alveolitis. This lung condition he attributed to the action of fluorine, which was separated from beryllium oxyfluoride in the bronchioles and alveoli. These observations were followed by a report on 46 cases of poisoning by Berkovits and Izrael (1940). Inhalation of beryllium fluoride resulted in fever which subsided in a few days but was followed after a similar period by a second phase characterized by cough, scanty sputum, dyspnoea, temperature of 102° F. to 103° F. and cyanosis of face and extremities. Râles were heard in the lungs, particularly at the bases. The blood showed a fall of haemoglobin and leucocytes, lymphopenia, and a raised sedimentation rate, while radiographs revealed many small ill-defined discrete opacities, especially in the middle and lower areas. The patient usually recovered completely in from ten days to two months. The beryllium fluoride affected mainly the smaller bronchi, which have no cartilage in their walls. These showed desquamation ; their walls became infiltrated with leucocytes ; and later they became blocked with inflammatory exudate, leading to minute areas of collapse, with compensatory emphysema. Though resolution of the acute stage was usually complete, it might be followed by fibrosis.

Full accounts of this form of poisoning appeared during the war in Germany (Meyer, 1942, Wurm and Rüger, 1942), and in the United States, where van Ordstrand and others (1943) gave an account of three cases of chemical pneumonia among workers extracting beryllium oxide ; Kress and Crispell (1944) reported four cases in men working with fluorescent powders containing beryllium ; and Shilen and his colleagues (1944) reported disease of the respiratory tract in industries extracting beryllium from beryl ore. More recently, van Ordstrand and others (1945) have described 170 cases of poisoning which

have occurred among workers in three plants during the past four years. The manifestations were dermatitis, chronic skin ulcer, and inflammatory changes in the respiratory tract producing in extreme cases diffuse pneumonitis. Ninety of these patients had either upper or lower respiratory infections. Soreness of nose and throat and epistaxis were the symptoms of upper respiratory infection, and most cases cleared in from three to six weeks. Lower respiratory tract infections were characterized by non-productive cough, but with occasional blood-streaked mucoid sputum and dyspnoea. Râles were characteristically present in the early phase of inspiration, fine at first, becoming coarse. Vital capacity was reduced by as much as 30 per cent ; there was slight fever. If the patient was removed from contact, no case of pneumonitis developed after three weeks from the onset of the bronchitis. The condition can be prevented if the workers use a face mask to stop dust entering the respiratory tract. Chemical pneumonitis developed in 38 workers, 5 of whom died. Symptoms were cough with occasional blood-streaked sputum, substernal burning pain, shortness of breath, cyanosis, abnormal taste, anorexia with some weight loss, and increasing fatigue. The onset was insidious and without symptoms. The vital capacity was reduced as low as 2,000 ml., but the sedimentation rate was normal. Bronchoscopy showed a hyperaemic mucosa with some oedema. Radiographs showed diffuse haziness of both lungs, followed by soft irregular areas of infiltration with prominence of bronchial markings, and then by absorption of the soft infiltration and the appearance of discrete large or small conglomerate nodules scattered throughout both lung fields, and finally by the clearing of the lung fields after one to four months. Necropsies showed atypical pneumonitis ; striking features in lung-tissue sections were large numbers of plasma cells, relative absence of polymorphonuclear infiltration, diffuse pulmonary oedema, and haemorrhagic extravasation ; fibroblasts, with evidence of organization, were present. Beryllium present in the lung varied between 6.20 and 1.89 mg. per 10 g. of dry tissue.

Another and most interesting syndrome has been reported by Hardy and Tabershaw (1946), Gardner (1946), and Hardy (1947). It appeared among employees of a firm manufacturing fluorescent lamps. Seventeen people (14 men and 3 women) who worked in one building and handled a fluorescent powder, zinc manganese beryllium silicate, during the same period of time developed delayed pneumonitis, from which 6 of them died. The symptoms started either during the period of work or up to three years after leaving the factory with an average of twenty months' duration, and consisted of loss of appetite, loss of weight, dyspnoea on exertion, cough, and fatigue. At most times râles were audible at the bases and in the axillae, and in a few instances there was a pleural friction rub. The radiographic appearances fell into three groups. Stage 1 showed a fine granularity, uniform and diffuse and extending to the periphery. Stage 2 was characterized by a diffuse reticular pattern on the granular background, the hilar vascular shadows becoming fuzzy, indistinct, and

slightly enlarged. In stage 3 distinct nodules appeared uniformly through the lungs, giving the appearance of a snow-storm, the hilar shadows appearing large and indistinct. The appearances in this stage closely simulated sarcoidosis. Necropsy on one patient revealed focal and diffuse chronic granulomatous inflammation of all lobes of both lungs with organization and fibrosis, diffuse chronic granulomatous inflammation of hilar lymph nodes with fibrosis, right ventricular and atrial cardiac hypertrophy and dilation, subacute progressive focal and diffuse hepatic necrosis, and splenomegaly (400 g.) with fibrosis. There was apparently focal intra-alveolar granulomatous change characterized by centrally and eccentrically located giant cells of a foreign-body type, surrounded by epithelioid cells; plasma cells, lymphocytes, and mononuclear cells infiltrated the lesion throughout. There is manifestly a close clinical resemblance between this syndrome and Boeck's sarcoidosis, even though the histology is somewhat different.

Vanadium

World consumption of vanadium was more than trebled between 1936 and 1939. It is used for making special alloy steels and is an ingredient of the best grades of high-speed steels, since it exerts a degasifying and deoxidizing action. It promotes fineness of grain and causes a stable solution of carbides to form in the iron matrix. The principal advantages of vanadium lie in the fatigue resistance which it imparts to the alloy, resistance to shock impact, and retention of strength and hardness when hot; and there is therefore no satisfactory alternative metal for vanadium in the alloy steel field. It is also used as a catalyst for various chemical processes.

Dutton (1911) first described vanadium poisoning, the symptoms being dry, irritating, paroxysmal cough with severe haemoptysis, together with general toxaemic symptoms; he thought that tuberculosis was apt to supervene. The best review of vanadium poisoning, however, is that by Symanski (1939), who made a careful study of 19 cases occurring in a metallurgical works in Germany. The effects appeared to be those of an acute irritant which resulted in the development of chronic bronchitis. Balestra and Molfino (1942) reported lung damage occurring in labourers occupied in working over petroleum ash containing vanadium. Wyers (1946) described 10 cases of respiratory disease among 90 workers manufacturing vanadium pentoxide. These patients had complained of chronic bronchitis and shortness of breath, sometimes with haemoptysis, and three of them had developed pneumonia, as a result of which one died. Four cases showed radiological reticulation (Plate VII). He concluded that colds and pneumonia were more frequent than in the general population; but was not able to produce statistical evidence to support this.

Osmium

Osmium is one of the precious metals, and it is increasing in usefulness. Raymond (1874) reported a fatal case of poisoning occurring in a man employed by Vulpian in Paris. Osmium tetroxide fume had given rise to a capillary bronchitis which killed the patient. Necropsy revealed a confluent bronchopneumonia. Brunot (1933) confirmed that in animals osmium tetroxide produces this condition, and the rarity of cases of the disease is doubtless due to the extremely unpleasant nature of the fume; however, McLaughlin and

others (1946) recorded the occurrence of respiratory symptoms in workers handling osmium in a precious metals refinery.

Organic vegetable materials

Agriculture, employing somewhere about 800,000 persons, is one of the largest industries in the country. Its workers are liable to affection by dust of organic material such as that arising from hay, straw, and grain; and they develop a group of diseases labelled "asthma," "bronchitis," and "pneumonia," which frequently run a protracted course. The condition was first mentioned by Campbell (1932), has been clearly described by Fawcett (1938) in Westmorland and Cumberland, and is seen by Fuller (1947) in Devonshire. It has been called "farmer's lung." Fawcett ascribes the disease to fungi; but though he clearly establishes the disease as an entity, the mycotic aetiology is not by any means proved. There is no doubt that rotting organic vegetable material contains innumerable fungi, including aspergillus, and if these organisms are in the dust they also appear in the sputum; but this is no proof that the pneumonia is of mycotic origin. A similar condition has recently been reported from Scandinavia, where 9 cases were reported by Törnell (1946) and where the disease was called "thresher's lung." This author also reported marked improvement after treatment with potassium iodide. Hoffmann (1946) reported a similar disease occurring in the canton of Appenzell in Switzerland during the harvest of 1945. The crops of barley and oats had stood out in wet weather and were dried for only half a day before threshing. They were infected with a fungus *Chaetomium (ascomycetes).*

The patients suffer from gradually increasing dyspnoea which may get progressively worse over a period of months, cough with scanty frothy sputum, slight fever, and increased sedimentation rate. Clinical signs include patches of dullness and crepitations. In more severe cases shortness of breath is extreme, and muco-purulent sputum, haemoptysis, emaciation, and fever develop. Radiographic appearances in the early stages consist of a fine reticulation which is followed by a rather soft snowflake mottling, widely distributed throughout both lungs. The progress to the third stage is gradual, with an increase in the density of the mottling and an increase in the hilar and perihilar shadows; finally the lungs show patches of density due to coalescent areas of fibrosis, marked restriction of the diaphragm movements, and considerable areas of emphysema.

If the diagnosis is made early the condition responds readily to treatment with potassium iodide; but if it is undiagnosed it progresses and the patients become chronic invalids with fibroid lungs. Necropsy on one case showed voluminous lungs with much emphysema and thickened pleura. There were many bands of fibrous tissue traversing the lung substance.

Bagasse

Bagasse is sugar cane after the sugar has been extracted. It is used for making boarding for interior decoration and for thermal insulation. The sugar cane which is used for

this purpose is grown in Louisiana and shipped to this country in bales. Amorphous silica forms roughly 1 to 2 per cent of the bagasse, and quartz 0.1 per cent, many of the quartz particles being 20 to 30 μ in size. It is most improbable, therefore, that there could be any risk of silicosis. Duncan has examined samples of bagasse and found many fungi present. He estimated that 1 g. of air-borne dust contained 240 million fungal spores.

W. E. Lloyd in 1940 first noticed an unusual kind of pneumonia in workers with bagasse, and since then 14 cases with one death have been described in the United States (Jamison and Hopkins, 1941 ; Jamison and others, 1944 ; Sodeman and Pullen, 1943 and 1944), and one death from Mexico (Manas, 1945). Reference to the disease in England has been made by Castleden and Hamilton-Paterson (1943), while a full account of the incident has been recorded by Hunter and Perry (1946). The disease arose in England in workers on the machine which broke up the bales of bagasse. This machine was housed in a separate building, and there did not appear to be any risk in the main factory buildings. Before 1940 the bales were broken in a soak pit by a wet process causing no dust, but this became unsatisfactory when the bales were packed more tightly to save shipping space. In 1940, 14 men were employed on this bagasse " shredder," as the machine is known, for continuous periods of more than three days ; in 1941, 20 men ; and in 1942, 10 men. Of the 14 men employed in 1940, 13 were traced ; of the 20 employed in 1941, 18 were traced ; and of the 10 employed in 1942, 8 were traced. Out of the 13 men traced who worked the shredder in 1940, 7 suffered from a characteristic acute respiratory illness, and of the 8 men employed during the first three months of 1941, 3 showed signs of this illness. Thus, out of 21 men employed on the shredder in a period of 15 months, 10 (47.5 per cent) developed the illness. Symptoms usually began after the men had been working on the machine for eight weeks. The disease manifested itself as an acute febrile illness with extreme shortness of breath, cough with scanty, black, stringy sputum, and occasional haemoptysis. Signs were scattered throughout both lungs, and radiographs showed miliary shadows throughout the lung fields. The appearances were, therefore, those of an acute bronchiolitis. The symptoms gradually improved over a period of six weeks, at the end of which time radiographs showed the lung fields to be clear. One patient in 1941 died after 25 days' illness, but unfortunately no necropsy was performed. These cases give strong evidence that a specific disease, manifesting itself by acute bronchiolitis, collapse, and pneumonia, has occurred in the lungs of men handling bagasse.

In 1943 all the workers in the factory were interviewed and occupational histories taken. It was found that out of 163 men employed 22 had worked in the coal mines, and of these 19 showed radiographic evidence of either reticulation, nodulation, or massive shadows, a complicating factor not considered in certain reports on the disease. In 1944 further radiographs were made, and these

revealed a man who had worked on the shredder for a year and who showed the radiographic changes in his chest already described. He had a cough and was short of breath. It seems probable that the bronchioles become filled with vegetable dust which swells under the influence of the bronchial secretions, blocks the bronchioles, and gives rise to small areas of collapse. When these areas become infected, typical acute bronchiolitis and pneumonia result. In two men the condition did not resolve, and they developed a fibrosis of lung with cough and sputum and great shortness of breath, together with radiographic changes, which showed thick bands of fibrous tissue transversing the lung field so as to simulate cavities. One of these men died, and at necropsy he was found to have chronic bronchiolitis and bronchiectasis of similar distribution to that associated with dust diseases. No large cavities were found. Fungi may play an important rôle in breaking down the fibre into a very fine vegetable dust, and may possibly even render this toxic. Aspergillus had been present in his sputum, but this is not evidence that it was the cause of the disease. It is present in all specimens of bagasse dust, and therefore can be grown from the sputum of all men exposed to this dust. But the post-mortem material from the lung of the fatal case showed no evidence of aspergillus, and histological examination showed nothing to suggest a disease of fungal origin. The appearances were certainly not those of silicosis. This disease resembled farmer's lung, "broken wind" of horses, and in some ways byssinosis. These diseases may belong to a single group, so that when the pathology is fully elucidated it may be found common to all four. The radiographic changes in the lungs in bagassosis are illustrated in Plates VIII *a, b,* and *c.*

Cotton

In America, Neal and others (1942) described an acute febrile illness with cough, dyspnoea, tightness of the chest, and more general symptoms, occurring among workers making mattresses from low-grade cotton, in cotton mills, and in cotton-seed processing plants where workers were exposed to high concentrations of stained cotton dust. They thought the condition was caused by a Gram-negative rod-shaped bacillus or its products in or on the dust from the stained cotton. This may be so, but cotton is certainly a vegetable dust, and the aetiology of this condition may possibly be similar to the above diseases. First Collis (1914) and then Middleton (1926) recorded that cotton weavers in Lancashire suffered from an acute bronchitis from handling cotton fibre. This was an acute illness with fever, cough and sputum, and residual symptoms which might last up to two months. The most probable cause of the outbreaks was thought to be the amount of moisture left in the warp, producing an abundant growth of fungi, which developed during storage on the beams and produced, on weaving, a dust laden with conidia, spores, and fragments of mycelium which was breathed by the weavers.

Asthmatic Conditions

Nearly every dust has been reported at some time or another as giving rise to asthma, but there seems nothing to be gained by referring to all the individual examples of this condition that have been described in the literature. There are, however, certain well-defined allergic diseases resulting from exposure to occupational dusts.

Platinum

Platinum refiners recently complained that there was a high incidence of asthma among their workers. Karasek and Karasek (1911) reported that they found among workers in forty photographic studios in Chicago eight cases of poisoning characterized by irritation of the throat and nasal passages, causing violent sneezing and coughing. There was also bronchial irritation causing respiratory difficulty so great that some were unable to use paper containing potassium chloro-platinate. Workers in all the platinum refineries in England have been examined (Hunter and others, 1945). Clinical and occupational histories, blood examinations, and radiographs of their chests have been taken. There are 114 ; 20 under 30 years of age, 30 between 30 and 40 years, 39 between 40 and 50, 22 between 50 and 60, and 3 over 60. Eighteen had worked in the refineries for more than 25 years, 24 for more than 20 years, and 25 for more than 10 years. Ninety-one of these workers came in contact with the complex salts of platinum during the processes, and of these 52 suffered from a syndrome similar to that described by the Karaseks. It started with repeated sneezing, and was followed by profuse running of the nose with watery discharge. Then followed tightness of the chest, shortness of breath, and wheezing. These symptoms persisted as long as the men were in the factory and for about an hour after they left. The symptoms would then subside, but the men would wake in the early hours of the morning with a bout of coughing which might last half an hour. A polycythaemia was observed in one man during an attack (red blood cells 6,350,000 per c.mm. of blood, haemoglobin 130 per cent).

An analysis of the occupation of the workers who complained of this asthma, as well as the circumstances in which it occurred, produced conclusive evidence that the complex salts of platinum are the cause of this syndrome. The sieving of spongy platinum produced a much higher concentration of platinum in the atmosphere (400 to 900 μg. per cu.m.) than the process involving the complex salts. Workers were observed sitting over this process without exhaust ventilation and without wearing masks, and yet in no instance was asthma found to arise among them. In five instances workers who complained of asthma were moved from the platinum refining department to the department refining rhodium and ruthenium without recurrence of symptoms. The incidence was highest in those in contact with the complex salts in dry form, but the disease did occur if droplets were present in the atmosphere. The concentrations giving rise to the symptoms varied from 5 to 70 μg. per cu.m.

Byssinosis

Jackson as early as 1818 drew attention to the fact that those who worked in the cotton industry, particularly card-room workers, suffered from a characteristic respiratory disease. In 1862 Greenhow described the condition in a report to the Privy Council, and in 1932 the Home Office issued the *Report of the Departmental Committee on Dust in Card Rooms in the Cotton Industry.* This report collected the existing information about conditions in the mills, types of machinery in use, and the nature of the cotton dust. It included an account of the clinical examination and radiological findings in a group of workers at Ashton-under-Lyne who suffered from the disease, and described the necropsy findings in a few cases. The disease was named byssinosis from the Greek word βυσσος, meaning fine linen. Foreign literature on the condition is scanty, but Zipperlen (1935) investigated patients suffering from asthma in the textile industry of Würtemberg, and recorded that there were many with severe attacks in whom strong allergic reactions could be obtained with suitably prepared dust from the factories; while Bolen (1943) reported two cases in the United States. In England Prausnitz (1936) reported to the Medical Research Council on respiratory dust disease in operatives in the cotton industry ; and Gill (1947) described the process of carding and the symptomatology of the disease.

Cotton is obtained chiefly from Egypt, America, and India, and arrives at the mill in tightly compressed bales, which also contain much foreign material such as particles of leaf and seed coat, cotton hairs, fragments of mould, and fine sand. The raw cotton is therefore first cleaned, and the tightly compressed cotton is pulled off in layers and hand-fed into a machine called the "hopper bale opener." It then passes into the blowing-room where a series of machines, by means of beating actions and currents of compressed air, frees it from most of the impurities. All these machines are enclosed in dust-proof covers ; the machines, however, have to be cleaned, and this is a particularly dusty job for which the workers have to be suitably clothed and wear respirators. The clean cotton coming out of the blowing-room passes to the carding engines, and it is principally the strippers and grinders in charge of these engines who suffer from the disease. Usually each stripper and grinder is in charge of sixteen carding engines ; each engine is cleaned four times daily by a vacuum process, and twice weekly it is brush-stripped. The latter process is necessary since the vacuum-cleaning fails to clean sufficiently deeply. The evidence is very strong that the dust liberated by the carding process in the immediate vicinity of the carding engine contains the injurious dust which causes byssinosis. The atmosphere is progressively safer the greater the distance from the carding engines.

The usual history is that after working for several years in the dusty atmosphere the man begins to sneeze and develops a dry and irritating cough with a tight feeling in the chest and restricted intake of breath. At first the trouble is temporary, passing off in one or two days ; but it returns after a short absence from work such as a week-end. It has, in consequence, been called "Monday morning fever," "mill fever," and "factory fever." Disablement and incapacity for work does not result at this stage, and the breathing will become normal on removal from the dusty atmosphere. After exposure to the dust for ten or more

years, however, the symptoms become more severe and persistent, and the worker suffers from asthma and bronchitis associated with shallow breathing, cough, and a small amount of sticky mucoid phlegm. He loses weight and develops a sallow complexion. Absence from work and incapacity increase, though removal from the dust causes improvement, with capacity to work in another atmosphere. In the last stage emphysema is severe. Cough is present, with mucoid or mucopurulent expectoration and great shortness of breath. In extreme cases the man may stand with his hands pressed against his thighs to aid his breathing. The disease is incurable at this stage and many of these workers die before the age of 50.

The physical signs are those of chronic bronchitis and emphysema, the respiration is laboured, with the accessory muscles called more and more into use. The thorax becomes typically barrel-shaped, and the patient tends to sit in a characteristic attitude with the body bent slightly forward, the legs widely spaced, and the hands placed on the knees. The vital capacity is greatly reduced, and the chest expansion may be less than one inch. Radiographic appearances are consistent with chronic bronchitis and emphysema, though the shadows radiating from the hilum are of greater intensity than those usually found in these conditions and suggest considerable fibrosis.

Necropsies have revealed chronic bronchitis and emphysema but do not show any specific feature which enables the bronchitis of card-room operatives to be distinguished from that which occurs in the general population. Dunn and Sheehan (1932) have reported that dilatation of the right heart was frequent.

Claims for compensation are made under " The Byssinosis (Benefit) Scheme, 1941," which places assessment in the hands of a medical board appointed by the Secretary of State. The Scheme decrees that the man must have been exposed to cotton dust for a minimum period of twenty years. Women do no stripping or grinding, and though they work in carding rooms and are liable to respiratory troubles they cannot claim benefit under the Act.

No special medicinal treatment is of any value, and timely removal from the work is the only successful method of preventing the disease from progressing. Respirators have been tried but are not satisfactory since the men find them difficult to work in and therefore do not wear them. Methods of suppression of dust by exhaust ventilation and enclosing the machinery have been highly developed in the industry and the disease is thus kept to a minimum. It is still, however, one of the most important industrial diseases at the present time.

Wheat dust

Large amounts of vegetable dust in a fine state of division may produce asthma and bronchitis, and Duke (1935) reported asthma occurring in men exposed to the dusts arising from the first cleaning of the wheat grain in the flour mills of Kansas City. The minute hairs of the grain were found responsible, and the injection of infinitesimal amounts of wheat hair extract gave rise to severe local reactions and to asthma in sensitive millers.

Neoplastic Conditions

Agricola and Paracelsus in the sixteenth century described at length disease of the lungs with a high mortality in the miners of the Erzgebirge in Saxony. The condition was known as " Bergkrankheit " (mountain disease). It was not until 1879 that it was identified by Haerting and Hesse as cancer of the lung. These authors attributed 75 per cent of all deaths in miners to the disease, which usually developed after a period of 20 years' work in the mines. Arnstein (1913) distinguished the tumours as carcinoma, and differentiated them from lympho-sarcoma and endothelioma, as most neoplasms of lung at this time were thought to be. Since people in the district other than miners were only rarely affected by this disease, all the early workers attributed the condition to the occupation of mining. The main ores in Schneeberg are sulphides and arsenides of nickel and cobalt—" Speiskobalt " ($CoAs_2$) " Wassnickelkies " (Ni_2As_2) and " Rotnickel-kies " (NiAs)—while at Joachimstal on the southern side of Erzgebirge there is a high content of pitchblende and radium. Rostoski, Saupe, and Schmorl (1926) showed that the bore dust of these mines contained up to 0.45 per cent of arsenic, while the dust in the pits contained 0.1 per cent ; but it has not been found possible to prove that arsenic is either the cause or a contributory agent. In Southern Sweden, where Speiskobalt is also mined, pulmonary cancer is said to be unknown, and in Schneeberg no other signs of arsenic poisoning are reported. Hueck (1939) has pointed out that multiplicity of tumours which is so frequent in arsenical cancer does not occur in Schneeberg miners, while estimations of arsenic in the urine, hair, and nails (Rostoski and others, 1926), and in the lungs (Pirchan and Sikl, 1932 ; Zeil, 1935), give equivocal results. Work in the Joachimstal area by Loewy (1929) and Sikl (1930) has suggested that the radio-activity of the ores is the principal aetiological agent. Peller (1939) showed from necropsies that 50 per cent of the miners in this area died with lung cancers, while Lange (1935) gave similar figures for Schneeberg. Recently Lorenz (1944) expressed the opinion that the radio-activity could not be the sole cause of the carcinomata, since similar doses of *x*- or *y*-rays do not produce lung tumours in animals. He suggested that both arsenic and radio-activity might be contributory factors.

Amor (1938) reported ten cases of carcinoma of the lung and numerous cases of carcinoma of the nose and nasopharynx among workers who had been exposed to arsenical dust for an average period of six years at a nickel works in South Wales. The ores were from Sudbury in Canada and contained 2 per cent of arsenic, and for a period of 25 years had had the copper extracted by the addition of sulphuric acid which had not been freed from arsenic ; the subsequent grinding and calcination of the arsenical residue resulted in the escape of much dust from the apparatus then in use. Recently Thompson (1946) has been able successfully to perform pneumonectomy for carcinoma of the lung on two of these workers ; both were squamous cell carcinomata (Plates IX and X). The arsenic contents of the lungs

were 3.82 and 3.40 parts per million (p.p.m.), the content of the hair of the men was 2.2 and 10.5 p.p.m., and they were excreting 41.5 and 39.8 μg. per 24 hours.

Other evidence suggesting that arsenic produces carcinoma of lung is the experimental work of Leitch (1922), who has succeeded in producing such growths in mice with potassium arsenite. But the most conclusive evidence is the statistical work of Bradford Hill (1947), which has shown that in one town in England where the principal industrial work is the manufacture of arsenical sheep-dip the mortality rate for carcinoma in the chemical workers handling arsenic is double that in the rest of the population of the town, a statistically significant difference. Bradford Hill showed that the difference is made up almost completely by carcinomata of the skin and bronchi.

Pneumonokoniosis

It is only since 1930 that radiography of the chest has been used on a wide scale. It is not surprising, therefore, that much new knowledge about the effect of dust on the lungs is beginning to accumulate. The characteristic radiographic change attributed to pneumonokoniosis is diffuse shadowing, which if soft and ill-defined is called reticulation, and if hard is called nodulation. These shadows may become either fluffy and confluent or else conglomerate, when a massive shadow results. The appearance varies widely with the degree of penetration of the *x*-ray; a soft film may show reticulation, a hard film a normal chest, while on an intermediate film opinion may vary substantially. There is no visible difference between the nodulation caused by deposits of iron in the electric arc welder's chest and that appearing in the classical silicotic or even in the person suffering from chronic miliary tuberculosis, yet the pathological lesions are fundamentally different.

It cannot be too strongly urged that these radiographic appearances are only shadows: more than that must not be read into them. The assessment of their significance is difficult, and since pneumonokoniosis is an industrial disease the problem is always complicated by the question of compensation under the Workmen's Compensation Act. This compensation is paid for the disability acquired by the workman through the inhalation of dust while at work. It has always been recognized that the diagnosis of the disease and the assessment of disability are difficult. For this reason the highly complicated Silicosis Acts were passed by Parliament and the Silicosis and Asbestosis Boards were created. The assessment of disability, however, remains difficult and at present is largely decided by radiological appearances. After their survey of chronic pulmonary disease in South Wales coalminers, Hart and Aslett (1942) suggested that the inclusion of reticulation as a compensatable disease would not lead to any large increase in the certification rate because only a small proportion of these cases were significantly disabled; but the enactment of the Pneumonokoniosis in Coalminers Bill, 1943, has been followed by a very large increase in the number of certifications. This is because, in fact, if a man has been exposed to a dangerous dust and has radiographic shadows it is impossible with existing methods to assess minor degrees of disability, and in English law, quite rightly, the workman must be given the benefit of any doubt.

Assessment of disability

Attempts have been made to arrive at some assessment of disability by methods other than plain radiography. In the clinical examination of pneumonokoniotics the two most valuable observations are certainly the marked diminution of air entry on auscultation and the poor chest measurement on inspiration and expiration, and it is hardly likely that more information than this about functional disability can be obtained from radiographs. However, McCann and Kaltreider (1937) have suggested that if the ratio of the areas of the lung fields at maximal expiration and at maximal inspiration exceeds 71 per cent, measured with a planimeter on doubly exposed radiographs, there will certainly be disability. The most important laboratory methods are reaction to standard exercise, measurement of oxygen content of blood, blood sedimentation rates, and estimation of lung volume, including vital capacity and residual air. The fact that no two workers employ exactly the same exercise tolerance test is in itself a testimony to the unsatisfactory nature of these tests, and it is doubtful if the more complicated exercises such as those involving the use of dumb-bells or pedalling bicycles give much more information than simple stepping up and down on a box. In any case little information is likely to be gained unless the disease is advanced and the disability obvious. Measurement of the oxygen content of the arterial blood is complicated by the fact that the technique of arterial puncture is difficult, and the range of complete and incomplete saturation of the blood with oxygen is narrow; nevertheless, this is a most promising field of research, and if it is possible to develop an oximeter which can estimate the oxygen saturation of the blood from the lobe of the ear, the solution of the problem might be in sight. Little work has been done on blood sedimentation rates, and nothing is known of normal levels throughout the day in the industrial population as a whole. Most patients, however, who have real disability from pneumonokoniosis show an increased sedimentation rate; this may in part be due to infection either tuberculous or non-tuberculous, but it is possible that the onset of the disability is closely related to the complication of infection. It is, therefore, possible that the blood sedimentation rate will prove to be a valuable simple test.

Vital capacity is at the present time the best guide, and if tracings are taken in the way suggested by Christie (1934) a permanent record is obtained. Vital capacity tracings are of little value for comparing one individual with another, but a series of tracings which show a decreasing vital capacity in a given individual over a period of time are of great significance. By taking the weight and height or stem height of the individual it is also possible to compare his actual vital capacity with the expected one for an individual of his surface area. The greatest difficulty with these tracings is the variable co-operation of the patient; some develop a sense of suffocation, and with men seeking compensation the operator can never be sure that they have emptied or filled their lungs to the limit. This can to a certain extent be checked by measurement of the residual air, and McMichael's modification of the Christie method is comparatively simple, as is that described by Cournand and others (1941). McCann and Kaltreider (1937) have shown that a decrease in the total capacity and the vital capacity of the lungs with an increase in residual air is usual in advanced pneumonokoniosis, and they consider that when the ratio of residual air to total capacity exceeds 40 per cent some degree of functional impairment is usually apparent. These authors also attempted to combine this method of investigation with a response to exercise, and thought that a better method of estimating disability is that of measuring the total ventilation during five minutes of exercise at a rate of 300 kg.-m. per min. and 3 min. of subsequent rest. The resulting ratio of total ventilation to vital capacity is of great functional significance. They found that in normal healthy men the ratio varied from 20 to 48, and that when the value rose above 55 shortness of breath was experienced. Estimations of the pulmonary reserve at definite rates of work also give an index of functional ability. Normal men have a pulmonary reserve of 55 to 73 per cent when working at 300 kg.-m. per min. when they are using 27 to

45 per cent of their maximal ventilating capacity, and dyspnoea is experienced when 50 to 60 per cent of the maximal ventilating capacity is used.

It would be better if compensation were not paid for partial disability but if the man were removed from the dangerous dust and trained to fill a job which would give him equal or better prospects in life.

Siderosis

Metals and their salts throw *x*-ray shadows the densities of which vary with their atomic weight. Thus Arrigoni (1933), Pendergrass (1938), and Preti and Talini (1938) have shown that the inhalation of the dust of barytes and barium (atomic weight 137.4) produces the densest of nodular shadows in the lung. Iron (atomic weight 57) also produces radiographic changes, a fact first described by Zenker (1866). Much work has recently been done on this subject.

Doig and McLaughlin (1936) described the radiographic change which they observed in a group of six electric arc welders. These men showed well-developed reticulation and in some cases nodulation, and yet suffered very little in the way of symptoms. Harding (1945) produced similar changes in rats by intratracheal injection of iron oxide in the form of rouge. Autopsies on these animals, and by Enzer and Sander (1938) on an embalmed human being exhumed eighteen months after death, showed that the iron oxide was in the perivascular and peribronchial lymphatics and that it did not produce fibrosis in the lung. It is remarkable that there is no more satisfactory necropsy reported in the literature, and it is to be hoped that at the first opportunity this deficiency will be remedied. Since this time the clinical picture has been observed by many more workers, including Britten and Walsh (1940), Harrold and others (1940), Sander (1944), and Groh (1944), while Enzer and others (1945) described a group of 15 cases with full physiological tests, showing that they suffered no disability. Pendergrass and Leopold (1945) recorded 4 cases of siderosis occurring among 50 grinders of bearings made of chrome vanadium and chrome molybdenum tool steel containing 98 per cent iron, about 2 per cent alloy, and not more than 0.2 per cent silica. Artificial abrasion wheels composed of bakelite, silicon carbide (carborundum), and aluminium oxide had been used exclusively in the works for the previous seventeen years. The four workers who showed radiological changes had been employed for twelve, thirteen, fourteen, and seventeen years respectively. The dust to which they were exposed had a concentration of 4.9 million particles per cubic foot of air, of which 96.5 per cent were less than 5 μ in size and 99.5 per cent less than 10 μ. The average percentage of silica, as quartz, in the dust was 0.43. These men had no symptoms apart from cough. Buckell and her colleagues (1946) described the survey of 171 individuals exposed to iron dust in iron turneries. Radiographs of the chest were taken in all, and reticulation was found in 15 persons; 5 of these had worked in the turnery for more than twenty years. The changes are related to length of exposure and not to age,

no reticulation being seen in 15 men over sixty years of age. Symptoms were few; only 1 complained of shortness of breath, though 6 had cough. The dust obtained from rafter samples contained 37.1 per cent metallic iron and 23.3 per cent iron oxide, while the total silica content was 5.4 per cent and the silicate residue 1.3 per cent.

No reference will be made to the incidence of lung disease in foundry workers since, though iron figures prominently in the dust of the workshops, the problem is complicated by the presence of large quantities of silica. Likewise, though the radiographic changes in the lungs of haematite iron ore miners are highly characteristic, these workers are exposed to a mixture of iron and quartz, and the result is a sidero-silicosis with all the symptoms and disability caused by the more severe disease.

Plate XI shows the radiographic appearance of nodulation in the lungs of an electric arc welder.

Argyro-siderosis

McLaughlin and others (1945) recorded their clinical and radiological findings in four silver finishers who had been polishing silver-plated articles with rouge for periods between twenty and forty years. The radiographs of the lungs of these workers showed the typical picture of reticulation (Plate XII *a* and *b*). All of the men had cough, and one brought up copious rouge-coloured sputum. One died from bronchopneumonia after an operation for gastric ulcer, and a necropsy was performed. The lungs were emphysematous and of uniform grey-black cut surface, but on microscopical examination there were no fibrotic changes, either collagenous or reticular, which could be attributed to the dust (Barrie and Harding, 1947). Of great interest was the intra-vitam staining of the elastic tissue in the arterial and alveolar walls from the inhalation of the particles of silver over a period of forty years (Plate XIII *a* and *b*); and, though the great part of the radiographic reticulation was due to the iron oxide, a small part was doubtless acccunted for by the silver. Chemical examination of the lung showed that ash constituted 10.17 per cent of the dry weight, and of this ash 72 per cent was iron (reckoned as Fe_2O_3), and 6 per cent was silver, as metal.

Aluminium

Aluminium dust has been used extensively in making explosives, incendiary mixtures, and paints, but there is conflicting evidence about its effect on the lung. There is evidence from Canada that the inhalation of aluminium dust will prevent silicosis, though the subsequent "patenting" of the method seems a little unfortunate. Denny and others (1939) exposed eight rabbits to metallic aluminium dust of particle size below 5 μ, and showed that no fibrosis was produced, but Belt and King (1943) repeated these experiments in rats and showed that the rats' lungs treated the particles as foreign bodies, and formed small concretions with fibrous tissue around.

Repeated papers by Goralewski (1939, 1940, 1941, and 1943) from Germany gave details of various types of disease attributed to aluminium dust, and pressed for them to be scheduled under the Workmen's Compensation Act in that country. Aluminium powder can be made by two processes; either it can be blown, when the particles will be covered with a thin film of alumina, or it can be stamped, when they are covered with a thin film of stearine. Seven hundred workers were examined; they were making aluminium powder by a stamping process, but paraffin-like substances were used owing to the shortage of stearine in Germany. The aluminium powder, which was 98 per cent metallic aluminium, contained about four milliards of particles per gram of dust, and many of the factories were blown up because an explosive concentration of aluminium dust was reached. The workers complained of dry cough with pain on breathing, shortness of breath, poor appetite, and gnawing abdominal pain. Spontaneous pneumothorax was described in four workers. Blood counts showed a relative lymphocytosis with an eosinophilia up to 10 per cent. The sedimentation rate was within normal limits in 78 per cent of the cases, and the vital capacity was decreased in 27 out of 125 men examined. Radiographs showed focal shadows in the apical region with an increase of normal bronchial markings in the upper and middle thirds of the lung, giving a reticular appearance which at a later stage tended to increase and become confluent (Plates XIV, XV *a, b, c,* XVI, and XVII). The development of the illness was rapid, often within three months of starting work. It appeared to bear no relation to the length of exposure to aluminium. Histological examination of the lung of a patient who died revealed coarse, branching, hyaline, collagenous fibres which enclosed phagocytes containing fine and coarsely granular particles distinguished by their jagged outline from carbon particles. A similar picture was produced in animal experiments by Jötten and Eickhoff (1942). Jäger and Jäger (1941) suggested that while aluminium powder is highly resistant to aerial oxidation it is freely soluble in sodium chloride solution, giving sodium aluminate and aluminium chloride in equilibrium. A colloidal aluminium hydroxide complex results if the sodium and chloride ions are allowed to diffuse away, and if protein is also present it is co-precipitated round the partly dissolved aluminium particles. Jäger and Jäger think that this complex caused the lung changes. Koelsch (1942), however, believed that mechanical factors were responsible, and that the disease was a consequence of the unsatisfactory ventilation of workrooms resulting from the black-out. This year a similar condition has been reported from Canada by Shaver and Riddell.

While there is no doubt about the entity described by these workers, evidence of a different kind has come from America and England. Crombie and others (1944) investigated 125 workers employed in the Pittsburgh stamp mills of the Aluminium Company of America. The workers here had been exposed to aluminium dust for periods ranging from 6 to 23 years, and their health was found to be as good as that of the 3,000 other workers in the plant. Radiographs

of the chests of all the men taken each year for three years showed no abnormalities that could be attributed to the inhalation of dust. They then treated 34 silicotic miners by the daily inhalation of fine aluminium powder freshly ground from small aluminium pellets in a specially constructed mill. Daily treatment began with five-minute inhalations, which were gradually increased to 30 minutes. Some of the men received 300 treatments, but the majority only 200. Out of 34 cases thus treated, clinical improvement in 19 was manifested by lessening or disappearance of shortness of breath, cough, pain in the chest, and fatigue. In 15 cases the condition became stationary, and remained so in spite of continuous employment in silica dust throughout the treatment. The progress of the disease was assessed by means of tests of respiratory function, repeated at three-monthly intervals. Crombie and his colleagues therefore concluded that the inhalation of finely particulate aluminium powder is not harmful to human lungs.

Hunter and others (1944) endeavoured to throw light on this problem by studying the effect of dust on the lungs of grinders of duralumin aeroplane propellers and of workers in a factory making aluminium powder by a blowing process, but no evidence has been found so far to show that the aluminium dust has any harmful effect on these workers' lungs. The grinders were exposed to a mixture of aluminium dust arising from the propellers and alumina from the alundum grinding wheels. The average concentration of aluminium dust close to the operator's mouth was found to be 3 mg. per cubic metre of air, of which the particle size in 1 mg. was between 2 μ and 7 μ, the remaining 2 mg. consisting of larger particles. The alumina particles were mostly of less than 1 μ in diameter. The health of these workers, as shown by the number of sicknesses during the preceding year, was better than that of those in a machine shop belonging to the same company. None gave any history of spontaneous pneumothorax, and no abnormality was found in their blood counts. Radiographs were taken of the chests of 92 workers: seven of these showed shadows in the peripheral parts of the lung which were different from those usually found in this situation. These shadows are probably of little significance, but attention is drawn to them since it is conceivable that they are shadows caused by concretions of aluminium dust as described by Belt and King in their rats. Nevertheless the men worked for long periods in the dust without any obvious effect on their health. Sixty-two of the workers were under, and 30 over, 40 years of age: 28 had been on the job for more than seven years, a further 9 for more than five years, and 31 for more than two years.

The German work cannot, however, be disregarded, and it must be assumed that metallic aluminium in sufficient concentration and of sufficiently small particle size will produce an acute and rapidly progressive disease in the lungs. This may possibly be related to the paraffin substances used to coat the German aluminium powder.

Graphite

No systematic survey of graphite workers has been carried out, but Dunner (1945) argued from the finding of various degrees of fibrosis in the radiographs of five workers who had been unloading graphite from ships for five years that this material could produce a pneumonokoniosis. In contrast to the marked radiographic abnormalities, there were but few physical signs on auscultation, and while 3 of the men were " chesty " (cough, sputum and shortness of breath) 2 had no complaints, and all were fit for work. Later Dunner and Bagnall (1946) reported a case of " necrosis cavitation " in the lung of a graphite worker. The sputum contained graphite though the patient had not worked for five years. It is not easy to understand what conclusions the authors wish to draw from their discussion of these cases. It is known that the graphite to which they were exposed was not pure but contained as much as 30 per cent silica. Graphite, which is found in Ceylon and Madagascar, is a crystalline modification of the element carbon, and may be looked upon as intermediate between well-crystallized diamond on the one hand and amorphous carbon black or ash-free anthracite on the other. Animal experiments with diamond, anthracite and other forms of coal, carried out by Gardner (1938), did not cause any fibrous reaction in the lung. It can therefore be accepted that pure graphite will not produce fibrosis of the lung.

Silicates

Most silicates will produce dust; and if workers are exposed to these dusts they are likely to show radiographic changes in the lungs, and in certain instances they will develop serious diseases. The most important of these silicates are china-clay ($H_4Al_2Si_4O_9$), talc ($H_2Mg_3Si_4O_{12}$), mica, sillimanite (Al_2SiO_5), and asbestos, the last-named producing by far the most serious illness.

China-clay, or kaolin.—This is hydrated silicate of aluminium, and at present is one of the most important exports from the United Kingdom. It is an important ingredient in china and earthenware pottery, and is used in the manufacture of a large number of other commodities, including cotton cloth, paint, soap, and fine art paper. 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. Men are exposed to the dust in shovelling the clay after it has been dried in the kilns. No medical survey has been carried out in the Cornish china-clay industry, and therefore little is known of the effect of this dust in the lungs. Middleton (1936), however, recorded the case of a worker aged 49 who showed the whole of both lung fields covered with extremely fine mottling, most marked in the middle zones and at the right base. He considered the film suggested pneumonokoniosis not of a silicotic type. Hale (1946) has some radiographs of workers in the

industry showing well-marked reticulation. It is probable that these workers have few symptoms and that the dust does not produce great disability.

Talc, or French chalk.—This is hydrated magnesium silicate, and in mineral form is known as steatite or soapstone. It is used as a filler for paper, soaps, and paints, for leather dressing, in the manufacture of rubber motor tyres, in making electric switchboards, and for heat insulation of steam pipes.

The first account of changes in the lungs due to talc was given by Thorel (1896), followed by the report by Devoto and Cesabiachi (1911) of the necropsy of a woman who had worked in talc. The lung showed much talc dust but little fibrosis resulting. Zanelli (1931) recorded the case of a woman worker in a tyre factory, aged 27, who had early tuberculosis but also reticulation in the lung radiograph. Dreessen (1933) investigated the workers in a tremolite talc mine and mill, and found that those who had been employed for more than five years had radiographs which showed fine diffuse bilateral fibrosis, but this appeared to cause little disability. With Dalla Valle (1935), he carried out a further survey in Georgia and found that 16 out of 33 mill workers exposed to high concentrations of talc and 6 of 13 miners had pneumonokoniosis. In 4 cases there was evidence of added pulmonary infection. Merewether (1933, 1934) examined 24 workers who were exposed to talc in the manufacture of rubber tyres for periods of between four and a half months and forty years. They showed no disablement though there were radiographic changes suggesting diffuse interstitial fibrosis of the lungs. Siegal and others (1943) investigated tremolite talc millers and miners in St. Lawrence County, New York. The talc ore, which was of the fibrous variety known as asbestine, was mixed with tremolite and anthophylite, and showed a free silica content of 1 per cent or less. Microscopically, the dust was found to contain fine, straight, needle-like fibres, and dust counts ranged from 6 million to 5,000 million particles per cubic foot. This material was obtained by underground mining with inclined shafts to depths as low as 1,000 feet; each mine employed 15 to 17 men underground; 221 miners and millers were examined and radiographic evidence of fibrosis was found in 32, giving an incidence of 14.5 per cent. All the cases occurred in men who had worked for ten years or longer, and the incidence in this group was 25.9 per cent. The appearances were of fibrosis of a fine, diffuse type, showing granulation or nodulation on a hazy background. The resulting dyspnoea, cough, and fatigue were disabling, and many of the men showed clubbing of the fingers. There was some increased susceptibility to tuberculosis, three men showing evidence of the disease. Besides the fibrosis, deposits of unidentified opaque material in the periphery of the lung called "talc plaques" were observed in 6.3 per cent of the workers. These plaques varied from single linear deposits in the region of the diaphragm to massive deposits, bizarre in shape, extending over a large part of the lung fields; they were irregular in shape, density, and the sharpness of their outlines. No deaths were recorded,

but the clinical and radiographic picture showed some resemblance to asbestosis rather than silicosis.

Plates XVIII *a* and *b* show the radiographic changes in the lungs of two talc-workers.

Sillimanite.—Sillimanite is an aluminium silicate, and is a very dense rock found in India. It is used as a refractory material in the manufacture of porcelain. When broken it produces masses of prismatic crystals in the form of fibres which cannot be broken down into fine fibres like asbestos but tend to break and become reduced to powder.

When handled in industry the rock is crushed, ground, and sieved, and then calcined, during which a considerable quantity of dust is given off. Middleton (1936) reported the investigation of 15 men who were employed handling this material. Their ages ranged from 24 to 50 years and the duration of their employment in the industry was from one and a half to sixteen years. Chest radiographs showed abnormality in only 4 men, who had been working with the material sixteen, eleven, seven, and five years, and in whom some reticulation was noted. These numbers are small, but suggest that the dust does not produce any great damage in the lungs. Jötten and Eickhoff (1944) studied the effect of exposing 25 rabbits, for periods ranging from six months to two years, daily for a period of two hours, the dust particles being less than 5 μ in diameter. Necropsies were carried out on 22 of these animals. Five died from collapse or pneumonia after being dusted for periods up to a hundred days, and showed minimal fibrosis; 14 survived for periods up to a year, and in this group there was more fibrosis with thickening of the interlobular septa. Three of the animals survived 673 days; these showed well-defined dust nodules in the interlobular septa and in the perivascular and peribronchial regions, consisting of dust particles with fibroblast and early connective tissue formation. These animal experiments suggest, therefore, that the dust may not be quite as harmless as the clinical observations of Middleton suggest.

Mica.—Mica belongs to a group of widely distributed rock-forming minerals, of which muscovite and phlogopite are important commercially. Muscovite is a silicate of aluminium with potassium, and phlogopite is magnesium-aluminium silicate. The former is used for its transparency and resistance to fire for windows of stoves and lanterns, while it is sometimes ground up and used in the manufacture of paper, wall-paper, and paint, and as a lubricant and absorbent. The latter is used exclusively for electrical purposes. Dust is produced when the materials are being ground and finished.

Middleton (1936) reports that Ferguson examined 12 workers who were exposed to mica dust, but only 5 of them had been exposed for more than five years and these complained of cough and shortness of breath. Radiographs showed some fibrosis of the peribronchial type with some diffuse shadows in the middle zone. Ferguson thought, therefore, that the dust of mica was probably capable of causing pulmonary fibrosis.

Asbestos.—The term "asbestos" is of Greek origin and means "unconsumable." Asbestos was worked into textiles by the Egyptians, Romans, and the inhabitants of the ancient empire of Tartary, the wealthiest among them using it as cremation cloth. Pliny called it "a rare and costly linen," whilst Plutarch recalls its use as perpetual wick in the lamps of the Vestal Virgins. The use of asbestos seems to have been lost in the Middle Ages, and an attempt to revive it in the eighteenth century met with little success, so that it was not until the middle of the nineteenth century that it again became industrially important. Mineralogically the industry is concerned with two main types of asbestos. (1) Hornblende, or amphibole, to which group blue crocidolite, readily identified by its lavender-blue colour, and amosite, white or yellowish-brown in colour, belong, is produced exclusively in South Africa. (2) Chrysotile, white in colour and of fine texture, is produced mainly in Canada, Rhodesia, Transvaal, Australia, Italy, and Russia. Their composition is given in Table II.

TABLE II

	Hornblende		Chrysotile	
	Blue crocidolite	Amosite	Canadian	Rhodesian
	%	%	%	%
Silica	50	47	41	41
Alumina	1	6	3	1
Ferric oxide ..	35	37	2	4
Magnesia	2	6	40	39
Alkalines	8	1	—	—
Water	4	3	14	15

It is imported as the crushed rock, but the fibres can be separated to an extreme degree of fineness which renders them capable of being carded, spun into yarn, and woven into cloth by processes similar to those used in the cotton industry. Besides being woven, asbestos is ground and mixed with cement and plastics to make insulating slabs, boarding, and many other articles used in industry. The dust given off during the process of manufacture is mostly less than 5 μ in diameter and 10 μ in length.

The first case of asbestosis was described by Murray (1907). The man had been employed on a carding machine for fourteen years, and was the last survivor of 10 men who had worked in the card-room, the others having all died at about the age of 30 with respiratory diseases. He died in Charing Cross Hospital in 1900 at the age of 34. Necropsy revealed extensive diffuse pulmonary fibrosis and the lungs were found to contain asbestos bodies. In 1906, Marchand and Reisal (Kruger and others, 1931) noted the presence of unusual bodies in the lungs of an asbestos worker; while Auribault (1906) in France recorded the high mortality in an asbestos textile factory. Fahr (1914) described a case of asbestosis in Hamburg. Real attention, however, was drawn to the disease by Cooke (1924), who recorded the death of an asbestos worker with diffuse pulmonary fibrosis and pulmonary tuberculosis; while McDonald (1927) and Cooke and Hill (1927) gave the first detailed descriptions of asbestos bodies and asserted that they originated in the inhaled dust. Seiler (1928, 1931) recorded the necropsy on an asbestos worker who showed fibrosis but no evidence of tubercle.

The condition was fully described by Merewether and Price (1930) and Merewether (1933, 1934). Clinically, the striking symptom is dyspnoea of gradually increasing intensity, often associated with a dry cough; in the early stages physical signs are slight or absent, but later râles throughout the lung develop and the fingers often become clubbed. Radiologically the most characteristic feature is the "shaggy" outline of the heart shadow, while in the rest of the lung field there is a fine fibrosis superimposed on lung fields which present a "ground glass" appearance. The reticulation and nodulation which are seen in silicosis do not occur in this disease.

At necropsy, pleural adhesions are extensive and dense, and there are often thick subpleural plaques of fibrous tissue. The consistency of the whole lung is increased, and there may be large areas of fibrous condensation which is tough rather than stony hard as in silicosis. Other parts of the lung not affected by the fibrotic change are congested or consolidated with inflammatory processes. Emphysema is extensive, but localized often to the lower and apical parts of the lung; pneumothorax sometimes occurs. The histology has been described by Gloyne (1932), Gardner (1933), and Beger (1933). There is generalized fibrosis with diffuse thickening of the alveolar walls, while throughout the lung tissue are seen fibres of asbestos, either singly or in clumps, as well as the characteristic asbestos bodies. The presence of these bodies in the sputum is described by Stewart and Haddow (1929), Simson and Strachan (1931), and Page (1935), and is a characteristic of the condition. Gloyne (1931) showed that they could also be found in the faeces.

Wood and Gloyne (1934) collected 100 cases of the disease and found that 21 were complicated by pulmonary tuberculosis, but Middleton (1936) studied 54 deaths occurring between 1930 and 1934 and found that tuberculosis was present in 24 (44 per cent). The periods of employment in the industry ranged from ten months in the disintegrating plant to twenty-three years' weaving, the average duration being just under ten years. He suggested that tuberculosis was a slightly less frequent complication than in silicosis and that it is more frequent as a terminal complication than as a concomitant disease during the earlier stages.

Wyers (1947) showed that, as a result of measures enacted by the legislation of 1931, the morbidity rate from asbestosis fell from 39.1 per 1,000 in 1932 to zero in 1940, but that there was a small rise during the war years. Concomitant with the fall in morbidity rate there were significant alterations in the character of the disease to a more chronic form. Clinically, this was manifested by an increase in long survival periods, clubbing of the fingers (48.2 per cent of cases) and sometimes of the toes, and perhaps by the high cancer rate in those who have suffered from the disease for several years (15.3 per cent pulmonary cancers and 20.4 per cent all cancers). Attention has also been drawn to this aspect of the disease by Linzbach and Wedler (1941). Radiologically, the fine, diffuse, cobweb-like film formerly characteristic has now

largely given place to a coarser, more granular appearance. (Plate XIX *a* and *b*.) Pathologically, confluence of the blue polygonal areas is not so advanced as that seen in the specimens of fifteen years ago. Wyers thought these changes were due to longer exposures to lower concentrations of dust. The post-mortem appearances of the lungs from 3 of Wyers' cases are illustrated in Plates XX, XXI, and XXII.

From his clinical studies of asbestosis Wyers concluded that it differed from silicosis in being a "clinical" rather than a "radiological" disease. Of the clinical signs he stressed the importance of clubbing of the fingers and symptoms of cardiovascular origin (raised diastolic blood pressure and accentuated second pulmonary sound) as being always present when once established; whereas the marginal crepitations and fibrotic crackles tend to be evanescent. Of the complications, emphysema was always present, bronchopneumonia the commonest termination, tuberculosis was of the acute, caseous type and occurred in one-third of the cases, right heart dilatation was common, and bronchiectasis rare. Pregnancy could not be shown to affect the progress of the disease adversely, and the obstetrician should be guided by the cardiovascular rather than the pulmonary signs.

The difference between the reaction of the tissues to silica and to asbestos is even more marked than the difference between the clinical pictures of silicosis and asbestosis and has been demonstrated by animal experiment. The "solubility" theory is at present generally accepted as explaining the mechanism of silicosis, but Gloyne (1930) and Policard (1933) have shown that the injection of asbestos dust does not cause an acute inflammatory reaction. Gardner and Cummings (1931) found that asbestos accumulated in the bronchioles and then in the alveoli, and that fibrosis first appeared after about 500 days' exposure. Gardner (1942) put forward the "mechanical" theory as an explanation of the disease. It was the long fibre in the dust which caused asbestosis, and if the asbestos were ground sufficiently finely no disease resulted. He concluded that the disease results from the mechanical irritation produced by a combination of the long fibre and the rhythmic movement of the lung. He also showed that different pathogenic responses resulted in different animals. King and others (1946), however, produced somewhat different results from the injection of asbestos dust intratracheally into rabbits; they reported that the injection of long fibres (15 μ) produced a nodular reticulinosis comparable with the experimental silicotic nodule, whereas short fibres (2.5 μ) produced a diffuse interstitial reticulinosis.

Conclusion

In this discussion it has been shown that non-siliceous occupational dust may give rise to distinct clinical diseases such as byssinosis and asbestosis, and distinct radiological appearances such as those produced by siderosis, and also that it may predispose to ordinary lung diseases such as pneumonia and

carcinoma. This latter effect may perhaps be more important, but its possible importance has only recently been realized, and the means at present available make the proof difficult. In the case of manganese dust a works medical officer studied the sick absences of a group of workers and noticed that there was a high incidence of pneumonia amongst them. He was able to compare this incidence with the large number of other workers under his care, and to carry out animal experiments, and by these means to prove the connexion between the high incidence of pneumonia and exposure to manganese dust. In the case of carcinoma resulting from arsenic, a statistician was able to survey the death register of a comparatively isolated small town with but one industry. Since circumstances resulted in little change in work and residence, a statistically significant result was found without undue difficulty. In the case of bagasse a disease was suspected and the small group of people affected by it were found to have been in ten different hospitals. The sarcoid-like syndrome resulting from exposure to beryllium was found to arise up to seventeen months after leaving the work which caused it. These examples of the methods and difficulties involved in the study of diseases caused by occupational dust serve to indicate how little is known of these conditions and how much work remains to be done on the subject. Certainly the conclusion that all dusts are potentially dangerous cannot be resisted. It also seems probable that the provision of adequate exhaust ventilation in factories in the next century will be a measure of public health comparable to the provision of adequate sanitation in the last.

SUMMARY

The literature of the diseases of the lung caused by occupational dust other than silica is reviewed.

Exposure to metallic dusts such as manganese, beryllium, vanadium, and osmium causes chemical pneumonitis.

A sarcoid-like syndrome may develop late after exposure to beryllium.

Mouldy organic material, such as hay, corn, bagasse, and cotton, gives rise to acute bronchiolitis, but there is no conclusive evidence to support the theory that this results from aspergillus or other fungi.

Complex salts of platinum give rise to a form of asthma.

Byssinosis affects a very large number of workers in the cotton-spinning area of Lancashire.

The incidence of carcinoma of lung is abnormally high in those exposed to arsenic dust and to asbestos.

Metals according to their atomic weight are opaque to *x* rays, and dusts such as iron oxide and barium give rise to radiographic shadows ; the changes causing these shadows have been called siderosis and baritosis.

Silver is deposited in the elastic layer of the arteries of the lungs of workers exposed to its dust.

The silicates, particularly china-clay, talc, mica, sillimanite, and asbestos, give rise to various kinds of fibrosis in the lung, though not to a nodular fibrosis like quartz silica.

Graphite appears to be harmless.

The methods of assessment of disability in the group of occupational dust diseases which might be called "radiological" are briefly discussed; these include siderosis, those due to mixed dusts of silicates, and the early stages of silicosis.

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