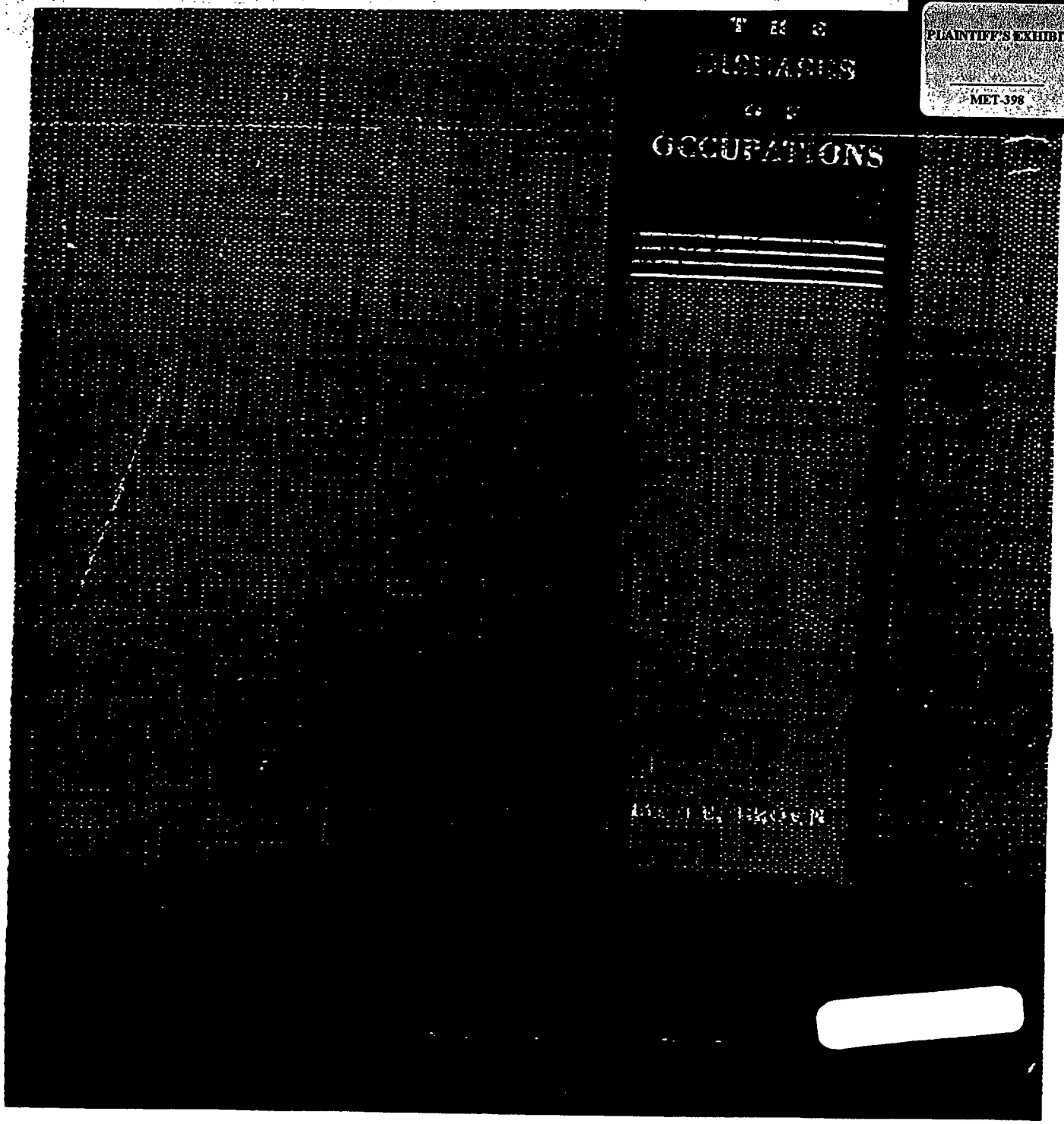

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MET-398

THE DISEASES OF OCCUPATIONS

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1955

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DONALD HUNTER.

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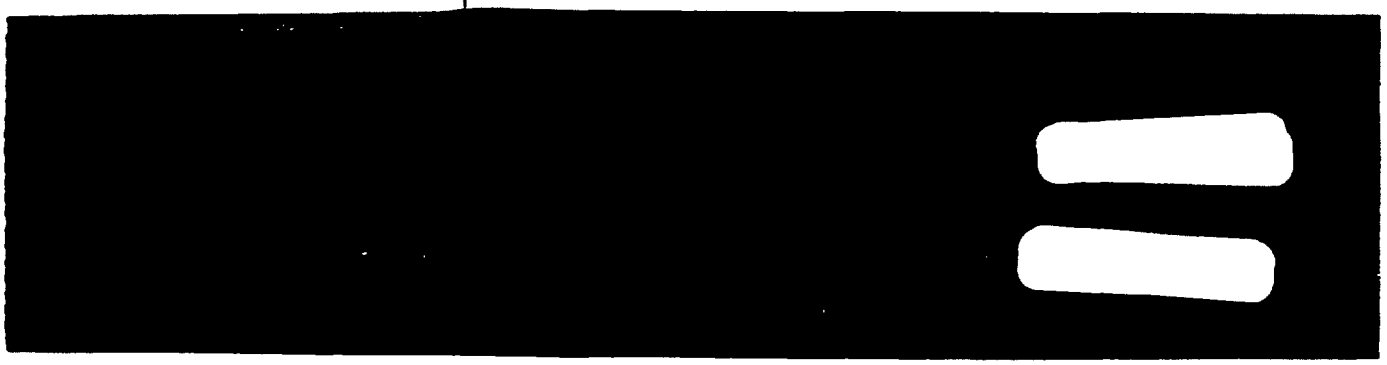

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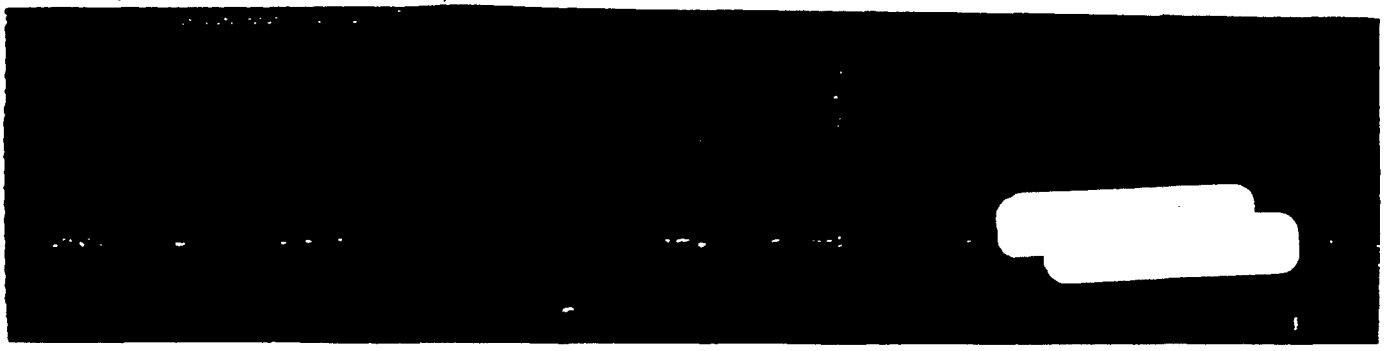

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CHAPTER XIV

THE PNEUMOCONIOSES

The pneumonokonioses (Greek, πνεύμων lung; κόνις dust) or, more shortly, pneumoconioses constitute a group of lung diseases which result from the inhalation of dust in certain occupations.

SILICOSIS

Silicosis is the most important disease of this group and is defined as a pathological condition of the lungs due to the inhalation of particulate matter containing free or uncombined silica, silicon dioxide, SiO_2. It is important to distinguish between silica in the free state as SiO_2 and in the combined state as the various silicates.

Silicosis and Pneumoconiosis not Synonymous

Silicosis and pneumoconiosis must not be used as synonymous terms. Logically one could classify the pneumoconioses as pneumoconiosis due to silica, pneumoconiosis due to asbestos, pneumoconiosis of coal miners and so on, but such a method would be too cumbersome. Legal considerations make it important that the term silicosis be reserved for that condition caused by the inhalation of uncombined silica, the special characters of which render it capable of being identified when it occurs in typical form. Dusts other than uncombined silica produce pneumoconioses, but these do not present the characters which are possessed by silicosis in its typical form.

Great Prevalence of Silicosis

Silicosis is widespread throughout the world. It is prevalent in many industries, and of all the pneumoconioses it claims the largest number of victims, either alone or with tuberculosis with which it is frequently allied. Silicosis has been known as dust consumption, ganister disease, grinders' asthma, grinders' consumption, grinders' rot, grit consumption, masons' disease, miners' asthma, miners' phthisis, potters' rot, rock tuberculosis, stone-hewers' phthisis and stonemasons' disease. The origin of these terms is obvious. Recognition of silicosis depends in a given case on a knowledge of the occupational history, on the clinical and the radiological examination, and in fatal cases on the necropsy findings.

Prehistoric Flint Implements

How early in the history of mankind silicosis appeared is a subject for interesting conjecture. It probably existed in the palæolithic period, for

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prehistoric man manufactured flint implements extensively. In 1915 Collis pointed out that the flint-knappers of the village of Brandon in Suffolk suffered a high mortality from phthisis induced by flint dust generated in their work (see p. 845). Probability suggests, therefore, that the starting-point of human progress was associated with at least one form of silicosis. Hippocrates in his *Epidemics* speaks of the metal digger as a man who breathes with difficulty, and Pliny mentions the use of respirators to avoid dust inhalation.

A Substance like Sand in the Lungs

We have seen (p. 23) how in the sixteenth century Joachimsthal in Bohemia was the greatest mining area in Central Europe. It was here that Agricola (1556) strongly advocated the ventilation of mines, for he knew that dust entering the lungs caused disease associated with dyspnœa, and he spoke of corrosive dusts which ulcerated the lungs, producing consumption. He relates that in the Carpathian mountains there were women who had married seven husbands, all of whom were brought to an early grave by the dust disease of the mines. But the first account of the pathology of what we now call silicosis came from Holland. In 1672 in his *Anatome corporis humani* Isbrand van Diemerbroeck (1609–74), professor of medicine in Utrecht, described how several stone cutters died of asthma, and he found at necropsy that to cut their lungs was like cutting a mass of sand. Ramazzini (1713) describes how stone cutters breathe in small splinters and turn asthmatic and consumptive.

Starting-point of Modern Studies

The starting-point of the study of silicosis in modern times was a paper by Johnstone in 1796, calling attention to the high mortality among needle pointers at Redditch. Arnold Knight was apparently the first to write of silicosis in Sheffield. In 1819 he propounded the view that criminals should be employed for the process of dry grinding. This proposal was immediately denounced. The period just after this date seems to have been the time when grinders' asthma became notorious, about the time that the water power of Sheffield, which originally had made the industry possible, was changed to steam power. Immediately there was a great increase in the amount and severity of silicosis. We have seen (p. 113) how Thackrah (1831) knew of Knight's work describing the deadly effects of the dry grinding of forks in Sheffield. But he knew much more than this, for he made it his business to survey many industries. We have seen (p. 112) that he knew of the danger from sandstone dust in mining and of the harmlessness of limestone dust. He had noted that bricklayers and limeworkers were long-lived and that sandstone masons usually died before they reached the age of forty. In men employed in grinding he recognized the association of dust inhalation and tuberculosis. He had noticed, too, that pulmonary disease was prevalent amongst men filing iron castings to remove burnt-on sand.

Fate of the Sheffield Cutlery Grinders

In 1843, Calvert Holland described the conditions of work amongst Sheffield grinders. He showed how grinding was always performed on a dry stone, in a room in which there were usually about ten people at work, and the dust rose in clouds pervading the atmosphere in which they were confined (fig. 360). He discovered on examination that among ninety-seven men, about thirty were suffering in varying degree from the grinders' asthma. He obtained records of sixty-one persons who had died between 1825 and 1840. Thirty-five of these were under thirty years of age.



FIG. 360.—Table-blade grinding in Sheffield (1886). The underground Workshop was known as a Hull or Stye. It was estimated at the time that the Conditions of Work robbed the Men of 25 years of Life
(By courtesy of The Illustrated London News)

In *The Vital Statistics of Sheffield* (1843) G. Calvert Holland, who was physician to the Sheffield General Infirmary, wrote:

> The analysis of the trade of fork grinding will present a vivid but painful picture of the condition of an extensive class of artisans in this town. It is perhaps more destructive to human life than any pursuit in the united empire. Wet grinding is generally confined to saws, scythes and edge-tools; the dry to an extensive class of small articles, such as razors, scissors, pen and pocket knives,. forks and needles. Fork-grinding is always performed on a dry stone, and in this consists the peculiarly destructive character of the branch. In the room in which it is carried on there are generally from eight to ten individuals at work,

and the dust which is created, composed of the fine particles of stone and metal, rises in clouds and pervades the atmosphere to which they are confined.

The dust, which is thus every moment inhaled, gradually undermines the vigour of the constitution, and produces permanent disease of the lungs, accompanied by difficulty of breathing, cough, and a wasting of the animal frame, often at the early age of twenty-five. Such is the destructive tendency of the occupation, that grinders in other departments frequently refuse to work in the same room, and many sick clubs have an especial rule against the admission of dry grinders generally, as they would draw largely on the funds from frequent and long-continued sickness. In 1,000 deaths of persons above 20 years of age, the proportion between 20 and 29 years, in England and Wales, is annually 160, in Sheffield, 184; but among the fork-grinders, the proportion is the appalling number 475; so that between these two periods, three in this trade die to one in the kingdom generally.

Between the ages of 30 and 39, a still greater disparity presents itself. In the kingdom, 136 only in the 1,000 die annually between these two periods. In Sheffield, 164; but in the fork-grinding branch, 410; so that between 20 and 40 years of age, in this trade, 885 perish out of the 1,000; while in the kingdom at large, only 296. Another step in the analysis, and we perceive that between 40 and 49, in the kingdom, 126 die; in this town, 155; and in this branch, 115, which completes the 1,000. They are all killed off. For in carrying forward the inquiry we observe that between 50 and 59, in the kingdom, 127 die; and in Sheffield, 155; but among the fork-grinders, there is not a single individual left. After this period of life, there are remaining in the kingdom, of the 1,000, 441; and in the town, 339; but none in this branch of manufacture.

Grinders' asthma in its advanced stages admits neither of cure nor of any material alleviation. In the early stages the only efficient remedy is the withdrawal from the influence of the exciting cause; but how is this to be effected by men who depend from day to day upon their labour, and whose industry from early life has been confined to one particular branch? Here then, is the melancholy truth that nearly one-third of this class of artisans, in addition to the poverty and wretchedness common to the whole, is in a state of actual disease—and disease which no art can cure. Fiction can add no colour or touches to a picture like this. Truth transcends the gaudy embellishments of imagination. The distempered fancy has here no room to exercise her powers.

Further evidence of the wretched state of the fork-grinders—and the remark applies with great truth to grinders generally—is the low state of education among them. Of the 197 men and boys, 109 can read only, and 69 can write. Thus, in a Christian country—a country that expends vast wealth in attempting to educate and enlighten the

dark heathen—one-half of an important body of human beings, near this source of benevolence and comprehensive charity, actually cannot read, and about two-thirds cannot write! The inability to work, and yet the necessity to labour, creates a degree of wretchedness and suffering easier to imagine than describe. But the wretchedness is not confined to the individual. A wife and increasing family are involved in the accumulated evils. Poverty, yoked with disease, embitters and shortens life in a thousand forms, but all forms of misery. We do not hesitate to assert that this is a picture of wretchedness, which has no parallel in the annals of any country, or in the records of any trade.

The Work of Greenhow and Peacock

Two other great Englishmen figure in the history of the pneumoconioses —namely, T. B. Peacock and E. H. Greenhow. Peacock, a physician on the staff of St. Thomas's Hospital, first established between 1860 and 1866 the existence of miners' disease as an entity and distinguished it clinically from pulmonary tuberculosis. Greenhow (1860, 1861), who was attached to the Middlesex Hospital, carried out the first large field investigation into the dusty industries such as the potteries, metal trades, cutlery making, tin and copper mining, coal mining, lead mining, cotton, flax, silk and woollen manufacture, hosiery and lace making, glove making and agriculture. In the *Transactions of the Pathological Society of London* (1860–66) are to be

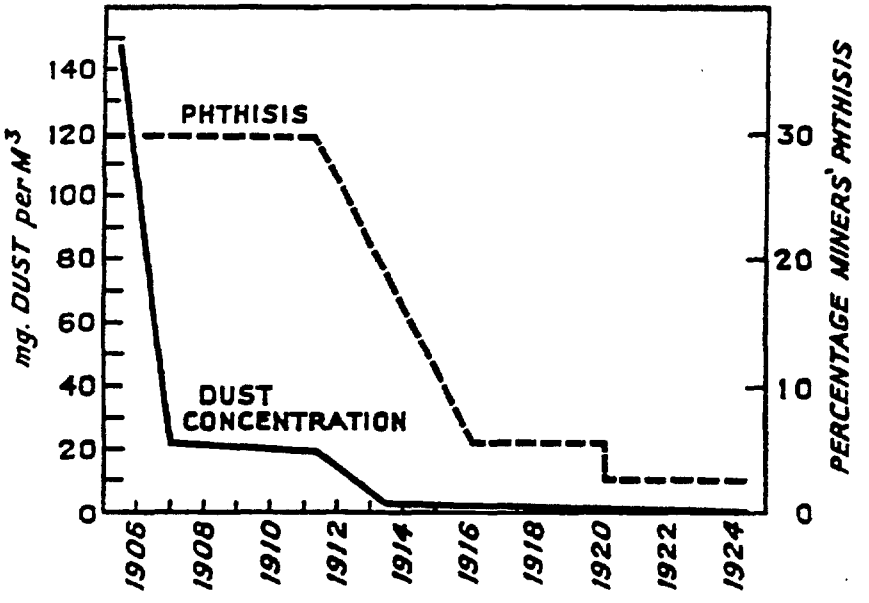

FIG. 361.—Conditions of work in the Gold Mines of the Witwatersrand. The Incidence of Miners' Phthisis began to fall steeply about seven years after the Dust Concentation was significantly reduced
(*After Mavrogordato*)

found excellent clinical and pathological descriptions by both these doctors of the disease which was later to be called silicosis by Visconti in 1870. They even found the dust of free silica in the lungs and examined it under polarized light.

World-wide Interest in Silicosis

In 1885 Arnold published a monograph on dust inhalation, pointing out that dust particles may be found in the liver, spleen and bone marrow, as well as in the lungs. The work of J. S. Haldane in tin miners (1904), Hay in granite workers (1909) and Wheatley (1911) in sandstone-quarry workers, and the report of a Commission on Miners' Phthisis in South Africa (1912) opened the twentieth century (fig. 361), the early part of which has been

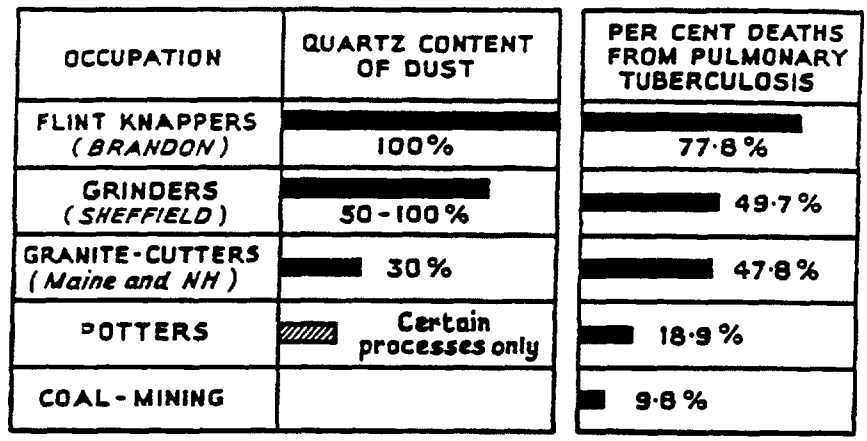

FIG. 362.—Relationship of Deaths from Tuberculosis to Quartz Content of Dust
(After Collis)

notable for a gradually extending and now world-wide interest in the subject (fig. 362).

Mechanization increases the Dust Hazard

By this time mechanization of industry had come to stay, and with increased speed of production there was, of course, more dust. On this subject McLaughlin (1953) wrote:

> With the turn of the century a new interest began to be taken in the problem, not only in this country, but also in other parts of the world. It is significant that about the same time there was a quickening of the tempo of life in general. The horse began to give way to the motor car, and soon there came the first aeroplane. Then, too, there was the telegraph and the telephone and all those "comforts" of present-day life which give us little or no time to think. In the factories

and in the mines there was a speeding-up of output. Hand labour began to be replaced by the machine. In the cotton industry the change had come a little earlier. But the air-hammer or the pneumatic tool began to be used more extensively in the early part of the century for such jobs as mining, quarrying and the cleaning of castings in foundries. There is also the spray gun, which is used to spray paint, glazes, metals and asbestos, and was introduced with the idea of speeding up the work. This, from a health point of view, is a dangerous instrument, because it is very difficult to control the spray and to prevent its inhalation by the workers.

If there is one thing certain about increased speed of production in industry, it is that a dusty process will become more dusty; and that there will be a greater incidence of the dust diseases, if attention is not given at the same time to increased control of dust. But quite often the efforts at dust control are inadequate and do not keep pace with the increased speed of production.

Mechanization of Mines and Foundries

Silicosis and silico-tuberculosis did not become a problem in hæmatite mining until the introduction of the pneumatic drill. Stewart and Faulds (1934) say that hæmatite mining has been carried on in Cumberland since the time of the Roman occupation.

> It was formerly considered a healthy trade, but evidence is accumulating to show that in this respect a definite change for the worse has taken place. The miners themselves believe that the trouble started with the introduction of the dry mechanical drill in 1913.

Previously the ore had been obtained by the old hammer and jumper method. Mechanical cutting and conveying of coal was introduced into the coal mines of Great Britain early in the present century. The change from hand-cutting of coal led to an increased dustiness of the air in the mines, not only at the coal face but generally throughout the workings. In 1950 H.M. Chief Inspector of Mines wrote:

> There is no doubt that one result of the adoption of many of the present methods of machine mining is that the production of dust in mines has increased in recent years and is still increasing. If we are to get rid of the scourge of pneumoconiosis, this process must be reversed (Bryan, 1950).

McCallum (1952) also thinks that mechanized coal-getting has increased the prevalence of pneumoconiosis in the Durham coalfield, and that future developments in the coalfield will intensify dust production and the risks of pneumoconiosis unless dust-suppression methods are considerably extended. The use of the pneumatic tools in place of hand methods for the fettling of steel castings has also increased the risk of silicosis in this occupation (McLaughlin and others, 1950).

Symptoms and Signs

Silicosis is generally divided into first, second and third stages, or slight, moderate and severe degrees. South Africa and Ontario speak of ante-primary, primary and secondary stages, terms of importance in connexion with their compensation laws. The first stage, the so-called simple silicosis, supervenes in a workman who has been employed in an industrial process involving exposure to siliceous dusts for a period of many years. The changes can occur from a few months after exposure to over sixty years. Commonly they are found half-way between these extremes. The onset of symptoms is marked by dyspnœa on exertion, slight at first, and later increasing in severity. Throughout the illness dyspnœa remains the most important symptom. Slight cough may be present from the first. It is usually unproductive or with scanty sputum. The general condition of the patient is unimpaired. Physical signs in the chest are slight. Diminished expansion is scarcely, if at all, present. Dullness can rarely be demonstrated, and in older subjects there may be areas of hyper-resonance due to emphysema. There is no alteration of the breath sounds, and there are no added sounds. In this stage, impairment of working capacity may be slight or absent. In the second stage, dyspnœa and cough become established and further physical signs appear. There is diminished expansion of the chest, patchy dullness, sometimes with bronchial breath sounds, and scattered rhonchi, especially at the bases. There is always some degree of impairment of working capacity. In the third stage, dyspnœa leads to total incapacity. Right heart hypertrophy and then failure may supervene.

Radiographic Appearances

In the first stage of silicosis the radiograph shows the presence of discrete nodular shadows, circular, and at the most 2 millimetres in diameter. They may be partially distributed throughout the films, more widespread, or even generalized, but they remain discrete (plate X). Sometimes they are, in part, obscured by emphysema (plate XI). In the second stage the whole of both lung fields are occupied by nodular shadows, and there is some coalescence to form more or less dense opacities. In the third stage the radiographs indicate areas of massive consolidation. It is sometimes necessary to differentiate the X-ray findings from those of chronic miliary tuberculosis, sarcoidosis, berylliosis (plate I), miliary carcinomatosis, siderosis, and the hæmosiderosis of chronic heart failure (plate XIV).

Pulmonary Tuberculosis Supervenes

Pulmonary tuberculosis may be present in any stage of silicosis. It may alter the symptoms, physical signs, radiological appearances and the whole course of the disease (plate XII). It is the most frequent accompaniment of silicosis. Since tuberculosis of the lungs may simulate silicosis in radiographs, no diagnosis should ever be made exclusively on radiographic appearances.

Morbid Anatomy

At necropsy the lungs are generally large and retain their shape on removal from the thorax. Pleural adhesions are nearly always present and they may be extensive at the bases. On parts of the lung not covered by thickened pleura, the surface is studded over with pale grey nodules, each of which is felt to protrude above the general surface and to be part of a nodule which extends into the lung substance.

Silicotic Nodules

The cut surface of the lung shows excess of pigmentation throughout, but the striking and distinctive feature is the presence of numerous round



FIG. 363.—Large Tissue Section of Normal Lung, natural size

nodules (fig. 363). These are dense, tough and black or grey in colour (fig. 364). Each nodule is from 2 to 5 millimetres in diameter, but several may be aggregated together to form large composite nodules, or many may be united in a massive fibrosis. In long-standing cases, individual nodules may be thrown into sharp relief by emphysema of the surrounding lung. The centres of the nodules may undergo calcareous change. In cases where exposure to dust has been intense and the course of the disease relatively rapid, the nodules may be so crowded together that practically no normal lung tissue can be seen. This condition is likely to occur in occupations, such as sand-blasting, where there has been exposure to dust containing a very high proportion of free silica. Histologically, the silicotic nodule is a mass of concentrically laminated dense fibrous tissue.

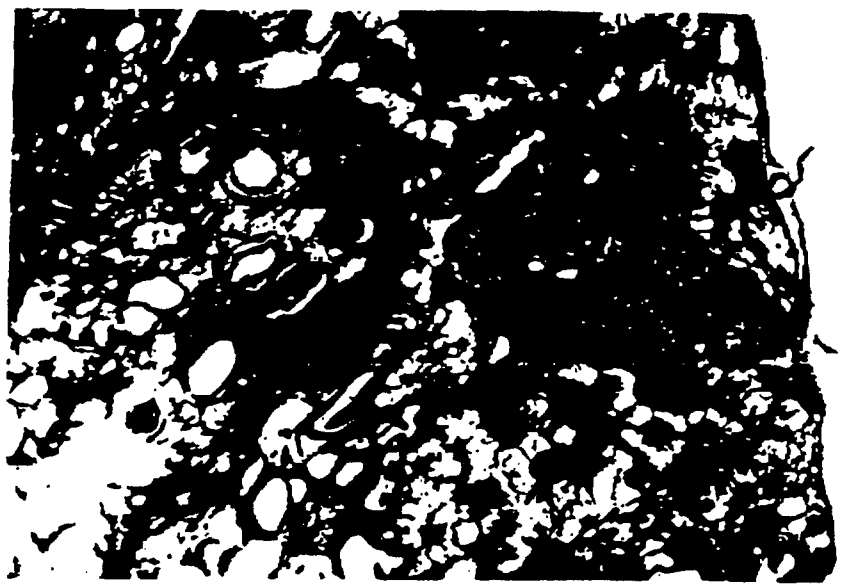

FIG. 364.—Large Tissue Section of Lung, natural size, showing Silicotic Nodules, both Individual and Confluent

Massive Fibrosis

Discrete masses of fibrous tissue, rounded or oval in shape, may be found centrally or peripherally situated in the lung, usually about the middle zones. The masses are nearly always bilateral and may be multiple, and frequently more extensive in one lung. In these cases the remaining parts of the lungs usually show discrete nodules. It is possible that the massive fibrosis is inflammatory in origin. Certainly the interlobar fissures are frequently obliterated by adhesions, and bands of fibrous tissue may extend from the periphery into the substance of the lung.

The Cuirass Form

There is a second form of distribution met with following exposure to very high concentrations of free silica dust. It is known as the cuirass form. In it there is a layer of fibrosis extending from the pleura to a distance of a centimetre or more into the lung substance, and frequently over its whole extent, and with nodules of denser fibrosis embedded in it.

Tuberculosis as a Complication

The presence of tuberculosis may render the diagnosis of silicosis at necropsy very difficult. Either the tuberculosis may be completely obscured by the silicotic process, or the silicosis may be obscured by the changes produced by tuberculosis, which may have left no unaltered silicotic tissue. In the first instance, the presence of tuberculosis may be determined only after the finding of tubercle bacilli in the lung or a positive

result to animal inoculation. In the second instance, great difficulty may be found in establishing a diagnosis of silicosis.

Changes in Lymphatic Glands

Changes in the lymphatic glands are important in the diagnosis of silicosis. The tracheo-bronchial groups are usually affected early in the course of the disease. At first enlarged, they are found in more advanced cases of the disease, hard and, on section, dark-grey or black, often showing several concentrically arranged systems of nodular fibrosis. Parts of the cut surface may show white points or bands of fibrous tissue, or the whole of the gland structure may be replaced by fibrous tissue. Some glands in a group may be more affected than others, and several stages in the replacement of the glandular tissue by nodules may be seen. The lymphatic glands may show foci of tuberculosis, while tuberculous lesions are not apparent in extensively silicotic lungs.

Absence of Predisposition to Cancer

Considerable difference of opinion exists on the question whether the presence of silicosis has any influence in predisposing the affected lungs to develop cancer. The two diseases are certainly found together. From a statistical study Kennaway found no occupation involving exposure to any kind of dust in which there was any very high incidence of cancer of the lung or larynx. It must be concluded that on the evidence at present available there is no justification for any statement that the presence of silicosis predisposes to cancer in the affected lungs.

Properties of Dusts and Fumes

In order to understand the dust diseases it is necessary to know a good deal about the chemical and physical properties of dusts and fumes, the behaviour of dust clouds and the reaction between them and the tissues of the respiratory tract. The nature of gases and vapours has already been discussed (see p. 557). Unlike gases and vapours, both dusts and fumes are composed of particles which, when airborne, can be inhaled into the lungs. Fumes consist of material in such fine particulate form as to resemble gases but which condense to solids at room temperature. Dusts, on the other hand, consist of larger particulate matter suspended in air. It must be realized that these definitions are not rigid and that they merely indicate somewhat roughly different stages of attenuation of matter.

Particle Size of Dusts and Fumes

While dusts have been classified as particles or aggregates of particles from 150 microns to one micron in diameter, fumes from one micron to 0·2 micron in size, and smokes as particles less than 0·3 micron in diameter, size alone represents at best only a rough separation of these three classes. The mode of formation must also be considered. Thus dusts ordinarily result from vigorous mechanized attrition, blasting, grinding, drilling,

rubbing, crushing, hammering and sawing. In general they have the same chemical composition as the substances from which they are derived; they have not been altered chemically by the process of subdivision. Fumes and smokes are formed and carried into the air usually as the result of chemical reaction, or some physio-chemical change, or the sudden dispersion of a chemically active substance by release of pressure or by explosion.

Fumes and Smokes

In industry, fumes are usually the oxides formed from hot or boiling metals. Thus lead fume produced in the smelting of lead is lead oxide, and zinc fume from molten brass is zinc oxide, the particle size being between 0·1 and 1·0 microns. In electric welding, iron oxide coming hot from the welding arc is a fume, but when it is in the form of rust knocked off an iron girder it is a dust. Smokes from burning carbonaceous fuel like coal, oil and wood contain droplets as well as dry particles. Tobacco smoke is typical of the wet smokes and is composed of minute tarry droplets of particle size about 0·25 microns (Drinker and Hatch, 1954).

Fogs and Mists

Mists are represented by atmospheric fogs and are essentially tiny liquid droplets, often microscopic in size, condensed about solid particles such as carbon, as a nucleus. They are composed mainly of water, and their particle size is of no particular importance, except that the particles must be in the microscopic range if they are to travel any distance. We have already seen (p. 484) the importance of the drift of mist in agricultural spraying where the weed-killers used are poisonous to man. Another example of interest in industrial toxicology is the mist entrained from the acid baths in chromium plating (see p. 404).

Size of Vegetable Particles

No fine particles can float for long in still air unless their particle size is well under 10 microns. The diameter of a red blood corpuscle is 7·2 microns, a white blood cell about 16 microns and a pollen grain of the common ragwort, *Senecio Jacobæa*, about 20 microns. A person with normal eyes can easily see an object 50 microns in diameter or pick up a shiny fibre of quartz 20 microns in diameter. Animal and vegetable dusts including pollen grains can be air-floated, and if inhaled can produce hay fever and asthma in susceptible persons. Particles of this size become trapped in the nose and upper respiratory passages.

Size of Lung Alveolus

It is incorrect to assert that in occupational medicine we are concerned solely with dusts in the range 0·5 to 5·0 microns. In the sputum of men exposed in dusty occupations, in the ashed lung specimens of men who have died of silicosis, and in the lungs of animals which have been dusted experimentally, the most representative dust particle found measures about

1 micron in diameter. It is often assumed that anything larger than 3 microns in diameter cannot get into the alveoli of the lungs. But these structures measure up to 100 microns across and there seems to be no reason why particles much larger than 3 microns cannot get into them. Surely the factor which limits the size of dust particles found in the lungs is the diameter of the lymphatic channels and not the alveoli (McLaughlin, 1953).

Defence Mechanisms of the Body against Dust

Healthy lungs can deal with a certain quantity of dust or fume without becoming damaged. The defence mechanisms of the body against dust are briefly: (*a*) the vibrissæ of the nose, which act as a partial filter for the larger particles; (*b*) the mucous secretions of the nose and the upper respiratory passages, in which a large proportion of the particles is trapped and then expelled by (*c*) the wave-like action of the cilia of the nasal and bronchial epithelium. Below the respiratory bronchioles, where there are no cilia, (*d*) the phagocytes come into play. They engulf the dust particles and take them up to the area of ciliary action, or into the lung lymphatics. There is some doubt whether or not dust is taken into the lung parenchyma by phagocytic action, but the fact remains that dust does get into the lungs, whatever may be the exact method of locomotion.

Amount and Type of Dust Inhaled

The concentration of dust which can be inhaled without danger varies according to the nature of the dust and also to the length of time it is breathed. Again, the intermittent exposure to high concentrations of dust may be more dangerous than exposure to lower concentrations over a longer period. The harder the job is, the more deeply will a man have to breathe, and in consequence he will breathe more dust. In tunnelling through siliceous rocks for hydro-electric schemes in the Alps of France, Switzerland or other high mountains, the dyspnœa brought on by the high altitude increases the quantity of dust inhaled.

Individual Susceptibility

Individuals, too, vary greatly in their capacity to deal with dusts, and of two men who have been working at the same job for the same length of time one may get a disease of the lungs and the other may be unaffected. The reasons for the differences in individual reaction to dust are not accurately known, but it is likely that they depend on anatomical, physiological and biochemical variations from one person to another. It is known, however, that previous damage to the lungs is a factor which leads to the retention of dust in them. In any case, there are instances where people have spent many years in the dusty trades and have died from causes other than the dust diseases. On the other hand, many thousands have died as a direct result of the inhalation of dust.

Action of Silica on Lung Tissue

Although in silicotic lungs van Diemerbroeck (1672), Peacock (1860) and Greenhow (1865) all showed the presence of hard gritty material and isolated what they called *sand* from the ashed tissue, the manner in which this material exerts its effect on pulmonary tissue remained a matter for speculation.

The Mechanical Theory of Silicosis

It was first thought that the fine particles of stone dust cut and lacerated the tissues with which they came into contact. As viewed under the microscope the mineral particles certainly showed sharp edges and points, but it was nevertheless difficult to believe that such extremely fine material could cause trauma. As a consequence, doubts were early expressed as to the validity of this *mechanical theory* of silicosis. Two crucial experiments turned the attention of investigators to an alternative hypothesis. The first of these was by Gardner, who in 1923 brought convincing evidence against the mechanical theory by demonstrating that the dust of silicon carbide, which is extremely hard and has just as sharp edges as stone dust, failed to produce the typical fibrous reaction which powdered quartz dust would cause when placed in the lungs of animals. These classic experiments of Gardner's marked the beginning of the modern theories and investigations of silicosis. The second investigation which threw doubt upon the mechanical theory of silicosis was that of Gye and Purdy (1922). These workers produced in the lungs of mice treated with amorphous silica a lesion characterized by acute inflammation and necrosis, and in the liver a condition of necrosis by the injection of colloidal silicic acid. This work pointed to the possibility that stone dust might be harmful to the lungs, not because it was hard and sharp but because it produced a soluble substance which was toxic to the tissues. Policard (1933) showed that cells poisoned with silicic acid do not disintegrate and disappear as do other cells, but tend to preserve their structure in a fashion which suggests mummification.

The Chemical Theory of Silicosis

Kettle (1932) brought the final proof that the dissolution of a soluble substance from the stone particles was necessary to produce silicosis by demonstrating that the same quartz particles which readily produced silicosis in animals would no longer do so if they were first coated with a thin layer of iron oxide, which did not alter the sharpness but which effectively prevented any part of their substance from going into solution. Thus the modern *chemical theory* or *solubility theory* of silicosis supposes that the fine particles of stone dust which get into the lungs are pathogenic, not because they produce microscopic trauma but because something of a toxic nature dissolves from their surfaces. This toxic substance is thought to be silicic acid, and it might be supposed that the pathogenicity of any stone dust would bear a direct relation to the rate at which it will release silicic acid into solution. Broadly speaking, this is usually the case (King and Belt,

1938). Quartz and flint, which dissolve to the extent of about 10 milligrams of silica per 100 millilitres of blood plasma, are the most pathogenic of the mineral dusts, and shale and mica dusts which dissolve to the extent of only about 1 to 2 milligrams of silica per 100 millilitres are among the least pathogenic.

Rôle of Silicic-acid Polymers

Nevertheless King (1947) was careful to emphasize some anomalies which are not easily explained by the simple solubility theory. Thus cement, which releases high concentrations of silicic acid in the body, is relatively harmless. The work of Holt and Osborne (1953) and of Holt and Yates (1954) suggests that the rôle of silicic acid in silicotic tissue is analogous to that of the muco-polysaccharides of high molecular weight which are present in normal fibrous tissue, and that only highly polymerized silicic acids are capable of producing fibrosis. Ortho-silicic acid is formed at the surface of quartz particles at a low *p*H and, when neutralized by the tissues, the silicate ion must pass through the region of *p*H 5·5 to 6 in which polymerization is most rapid. Cement dust also dissolves to produce a high concentration of silicic acid, but at an alkaline *p*H, so that in diffusing away from the dust particle the silicate ion is brought to the *p*H of the plasma without passing through the *p*H range in which rapid polymerization occurs. Consequently, in the case of cement, large polymers (polysilicic acid) are not formed, and this may explain the relatively harmless nature of cement dust.

The Occupations in which Silicosis Occurs

In reviewing these occupations it is found that in all of them the workers are exposed to the risk of inhalation of dust of uncombined silica, SiO_2, in the form of quartz, or the chalcedonic forms as flint, chert and Tripoli, and occasionally in an amorphous form as in kieselguhr and Neuburg chalk. The free silica may be the only constituent of the dust as, for example, in sandblasting with quartzose sand and in crushing flint, or it may occur in the dust mixed with other substances, either naturally, like the quartz in granite, or artificially like the ground flint in earthenware pottery. The characteristics of the diseases which result from exposure to pure silica and mixed dusts vary in a way which appears to bear a definite relationship to the mineralogical composition of the dust.

(1) The Gun-flint Industry

The flint-knapping industry at Brandon, Suffolk, represents the only survival in any civilized country of the most ancient industry of mankind. Prehistoric man made flint tools and weapons in this district at a very early date, estimated at about 6000 B.C. Flint-lock firearms were introduced about the year 1695. The flint is mined at Lingheath, south of Brandon, at a depth of about 30 feet and the large nodules are brought to the surface by hand.

Operations and Processes

These men still use tools similar in shape to the deer-horn pick of their prehistoric ancestors (fig. 365). The nodules (fig. 366) are conveyed to wooden sheds where the flints are shaped. The flint worker rests the nodule on a thick leather knee-pad and strikes it with a quartering hammer (fig. 367). The quarters are then worked up on the pad into suitable cores, and from these flakes are struck off with a flaking hammer. Operations are now transferred to the heavy wooden knapping block. In a hole in the block is a steel stake which is packed round with leather and is thus firmly held, not in contact



FIG. 365.—Flint Miner of Brandon with an Iron Tool similar in shape to the Deer-horn Pick of his Prehistoric Ancestors
(By courtesy of the Daily Mirror)

with the wood, but in such a manner that it possesses the resilience when struck which is necessary to the proper knapping of flint.

The Art of Flint Knapping

The flake is held in loose contact with the front edge of the stake and is struck a light blow with the knapping hammer. It is fractured by the resulting upward blow on the underside of the flake due to the rebound of the stake. In this way both ends of the flake are removed, leaving the central portion which, after trimming, is the completed gun flint. This is thrown into one or other of the receptacles standing on the block, according to its size and quality. Often three or four gun flints can be made from one flake. The art is one which requires a very high degree of skill. Gun flints are made to standard sizes as judged by the trained eye of the skilled knapper.



FIG. 366.—Large Nodules of Flint which have been brought to the Surface by Hand

(By courtesy of the Daily Mirror)



FIG. 367.—Flint Knapper of Brandon at work in his Shack. Resting a Nodule on his thick Leather Knee-pad he is about to strike it with his Quartering Hammer. Note the absence of Ventilation

(By courtesy of the Daily Mirror)

These are known as wall piece, long dane, fowling, musket, carbine, horse pistol, single pistol, and pocket pistol, and they vary from pieces ½ inch to 1 inch in diameter. They are square, flat on one side and with bevelled edges on the other side, the faces showing the typical conchoidal fracture of flint (fig. 368).

A Dying Industry

The flints are packed in small sacks of 200 and are exported for bartering with natives of Africa. Until the year 1835, when the percussion cap superseded the gun flint, the bulk of flints used in the British Army were supplied by the Brandon workers. Flints were also exported to other countries, and there is no doubt that Napoleon's soldiers at Waterloo shot down English soldiers with firearms provided with Brandon flints. In 1800 the industry employed 200 men. In 1846 there were about 100 workers. In the nine years prior to 1889 sixty million flints were manufactured (Bridge, 1933). In 1896 there were about eighteen workers and in 1911 only twelve. By 1931 the industry was rapidly dying and only a few part-time workers remained to make a few gun flints for the old flint-lock firearms which are still used in some parts of Asia and Africa.

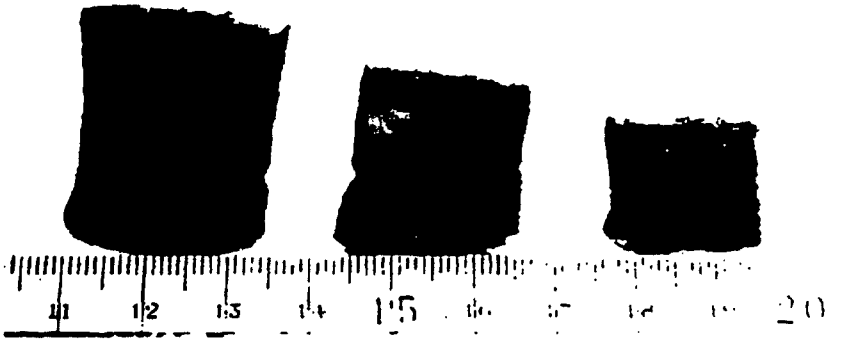

FIG. 368.—Bevelled Flints, actual size, as used for the Flint Locks of Guns, Pistols and Lighters
(London Hospital Medical College Museum)

Characters of the Dust

The flint knappers generated by their hammering a large amount of fine flint dust. Middleton (1930) took samples of atmospheric dust at breathing level of two men knapping flints. The counts gave up to 1,313 particles per millilitre with 2 per cent of particles over 2 microns in size. Most of the particles were under one micron in diameter. They worked in

ill-ventilated sheds and undoubtedly they had silicosis and suffered a high mortality from phthisis. A similar state of affairs was described in 1831 among the population of Meusnes in France when the gun-flint industry was introduced (Collis, 1915).

Methods of Prevention

As the workers maintain that the flint must be dry for proper working, water cannot be employed. Localized exhaust draught would be too costly for this decadent and precarious industry, in which no mechanical power is used. Respirators, standardized to protect against this very fine dust, would be difficult to attain, but at least partial protection might be afforded in this way and, together with the intermittency of the work and the possibility of working in the open air, as is done in summer, sufficient relief from exposure might be attained to carry the worker through his life without a disabling degree of silicosis (Middleton, 1930).

(2) The Sandstone Industry

The sandstone quarrying, mining and dressing industry represents perhaps the most widespread of all silicosis-producing industries in Great Britain. To a great extent, processes in cutting, shaping, dressing and crushing of the stone are carried out near the places where it is got from the quarry or mine.

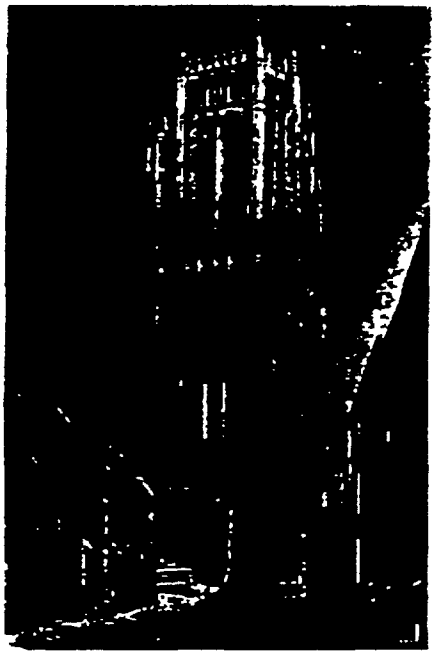

FIG. 369.—The Tower and South Porch of Liverpool Cathedral
(Photograph by Photochrom Co., Ltd.)

Distribution

The sandstone-producing districts are the northern, central and south-western counties of England and the south and east of Scotland. Chepstow Castle and Liverpool Cathedral (fig. 369) are built of red sandstone, and Bristol Cathedral and Furness Abbey are part sandstone and part limestone. Early in the nineteenth century a good deal of Edinburgh was built in Craigleith sandstone, the supply of which is now exhausted. The famous pennant stone from the Forest of Dean is grey sandstone, that from Yorkshire is mostly brown, and a beautiful red sandstone is quarried in Corsehill, Dumfries. Other quarries are in or near Elgin, Alnwick, St. Bees, Carlisle, Penrith, Lancaster, Bolton, Bacup, Darley Dale and Matlock. Sandstone

is found in many countries of the world; the Union Building in Pretoria is of local sandstone.

Characters of the Dust

Sandstones are sedimentary siliceous rocks containing from 75 to 95 per cent of free silica and consisting more or less of quartz grains mixed with other minerals, and held together by a cement of varying composition and proportion. The composition, amount and hardness of the cement are important factors in determining the dangerous characters of the dust produced. In Great Britain about 12,000 men are employed on the processes in quarries, and in addition there are about 20,000 sandstone masons employed in builders' and sculptors' yards throughout the country.

Operations and Processes

The workers include rock-getters who work at the stone face in quarry or mine; quarrymen or stone cutters who rough-hew the blocks; masons who shape and carve the stone to dimensions and patterns; planers, sawyers and turners who operate stone-cutting machines; drillers with pneumatic hand tools; crushermen, labourers and cranemen; and builders, fixers, wallers or wallstone dressers who frequently do some dressing of the stone. The action of wind has the most important beneficial influence for workers employed in the open. Unfortunately, the worker is not always able to stand to windward of the point of origin of the dust produced by neighbouring workers.

Work of Stonemasons in Closed Sheds

The stonemason working in closed or partly closed sheds is liable to be exposed to dust produced by his neighbours as well as by his own work. Danger is increased by the practice of brushing the dry dust and debris from the surface of the stone and by blowing with the mouth while carving. Wetting the surface of the stone by rain has some influence in diminishing dust, more especially in getting the stone from the quarry, but it has little effect in reducing the fine dust given off by the action of a cutting tool.

Work at Stone-crushing Plants

Stone-crushing plants are frequently found in quarry sites for using up rubble to make road material. At the crushers, elevators and screens, dense clouds of dust are frequently given off and travel for considerable distances, so that though few workers may be employed on the crusher-house plant, many may be subjected to the dust produced by it.

Incidence of Silicosis

In 1929 Sutherland and Bryson investigated the occurrence of silicosis in sandstone workers in England. They examined clinically 454 workers, and of these 266 were selected for radiological examination. Judged by X-rays, 112 of these men had silicosis. The workers most affected are the

stone masons and after them the quarrymen, rock-getters, planers and wallstone dressers. Tunnel miners and grave-diggers working in sandstone may be affected too. The disease appeared to become more common after forty years of age, and after twenty years in the industry.

(3) The Granite Industry

The granite industry assumed importance in Great Britain after the destruction by fire of the wooden town of Aberdeen in 1741. The present Granite City was planned and gradually built up of stones fashioned in the local quarries. Although the art of polishing granite stones for ornamental purposes was carried out in ancient Egypt, it became a lost art until it reappeared in Scotland about 1820. Since that date the city of Aberdeen has become the centre of the monumental granite trade, and the building and monumental sections of the industry have flourished side by side (Mair, 1951). Today, more than 80 per cent of the granite used for the manufacture of monuments in Aberdeen is imported from Norway, Sweden and Finland.

Distribution

Granites and allied rocks of igneous origin are found in many parts of the world. They are characterized by a crystalline structure and a certain hardness or toughness which demands special methods for quarrying them and adapting them for use. True granites are found in the west and north of Scotland and in Cornwall, Devon and the Lake District of England. Other igneous rocks resembling granite are distributed in many parts of Great Britain.

Characters of the Dust

True granite consists of orthoclase feldspar, quartz and mica. The chemical composition is distinctly acid, there being from 65 to 75 per cent of silica. In intermediate rocks—for example, the syenites and diorites—the silica percentage varies from 55 to 60; while in the basic rocks, dolorite and gabbro, the silica content is from 45 to 55 per cent.

Operations and Processes

The occupations in the granite industry may be classified as follows:

(i) *Labourers* are unskilled workers employed in removing overburden, or loading and filling granite, and they may assist in blocking, getting or drilling.

(ii) *Getters* are skilled quarrymen who get the granite from the quarry face and roughly square it into blocks of suitable size. The group includes blockers and rockmen.

(iii) *Drillers* include workmen employed in using all forms of drills, including hand drills, steam drills, and wet and dry air drills.

(iv) *Settmakers* shape the setts or stone blocks for road material by means of hand hammers. They work in a shelter or open shed.

(v) *Kerb-dressers* are sometimes referred to as masons, particularly in Leicestershire.

(vi) *Crushermen* include all workmen employed in crushing mills and in concrete works. The group includes breakermen, screenmen, oilers, labourers about the mill and loaders.

(vii) *Building masons* are skilled workmen engaged in cutting and dressing granite in builders' yards. Some of these work only with hand tools, and others use pneumatic tools. The use of the pneumatic tool varies considerably in different districts. In Cornwall it is used to a comparatively



FIG. 370.—Dunter used for smoothing Granite. Note extensive Dust Cloud produced in spite of the arrangement for providing localised Exhaust Ventilation

slight extent. In Aberdeen the pneumatic tool was found to be more in evidence among building masons. These pneumatic tools may be for cutting or surfacing. Building masons work in open sheds.

(viii) *Monumental operatives* or monumental masons. This group includes squarers, duntermen, finishers and turners working in monumental yards. The squarer uses a hand chisel and a pneumatic cutter. The dunterman is employed in operating the pneumatic dunter or surfacing machine (fig. 370). The finishers use the pneumatic tool almost exclusively for their work. Turners work with a power-driven lathe. The squarers can work in a shed by themselves or in the same shed as finishers. The duntermen work in widely open sheds in the yard. Turners are, as a rule, in the same shed as the polishers.

(ix) *Polishers* are employed in monumental yards. Polishing is a wet process; it may be done by hand but, as a rule, machinery is used.

Incidence of Silicosis

In 1929 Sutherland, Bryson and Keating investigated clinically 494 workers in granite, of whom 211 were examined radiologically. Judged by X-rays, thirty-six of these men had silicosis. Examination of the radiographs in the positive cases classed as silicosis shows that the prevalent type of the shadows indicating fibrotic changes is somewhat different from that found in workers exposed to silica dust in other industries. In the radiographs of granite workers there are usually the increased hilar, linear and reticular shadows, but instead of the discrete, dense nodules found to a greater or less extent over the whole of both lungs, there is a diffuse, cloudy or woolly effect with more or less definite fine or very fine nodules occurring in areas irregularly placed over the lungs. In the whole series the radiograph showing the most discrete nodules of the silicotic type is in the case of a man who worked for nine years as a driller on granite, but had been employed for eighteen years as a tin miner underground. Amongst the other cases, who had been almost exclusively employed on granite, there appears to be some relationship between the composition of the rock and the character of the fibrosis, as shown on the radiograph.

Different Type of Fibrosis

Clinical evidence of fibrosis, as distinguished from radiological evidence of silicosis, was found in 260 cases, or 52·6 per cent of the 494 workmen examined; but X-rays showed silicosis in 17 per cent only. Comparing the results of the medical examinations in the sandstone industry and the granite industry, the proportion of cases of fibrosis amongst sandstone workers was 59 per cent of those examined, compared with 52·6 per cent in the case of granite workers. The proportion of cases of silicosis in sandstone workers was 42 per cent of those radiologically examined, and 17 per cent in the case of the granite workers. If fibrosis of the lungs, diagnosed by clinical examination, be regarded as representing a slighter or earlier involvement of the same character as silicosis, then it would seem that granites and the igneous rocks of granite type produce less injury to the lungs than do the sandstone. Having regard to the appearances of the radiographs in the two series of workers, there is a probability of a difference in character of the types of fibrosis produced by the two kinds of dust, and this probability is increased by the proportion of cases of fibrosis in the granite series showing an approximation to that found in the sandstone series, while the proportion of silicosis cases remains far behind (Middleton, 1930).

(4) The Pottery Industry

We have seen (p. 61) how the Potteries began before there were canals or railways, and how an export trade was established using packhorses and

mules for the transport of raw materials and finished goods. Today the North Staffordshire Potteries consist of the towns of Tunstall, Burslem, Hanley, Stoke, Fenton and Longton, federated in the County Borough of Stoke-on-Trent. Before 1939 they gave work to 67,000 persons, of whom 55 per cent were women and girls. Among these, nearly 35,000 were employed in general earthenware, more than 10,000 in china, just over 18,000 in tiles, nearly 6,000 in sanitary earthenware and sanitary fireclay, about 4,000 in electrical porcelain and just over 2,000 in Rockingham and jet (Meiklejohn, 1946).

Calcined Flints for Chinaware

In 1720 Astbury and Josiah Wedgwood introduced calcined flints into the body of their chinaware so as to produce on firing a remarkably white and hard product. In 1865 in her *Life of Josiah Wedgwood*, Eliza Meteyard states that at first the flint, being used but sparingly, was kept in cellars and private rooms, and when reduced to powder in large iron mortars it was passed through hair sieves. This process was slow and extremely injurious as, in spite of every precaution, the dust was inhaled by the workmen and produced lung diseases of various kinds. She relates in detail how a painter employed in the potteries was struck by overhearing the story of the fatal effects of the flint. It occurred to him that the crushing could be performed by millstones under water, and the adoption of this method led to a considerable reduction in the amount of dust. Unfortunately, in the manufacture of ceramics in certain circumstances the dry method for the milling of flint is still employed.

Branches of the Industry

The industry includes several distinct branches, sub-division being determined by the nature of the article being manufactured and the materials entering into the composition of the ware. The principal divisions are: earthenware, tiles, sanitary earthenware and electrical fittings; china; jet and Rockingham; sanitary fireclay; stone-ware; and coarse ware. The occurrence of silicosis is especially associated with the first two groups—namely, earthenware and china—and the others will not be further considered.

Earthenware

In the manufacture of earthenware, the ingredients—ball clay, china clay, china stone, feldspar and flint in a ground state—are mixed together in the form of liquid slip, from which excess water is removed by pressing. The resulting composite body is made into the consistence convenient for the manufacture of the various articles. So long as it remains moist it is harmless, but in the process of manufacture fragments fall on benches and floors or adhere to the clothing of the workers, become dry and give rise to dust. After the article has been made in the plastic form, partly dried, it is frequently smoothed on a revolving disc and fettled, to remove irregularities of surface and edges. The articles are then placed in fire-clay saggars

with some sand and fired in the oven. On removal from the oven the loosely adherent sand is brushed off. Subsequent processes of decoration and glazing are not associated with the occurrence of silicosis.

Chinaware

English china is made from calcined bone, china clay and china stone, but it contains no added flint. The processes of making the articles are similar, in general, to those employed for earthenware, but the articles require much more support in the process of firing than do earthenware articles, and to provide this support they are placed in finely powdered flint. The flint is prepared by calcining and subsequently crushing it in the dry state. When the saggars of china ware are taken from the oven, the flint, which was used in placing the ware, is removed by women in a series of processes known as scouring.

Incidence of Silicosis

Finally, after the ware is decorated and glazed, blemishes are removed by polishers, who frequently use finely powdered flint mixed with water as a finishing abrasive, on a rapidly revolving wheel. It will be seen that the exposure to flint dust in the pottery industry occurs in the processes of the preparation of the flint by grinding; in the manufacture of general earthenware, earthenware tiles, sanitary earthenware and electrical fittings, amongst those manipulating the earthenware body in the plastic and semi-dry states; in the manufacture of china, those employed in placing the biscuit ware in powdered flint and in removing the flint from it after firing; and in polishing the finished ware with the use of flint. It is in those processes that the highest incidence of silicosis is found to occur (Middleton, 1938).

(5) Gold Mining

The prevalence of silicosis in certain gold mines has been of immense importance to the industry, and the researches which have been carried out with a view to preventing the disease and providing compensation for injured workmen have contributed greatly to the sum of present-day knowledge on the subject. This applies to important goldfields in several countries, including those in the Transvaal and Ontario. In 1902 attention was drawn for the first time to the disease known as *miners' phthisis* (fig. 361). Careful inquiry at that time showed that out of 1,377 men who had been employed on rock drills in the Witwatersrand prior to the Boer War, 225 were known to have died from miners' phthisis between October 1899 and January 1902, an annual death-rate of 73 per 1,000. Medical examinations of all white miners, of whom there were over 21,000 employed in 1934–5, are made twice a year. Miners have been recruited from other countries for work in gold mines and many of them return to their own country if they become disabled by silicosis. During each of the six years, 1930 to 1935, an average of twenty gold miners from South Africa died from silicosis in England.

(6) Tin Mining

In the Cornish tin mines the mother rock is granite. The *Report on the Health of Cornish Miners* (1904) refers to the high mortality from respiratory disease among rock-drillers in the mines. Out of 142 deaths of rock-drill men, 120 were certified as from various forms of phthisis or *miners' disease*, and thirteen from other repiratory diseases. The majority of these rock-drillers had worked in other countries and ninety of them had worked drills in the Transvaal. There is no doubt, however, that silicosis has been caused by working in the tin mines of Cornwall, for thirty-eight of the rock-drillers referred to in the *Report* had worked in Cornwall alone.

(7) Hæmatite Iron-ore Mining

We have seen (p. 833) that hæmatite mining has been carried on in Cumberland since the Roman occupation and that silicosis really began there only with the introduction of the pneumatic drill. During the six-year period, 1930 to 1935, forty-eight hæmatite miners were certified in England as having died from silicosis. No case was certified during the year 1930, and the numbers for the subsequent years were 10, 7, 6, 13 and 12 respectively. Until about 1930 silicosis had not been generally regarded as an occupational risk in this industry. At that time it was pointed out that the Cumberland and Lancashire group of iron-ore miners had a higher mortality rate than the rest of the ironstone group for phthisis and other respiratory diseases, while the maximum incidence for phthisis was at a later period. Since that time a form of pulmonary fibrosis, frequently accompanied by tuberculosis, has been proved to exist in this industry. The change in the outlook may have been brought about by a change in methods of working. About 1913 hand-drilling was replaced by machine-drilling and with it more frequent blasting was made possible.

The dust to which hæmatite iron-ore miners are exposed differs from that with which other workers come into contact who suffer from silicosis, and in view of this important point in the ætiology of the disease, it may be well to consider certain geological data. Much of the hæmatite deposits of Great Britain are precipitates from ferruginous solutions which must have been in most cases charged with CO_2, and such solutions would carry dissolved silica, which would be simultaneously precipitated with the iron, generally in the form of finely divided quartz. Also, in cases where the calcium carbonate of calcareous rocks has been replaced by iron compounds, all the insoluble siliceous constituents of the original calcareous rock will find their place in the newly formed iron ore.

Thus a siliceous limestone will give place to a siliceous iron-ore, in which the silica is mainly in the form of detrital quartz. Further, quartzose rocks may form one side of a lode of ore and these have often to be blasted and cut in order to work the ore-deposit. Insoluble residue of better-class ores amounts to 5 or 7 per cent, but rises to 15 per cent, mostly in the form of free silica, and in lower-grade ores the insoluble residue and free silica may rise to much higher figures. Some ore bodies may have definitely

siliceous courses and the bounding wall of a lode may be highly siliceous (Middleton, 1938).

(8) Coal Mining

In 1920 Middleton, working as a tuberculosis officer, recorded the toll of silicosis among men driving hard-headings in South Wales coal mines. Almost simultaneously the doctors of Radstock in the Somerset coalfield noticed a chronic lung disease in miners employed in *branching*. This work involves machine drilling in driving roads through highly siliceous sandstones called Pennant rock, grit or flag and known locally as *greys*. In 1924 one of these men died and the lungs at necropsy showed silicosis. In 1925, twelve of these rock-drillers were examined by X-rays; all but one were found to be suffering from silicosis, the one case being doubtful (Fisher, 1935). A local study of the problem by Kemp and Wilson (1946) has confirmed the serious incidence of silicosis among Somerset miners engaged in drilling and blasting in Pennant rock.

Only a small percentage of coal miners develop classical silicosis. The processes underground in a coal mine which may involve exposure to silica dust are:

(i) *Ripping:* taking down the roof or top of a roadway, to make height.

(ii) *Brushing:* ripping or blasting of the roof and using the debris for building stone packs.

(iii) *Driving a hard-heading:* which is a drift, tunnel or roadway driven in rock or through hard measures.

(iv) *Driving a cross-measure drift:* which is driving a roadway in such a direction as is necessary to form a travelling road from stratum to stratum.

More than sixty names have been given to variants of this job (see p. 197). Hand drills and compressed-air percussive drills are used. The dusts met with in these processes are mainly those evolved from clift, bind or shale, which may contain as much as from 40 to 60 per cent free silica and rock, bastard rock or sandstone, in which the free silica may range from 60 to 85 per cent.

(9) Graphite Mining

The name graphite is derived from the Greek γράφειν (I write), and because in early days it was mistaken for lead it was called plumbago and black lead. Approximately 12 per cent of the world's graphite is used in pencils and crayons, whereas 60 per cent is employed in foundries and in making graphite crucibles for metallurgical purposes. Finely powdered graphite finds extensive use as a lubricant either alone or more commonly with grease. Graphite occurs in various parts of the world, always in association with quartz. Formerly, for many years Ceylon and Madagascar dominated the graphite market and they still have a virtual monopoly of high-grade crystalline graphite for crucibles.

Harding and Oliver (1949) found that samples of graphite from Ceylon, S.W. Africa and Korea contained from 3·6 to 10 per cent free silica; Par-

meggiani (1949) states that in Italian graphite from Pinerolo free silica is about 11 per cent. The clinical, radiological and pathological findings in workers exposed to dust of graphite have been described by Dunner (1945), Dassanayake (1948), Gloyne, Marshall and Hoyle (1949), Dunner and Bagnall (1949) and Harding and Oliver (1949). The radiological evidence of pulmonary changes often preceded subjective symptoms, and occurred after ten to fifteen years or more; it included scattered nodular shadows, and rounded conglomerate masses which varied in shape and size and, in some cases, were seen to contain cavities; their presence was confirmed by



FIG. 371.—Workman at a Slate Quarry splitting the Rock with a Chisel to make Roofing Slates

necropsy. The clinical picture was that of a slowly developing silicosis, whereas the radiological and pathological findings more closely resembled those of pneumoconiosis of coal miners; copious black graphite-containing liquid was found in the lungs, with number of *graphite bodies* resembling asbestosis bodies in general form. The large amount of carbon probably acted by upsetting the expulsive power of the lungs and thus allowed the quartz to exert prolonged localized action.

(10) The Slate Quarrying and Dressing Industry

This industry is concerned with quarrying or mining the rock and making and shaping slates for roofing and other structural work. The large blocks of slate are conveyed to the mills, where they are sawn to sizes with power-driven saws cutting across the grain. The blocks are then split with chisel and mallet and cut to required sizes by hand or machine (fig. 371)

Slate is the typical cleaved rock. The most important constituents quantitatively are silica, free as quartz, and combined as silicates of aluminium, iron, alkalis and other bases. In Penrhyn slates, quartz exists to the extent of from 35 to 43 per cent, while the total silica (free and combined) is from 58 to 63 per cent.

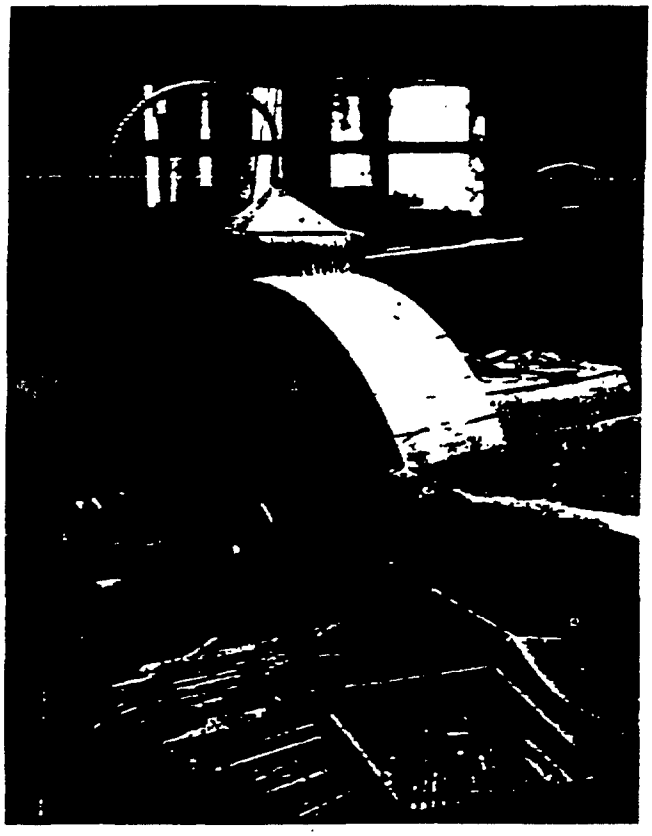

FIG. 372.—Dust Suppression at hacking and rodding a Grindstone. Note the continuous Stream of Water
(By courtesy of Messrs. Platt Bros. & Co., Ltd.)

(11) Millstone Dressing

This is an occupation in which a comparatively small number of persons are employed. It consists in dressing the millstones for the grinding of corn in distilleries, corn and fodder mills, and for grinding chocolate in chocolate factories. The process is carried on in many districts throughout Britain where the mills exist. The persons employed may be specially skilled men who have learned the trade and are solely employed on the process, or the work may be done as a part-time occupation. The corn mills consist of two circular stones placed horizontally. They may be composed of any hard stone, but are usually a hard quartzite grit, chert or buhrstone, a chalcedonic

form of silica, or artificial stones. Dressing of the stones is done with an adze-shaped steel tool, by chipping along the ridges which run radially from the centre. During chipping, a slight cloud of fine dust rises (Middleton, 1930).

(12) The Grinding of Metals

The processes which cause silicosis in the grinding of metals are confined to those in which grindstones composed of natural sandstone are used.

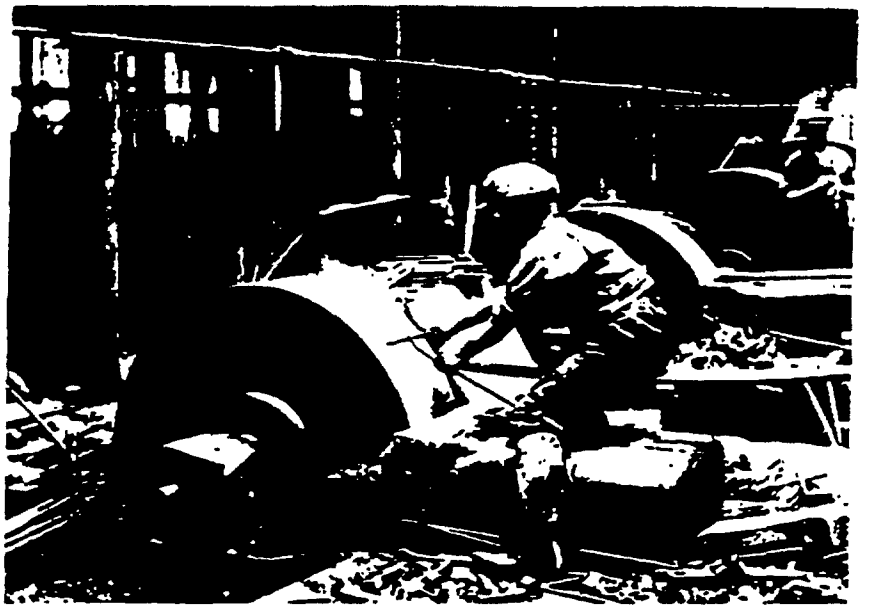

FIG. 373.—Rodding a Grindstone. Note the Cloud of Dust from the Sandstone Wheel as the workman smoothes its Surface by bearing down upon it with a Rod of Steel

(By courtesy of Messrs. Platt Bros. & Co., Ltd.)

The industries in which metal-grinding is an important process can be divided broadly into (i) the cutlery and edge-tool trades, and (ii) the machinery, general engineering and foundry works.

Grinding may be done wet or dry and the metal being ground may be moved by hand or by mechanical power on a machine. In wet grinding, water is laid on from a tap or spray above the stone and is drained away beneath it (fig. 372). Wet grinding is done by machines, especially in the manufacture of files, saws and machine-knives, when the metal being ground is fixed to a part of the machine which moves under or across the revolving grindstone. The processes of dressing the grinding surface of the grindstone are very important from the health point of view, on account of the high concentrations of dust given off (fig. 373). Dust is produced by attrition of the grindstone in all processes in the grinding of metal and it varies in amount with the consistence of the stone, the hardness of the

metal, the shape of the article being ground and the amount of force exerted (fig. 374).

The grinding of metals on sandstone wheels has been recognized for generations as a cause of silicosis and an increased mortality from tuberculosis. A decline in the number of deaths has occurred from two causes: (i) the change from sandstone to artificial abrasive wheels, and (ii) the effect of stringent preventive measures in the grinding rooms. At the



FIG. 374.—The workman is cleaning a Casting with a Portable Grinding Wheel. Sparks visible to the Naked Eye suggest that Dust is scattered laterally away from his Face. Photography using the Tyndall Beam shows a Vortex of Dust above the Wheel revolving in the same direction and travelling towards his Nose and Mouth

(By courtesy of Dr. A. I. G. McLaughlin, Mr. W. B. Laurie and the British Institute of Foundrymen)

coming into force of the Workmen's Compensation (Silicosis) Scheme for the grinding of metals industry on 1st July 1927, there was a widespread departure from the use of sandstone wheels and a corresponding adoption of the artificial abrasive. Artificial abrasive wheels are composed of carborundum (silicon carbide, SiC) or some form of aluminium oxide, and contain only a small proportion of silica or none at all.

(13) Polishing Compositions, Parting Powders and Silica Washes

The most important forms of silica used for polishing compositions and parting powders are Tripoli and Neuberg chalk. Tripoli is generally re-

garded as a chalcedonic variety of silica, and contains about 97 per cent of silica. The substance generally known by the name is imported from Seneca, Missouri, and is of two kinds, which differ in their mode of origin but resemble each other in composition. The name comes from Tripoli in North Africa, where the product is a true diatomite. It is extensively used as a mild abrasive in metal polishes, and as an ingredient in parting-sand in foundry work. Two fatal cases of silicosis with tuberculosis have occurred from exposure to dust of Tripoli in England. The two men had been employed for periods of ten and eleven years respectively, mixing the ingredients in the manufacturing of parting-sand in one case and of polishing-composition in the other.

Neuberg chalk is a natural deposit of siliceous material occurring at Neuberg on the Danube. The composition varies somewhat in different localities and the silica content is sometimes given as from 65 to 86 per cent. Besides free silica it contains from 7 to 8 per cent kaolin, and traces of iron and magnesium. There are indications that pulmonary changes have followed exposure to the dust. In one factory six of the men employed on a process in which this material was used for periods from three and a half years to seventeen years had died of tuberculosis or silicosis with tuberculosis. Following this experience, examinations were made and early signs of silicosis were found in three men. In another factory one or two of the men who had been employed ten and twelve years on the process showed early signs of silicosis.

Diatomite (kieselguhr, diatomaceous earth) is composed of minute, siliceous skeletons of aquatic plants of marine or fresh-water origin. When pure it contains up to 96 per cent of silica, but it may contain alumina, iron, lime, magnesia and alkalis, reducing the silica content to about 75 per cent. It is used as a mild abrasive or polishing medium, as filtering material and for insulating purposes. Where men handled a diatomaceous deposit in Santa Barbara County, California, which yielded, when dry, a silica content of 85 per cent, much dust was produced in conveying, grinding, drying and bagging. Clinical and radiological examinations were made of 108 men, and silicosis was found in 81 (68·5 per cent); very early in 15, early in 45, moderately advanced in 15, and advanced in 6.

In a process in the manufacture of steel, in which a number of men had been exposed to dust of kieselguhr for six years, in Great Britain, radiographic examination showed more fibrosis than usual, with very early indications of a modified form of nodulation in some of them (Middleton, 1938).

In foundries in the United States of America as late as 1930, one could see a silica wash being used to face the moulds in steel foundries and observe this wash being applied with a spray gun. All persons in the immediate vicinity were then showered with a mist, of which the essential ingredients were water and fine silica made from ground flint, one of the best silicosis producers known (Clark and Drinker, 1935).

(14) Flint Crushing

Flint, a chalcedonic form of silica, is crushed for making chicken grit, sandblasting grit, abrasive papers and brake-sand for tramcars. The flints may be first calcined, in order to make the material more friable and whiter, before crushing. High concentrations of dust are produced at the crushing and screening processes and it is an extremely difficult problem to control and dispose of it. Sand becomes dangerous when it is dried and manipulated in such a way as to give rise to fine particles of quartz dust. This occurs in sieving dried sand for sandblasting, for facing bricks, in the manufacture of glass and other products, and in mixing dried sand with other substances.

(15) Silica Milling and the Manufacture of Abrasive Soaps

These processes may be carried on in the same factory, but more usually silica milling is done in one factory and the ground material is supplied to manufacturers of abrasive soaps. The silica may be quartzite rock containing over 90 per cent of silica, or crystalline quartz, or quartz-sand. It is dried, crushed and ground to a fine powder by machinery, which is totally enclosed and provided with exhaust draught to remove dust. In the manufacture of abrasive soaps, ground silica is mixed with powdered soap and anhydrous sodium carbonate. Dust is produced in the processes of mixing, sieving and packing the powders. About 1928, numerous firms who had no knowledge of the dangers of exposure to the dust and were inadequately provided with protective measures became engaged in this manufacture. This resulted in the occurrence at one factory of three or four cases of respiratory disease which was regarded as an acute form of silicosis. The manufacture began in England in 1910, but was not important in quantity until 1921, and in 1928 it was given up owing to the death of workers attributed to the inhalation of the dust (Middleton, 1938).

(16) Refractory Products

This industry has a special interest in relation to silicosis, because it was the first to which the Workmen's Compensation (Silicosis) Act, 1919, was applied. It is engaged in the manufacture of refractory materials which contain over 80 per cent of total silica and are used in the construction of furnaces, flues and crucibles.

It is a small industry; some 3,000 workers are employed in Britain. The processes include the quarrying or mining of the raw material, usually a sandstone of the coal measures called ganister, which occurs in open quarries and in mines, or, in some localities, sands or pocket-clays are used. The raw material, which contains usually from 92 to 98 per cent of silica, is crushed, mixed with suitable bonding substances, and made into silica bricks, silica cement, steel-moulders' composition and similar products for use as refractories in the manufacture of metals, especially steel or steel castings, and in the construction of gas-retorts and flues. Mechanization is

improving conditions in the manufacturing processes, from the health point of view, especially with the use of continuous kilns which the workers do not enter.

(17) Sandblasting

This is the process of projecting sand or other grit, by means of compressed air or steam, against a surface. It is used:

(i) In metal works to remove adherent sand and irregularities from castings.

(ii) To produce, on clean metal, a surface suitable for subsequent treatment by coating with enamel or another metal.



FIG. 375.—Shot-blasting Cabinet, open only for purposes of Photography. Note Air Line to Helmet

(Johnstone, R. T. (1948), Occupational Medicine and Industrial Hygiene, The C. V. Mosby Company)

(iii) For etching glass and treating other non-metallic articles.

(iv) For removing paint from, and otherwise cleaning, large surfaces, as of ships and buildings.

Sandblasting of metal articles can be carried out in various ways. For large articles the operator works in a specially constructed room, and he is protected by special clothing and a helmet which is supplied with pure air under pressure (fig. 375). The dusty air is removed from the room by exhaust draught. Smaller articles are treated in a closed cabinet, the worker passing his arms through guarded holes to direct the abrasive while watching the process through a glass panel. Other types of apparatus are the mechanical turntable and revolving barrel, in which the sandblast is directed from a fixed point and close application of the worker is not necessary.

The dust hazard is from the abrasive when this consists of quartzose sand, crushed flint, quartz, quartzite or granite. On hygienic grounds, metal grit and certain other non-siliceous abrasives have now replaced the siliceous material to a considerable extent, and where these are used on clean metal no siliceous dust is produced; when they are used for cleaning metal castings, however, dust is produced from the adherent moulding sand, which is highly siliceous.

Risks are incurred by workers by faulty or careless methods, or through defects in the appliances, which require constant attention for maintenance. Silicosis is found amongst sandblasters in various parts of the world, and the same features—a short period of employment and rapid course of the disease—characterize all the cases. The average length of employment of

sandblasters before death was at one time about ten years as compared with forty years in the silicosis-producing industries (Merewether, 1936).

Prevention of Silicosis

In order to be certain that people will not get silicosis, it is clear that harmful dust must not get into the air which they breathe. But this presents great difficulties because many factory processes and most types of mining are inherently dusty. Nevertheless, dust clouds *can* be controlled, sometimes partially and sometimes completely.

No Room for Complacency

On the subject of the prevention of silicosis there is no room for complacency, because the figures for deaths are going up every year. And this is happening in spite of the fact that at no time in history has more attention been given to the problem by doctors, engineers, chemists, physicists and others; by organizations and individuals; and by employers and trade unions (McLaughlin, 1953). The practical details of dust control are largely in the domain of the engineers. But the problem is primarily one of health, and it is the business and the duty of doctors to lead the campaign and to inspire the engineers, if necessary, to greater efforts.

Basic Principles of Prevention

Not only can dust be controlled, but in some instances the process can be altered so that no dust is formed. Again, it is possible, occasionally, to replace the substance which gives rise to the dangerous dust by another which is non-toxic or at least not so harmful. But in this event the process will still be dusty if dust control is not applied. There are some jobs in which it has not been possible, up to the present time, to control the dust, and in these it is necessary for the workers to be protected by dust respirators or by breathing apparatus. The basic principles of the prevention of silicosis can be considered under the four headings: (i) replacement of the harmful material by a less toxic one; (ii) dust suppression or control; (iii) personal protection of the worker, and (iv) medical examination.

(1) Replacement or Substitution

Though the principle of replacing a harmful material by a less dangerous one is of the first importance, it is not always practicable to put it into operation. For instance, coal gives rise to enormous amounts of dust, but is indispensable. But when the material giving rise to a dangerous dust can be replaced, then the danger can be eliminated. Thus limestone, $CaCO_3$, has replaced kieselguhr, SiO_2, for use in the slow cooling of steel ingots, and zircon, $ZrSiO_4$, can be used instead of silica flour for mould paints in foundries. There are at least four industries in which the principle of replacement has been applied on a large scale—namely, flour milling, metal grinding, the pottery industry and sandblasting.

Flour Milling and Metal Grinding

The first of these is flour milling. About the year 1877 the introduction of steel rollers for milling corn removed the need for fashioning millstones from natural silica rock (see p. 855). The second is the grinding industry. For many years grinding wheels made of natural sandstone were used in the cutlery, edge tools and other trades. In fact, the natural abrasives—hard, gritty materials, most of them siliceous rocks—have been used in various forms for sharpening, grinding and polishing for 3,500 years. These substances were at one time quarried in vast amounts—for example, Grindstone City, Michigan, in the years between 1835 and 1929 produced 25,000 tons of natural silica stones each year.

Discovery of Synthetic Abrasives

And then in 1891 Edward G. Acheson, of Monongahela, Pennsylvania, made the first synthetic abrasive, silicon carbide, SiC. Seeking to synthesize diamond, he was heating clay and coke in an electric arc. He obtained a product as hard as the natural abrasive corundum, Al_2O_3, and wrongly thought that what he had produced was a compound of carbon with corundum, hence *carborundum*, a misnomer adopted as a trade name for silicon carbide. It has a hardness of 13, compared with 15 for diamond and 10·5 for sandstone. The method of manufacture is simple; sand, coke and sawdust being fused in the heat of the electric arc. Cheap hydro-electric power played its part, especially at Niagara. In 1899 fused alumina, Al_2O_3, or *synthetic emery*, was added to the manufactured abrasives. By 1945 the United States of America was making 30,000 tons a year of artificial abrasives.

Reduction of Silicosis among Grinders

Since about 1923 sandstone wheels have gradually been replaced by wheels made of carborundum, emery or alumina, so that now sandstone is rarely used for any grinding or polishing job. Though it is not certain that the dust of silicon carbide is completely harmless, it is so much harder than sandstone that it gives off much less dust. The number of cases of silicosis among grinders has been dropping year by year and, with the exception of those who grind castings on which there is burnt-on sand, there should be no cases of silicosis among grinders by about 1975.

The Pottery Industry

Another instance in which it has been possible to replace a siliceous substance by a less harmful one is to be found in the pottery industry (fig. 376). In the placing of biscuit ware in the saggars so that they can be fired in ovens, it used to be the practice to embed the china articles in ground flint, which is nearly pure free silica. The incidence of silicosis amongst this group of pottery workers was high, but since about 1934 powdered alumina, Al_2O_3, has been used to replace flint both for placing



FIG. 376.—The Potteries
(Reproduced by permission of the Sunday Times)



FIG. 377.—China Biscuit placing in the Potteries. The workman is seen placing a Plate in a Bed of Alumina which is a safe Substitute for Powdered Flint. Note the Exhaust Ventilation locally applied
(By courtesy of Messrs. Doulton, Ltd.)

china ware and for polishing articles of pottery (*fig.* 377). Since then the number of cases has been getting less.

Sandblasting and Shotblasting

A fourth example of replacement is seen in the process known as abrasive blasting. In this job a stream of abrasive material is blown under high pressure at articles to clean them. It is used, for example, in the foundry industry to clean castings; it is also used to etch glass and to smooth the surfaces of metal before it is painted or enamelled. About 1904 sand was introduced as an abrasive, and though the number of workers was small, the incidence of silicosis and tuberculosis was high and the disease came on quickly (see p. 860). Since about 1920 sand for blasting has been almost entirely replaced by other abrasives such as steel shot and calcined alumina. The use of sand in blasting operations is now prohibited by the *Blasting (Castings and Other Articles) Special Regulations, 1949.*

(2) Dust Suppression or Control

Ventilation may be either local or general.

General Ventilation

General ventilation is usually obtained by having open doors and windows, but this practice is not popular in cold weather and it puts up the cost of heating the workrooms. Air-conditioning is being adopted in some industries with good results. For this type of ventilation, in which filtered air is blown into the room and extracted by fans, the doors and windows should be kept closed. Where there are furnaces or ovens in a workshop, advantage is taken of the upward currents of air caused by the heat. In these rooms the incoming air is brought in low down in the walls and the outgoing air is extracted through the roof. By contrast, in rooms in which there are no hot processes, the air currents are reversed. For example, in a room in which castings are cleaned, the air is blown in high up, either through the roof or the walls, and the air exits are placed low down.

Local Exhaust Ventilation

When dangerous dust is given off, general ventilation is not enough because it allows the dust, even though it may be diluted, to get into the workers' breathing area. In these cases local exhaust ventilation should be installed. This consists of a hood, placed as near as possible to the source of the dust, and a duct leading from the hood to a fan which creates a current of air to draw away the dust. It may be taken to a dust collector or be discharged to the outside air. Exhaust ventilation has been applied to many industrial processes, but it is never really satisfactory unless it is combined with enclosure of the process. An example of this is the chemical fume cupboard, and the principle can be and has been adopted in many industrial processes with excellent results.

Changing the Process

This can be illustrated by the experiments now being made to avoid the use of the pneumatic tool for the fettling of castings. Attempts are being made to burn off the sand adhering to the castings with a very hot flame produced by acetylene, air and a fluxing powder. Though iron oxide is given off in the fume, no harmful dust is evolved, and altogether it is a promising development. Another way to avoid the use of the pneumatic tool is to put the casting into an annealing furnace, in which a great deal of the burnt-on sand is removed from the casting. However, the fundamental method of tackling this problem is to control the technique of moulding and casting in such a way that the casting comes clean away from the mould so that the use of the pneumatic tool or any other method of cleaning is not necessary. In one foundry, where clean castings are the rule, there has been no case of silicosis for about ten years. This is the ideal way of getting rid of a health risk, but so far it has not been possible to apply it generally (McLaughlin, 1953).

Isolating the Process

If the dusty process can be isolated from other parts of the factory, the number of people exposed to the risk can be reduced. In some foundries all processes, from sand preparation and core-making to the final cleaning of the castings, are carried out in one large workroom. The dangerous dust created in fettling becomes distributed through the workroom and affects to some degree all groups of workers. If the coremakers who handle damp sand work in a separate room, they inhale little or no dangerous dust. In general, the best policy is to isolate as far as possible all dusty processes, and to protect the workers engaged in them by good general and local ventilation together with personal protective methods. A less satisfactory way, but one which is better than nothing, is to operate a dusty process at night when the other workers have gone home. In some foundries the *knockout*, that is stripping the castings away from the sand moulds, is done at night.



FIG. 378.—John G. Leyner
(*Holman, T. (1947), Brit. J. industr. Med., 4, 1*)

Wet Rock Drilling

Water, foam and oil may be used for allaying dust. In hard-rock mining, wet drilling is widely used. John G. Leyner, of Denver, Colorado, has

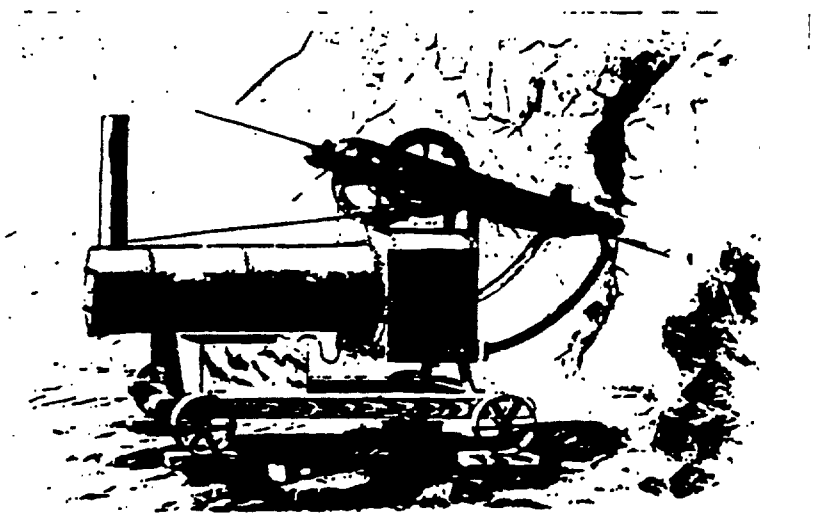

FIG. 379.—The first Percussive Rock Drill, made by Couch in Philadelphia in 1849
(*Holman, T. (1947), Brit. J. industr. Med., 4, 1*)

been called the outstanding mechanical genius of rock drilling (fig. 378). In 1897 he brought to practical success the principle of the pneumatic-hammer drill (figs. 379, 380). A blast of air was blown through a hollow drill-steel to remove rock cuttings from the hole. This produced so much dust that the drill became known as the *widow maker*, and miners refused to use it. In 1898 Leyner obtained a patent on the commingling of air and water in the drill-steel (fig. 381). This is the principle of the axial water feed and is the basis of all wet rock drilling (Holman, 1947). The adoption of the method had to wait upon the invention of suitable steel alloys, but by 1920 it was in use all over the world. As an engineering device it cools the drill and clears the chips of rock most efficiently from the hole: happily it also suppresses dust (figs. 382, 383).



FIG. 380.—Rock Drill of 1902 with Water Spray
(*Holman, T. (1947), Brit. J. industr. Med., 4, 1*)

The Dryductor and the Mist Projector

Where wet drilling proves impossible or objectionable, foam-drilling equipment can be used. By means of compressed air a viscous foam is forced through the drill-

steel from a closed vessel. Although the foam traps the dust, the method has the defect that when the bubbles burst they may release dust particles. Since the tungsten-carbide bit need not be cooled and will last for 300 feet of drilling as against 10 feet in the case of steel-alloy drills, a new drill called the *Holman dryductor* has been perfected which sucks the dust back through the axial channel in the drill steel and ejects it into a dust collector (Holman, 1954). Near the rock face after shot-firing (fig. 384), a mist projector is used to allay dust (fig. 385). It is really an atomizer using compressed air and spraying a mixture of castor oil and water in which the proportion of oil to water is about 1 in 100. After shot-firing nobody may enter the area until eight hours have elapsed for the dust to settle (fig. 386).

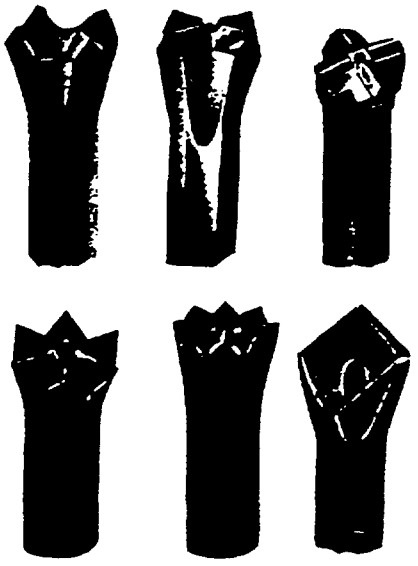

FIG. 381.—Various Patterns of Hollow Drill-steels. The most efficient Bit (top right) is of cemented Tungsten Carbide
(By courtesy of Mr. Trevor Holman)

The Hydro-blast for cleaning Castings

An apparatus used to clean certain types of casting, and the safest yet devised, is known as the hydro-blast. A high-velocity jet of sand and water is projected on to the castings, thereby removing moulding material, cores and scale. The velocity of the water as it leaves the hydro-blast gun is in excess of 3 miles per minute (fig. 387). It can be readily understood that this apparatus has greatly improved the cleaning of castings and much reduced the dust in the atmosphere of fettling shops where it has been installed. When it was first introduced it was hoped that it would take the burnt-on sand away from the castings and so avoid the use of dusty methods of fettling or cleaning. Unhappily, this hope has not been realized, because the cleaning has to be completed in the shotblast or wheelabrator chambers, or even with pneumatic tools.

Inefficiency of Wet Methods

It should be pointed out that water is not as efficient as good exhaust ventilation in controlling dust. The finest particles are not brought down by water; indeed, they may actually be held in suspension by droplets in the air. Investigations have shown that in wet grinding, for instance, there may be more dust present in the air than when the grinding is done dry under efficient exhaust ventilation. If the damp dust is not removed, it will

become dry and contaminate the air of the workroom. The addition to water of wetting agents which lower the surface tension of the droplets should theoretically help to engulf and control the dust particles more easily. They have been tried in some instances, but not with conspicuous



FIG. 382.—Wet Rock Drilling in a Cornish Tin Mine. Overhead Stoper using Drill with axial Water Feed. Note naked Flame of Candle fixed to his Helmet with Clay

(By courtesy of Mr. Treve Holman)

success. The use of water is therefore not the ideal method of dust control and may give a false sense of security.

Electrostatic Precipitation

Dust particles can be removed from a space containing air or gas by creating an electric field in it, thereby giving each particle a negative charge of electricity. The particles will then adhere to positively charged plates at the sides of the space. The method is used in Great Britain to clean certain chimney effluents and also coal gas, but it has not been applied to dusty



FIG. 383.—Overhead-stoping with a Compressed-air Drill operated by two Miners
(Drawing by Herman Giesen)



FIG. 384.—Shot-firer gathers the Wires attached to Detonators in Charges inserted into a dozen Holes drilled into the Rock Face



FIG. 385.—Mist Projector in operation in the Hæmatite Mines in Cumberland
(By courtesy of Dr. John Craw)



FIG. 386.—Eight hours after Shot-firing a Miner removes the Rock with a Mechanical Loader
(By courtesy of Anaconda Copper Mines, Inc.)

factory processes except occasionally in dust collectors. In the gold mines of the Transvaal it is used successfully on a fairly large scale to control the airborne dust. After a high initial capital outlay, the running costs are low.

Good Housekeeping

Factories should be kept in a tidy state, and the floors, walls and rafters should be cleaned regularly. Accumulations of dust on the rafters and



FIG. 387.—Workman cleaning a Turbine Casting. The Hydro-blast Gun is not turned on full otherwise he would be leaning forward to resist the Force of the Blast. Note Waterproof Clothing and Air-line to Helmet

ledges can be a source of airborne dust, especially when there is vibrating machinery in the workroom. Dust should not be allowed to accumulate on the floor of the workroom, because it is easily kicked up to become airborne. It should be removed daily by vacuum cleaning rather than by sweeping. Attention to these matters will reduce greatly the amount of dangerous dust in the air of the workroom.

(3) Personal Protection of the Worker

Where it is found impossible to control the dust completely, the worker must wear either a dust mask or an air-line breathing apparatus. A respirator must never be used as a lazy excuse for avoiding dust control.

Dust Masks

A dust mask is a filtering device for removing particulate impurities from inspired air. It does not remove gaseous impurities, and its use by

mistake as a gas mask could lead to a fatal result. The dust mask protects the nose and mouth. It is made of some light, durable material such as rubber, plastic or aluminium. The edge of the facepiece is lined with soft rubber and may be fitted with renewable cloth facings. The filter medium may be paper, wool, cotton, wool-asbestos, glass wool or various combinations of these substances. The filter is usually renewable (Drinker and Hatch, 1954). Dust respirators are most suitable for temporary use where there are short periods of exposure to dust, for example, when asbestos dust collectors are being cleaned out. It is almost impossible for a man to do a heavy job continuously when breathing against the resistance of the filter of the respirator.

Air-line Breathing Apparatus

If an air line has to be used, it is better to have it attached to a helmet as in the *breathing apparatus* illustrated in fig. 375. This apparatus can be used to protect people who have to go into a dangerous atmosphere, whether the contaminant is dust, fume or gas. A constant stream of warm clean air under slight positive pressure is blown into the top of the helmet, over the face and out at the sides. This type of helmet is used by shotblasters, and if it is maintained properly and not taken off before the worker is out of the dangerous atmosphere, it gives excellent protection.

(4) Medical Examination

Medical examination is used in the prophylaxis of silicosis in two ways: the initial examination of applicants for employment in an occupation with a silicosis risk, and the periodical medical examination of workers engaged in such occupations. The object of the initial examination is to prevent workmen whose respiratory physique is defective, through malformation or disease, from entering the dangerous industry. The periodical examinations enable silicosis to be detected at an early stage so that the workmen concerned can be transferred to other work. The examinations are also a means of discovering men with pulmonary tuberculosis who not only expose themselves to additional risk by remaining in the dusty occupation, but are possible sources of infection to their fellow workers who, following exposure to siliceous dust, have been rendered more liable to develop tuberculosis. In addition, these examinations provide cumulative evidence of the changes in the workmen's condition. But X-ray examination does not prevent a man from contracting silicosis. It does help to get him out of a dusty industry before the damage to his lungs has gone too far. X-ray and other examinations can also indicate in which part of the job the danger lies, and as such they are valuable because greater attention can be given to dust control in the dangerous job. It is not fair to the workers, however, to use X-ray surveys deliberately in order to save the time and trouble of the engineers whose business it is to control dust clouds wherever they arise.

Treatment of Silicosis

Since 1930 researches have been carried out upon the effects of various substances as antidotal dusts in the case of patients suffering from silicosis.

Antidotal Dusts in Experimental Animals

In 1932 Kettle showed that if silica dust particles were coated with iron oxide they did not cause fibrosis in the lungs of animals. Iron may therefore be regarded as an antidote to silica. No practical application has been made of this observation, but a great deal of attention has been paid to another antidotal dust—namely, aluminium. It was first tried in laboratory animals in Canada (Denny and others, 1939) and the United States of America (Gardner and others, 1944), and it was found that both aluminium powder and hydrated alumina gave the animals a measure of protection from the onset of silicosis. This observation has been confirmed in this country by King and his colleagues (1950). But it was also found by Gardner that hydrated alumina increased the susceptibility of the animals to tuberculosis. In the meantime it is not clear whether aluminium and its oxides damage the lungs or not.

Treatment of Patients with Aluminium Dust

Certain evidence suggests that aluminium is not altogether harmless and that, in some forms at least, it can cause fibrosis of the lungs (see p. 911). Meanwhile, aluminium is being used in some industries in an attempt to protect the workers against silicosis. Before beginning work they pass through an aluminium-dusting chamber and breathe a daily dose of the powder (fig. 388). It will be some years before any definite evidence of the value of the treatment can be obtained, and only then if adequate control groups are studied. It is difficult to believe that it is a good thing to put more dust into a lung which is already overloaded with dust. It is possible that aluminium could act as an antidote to those particles which have not gone deeply into the lungs, and in this respect the treatment might conceivably do good, but the method should be used with caution.

Inhalation of Spray charged with Calcium Salts

In Germany the effect of an electrically charged spray of calcium salts in solution is being tried as an antidote (Rosenthal, 1952). The droplets measure about 0·01 microns and it is claimed that they penetrate deeply into the lungs to combine with the silica and form calcium silicate which is said not to damage the lung tissues. There are now six calcium-inhalation chambers at the Ruhr coal mines, one in a porcelain works and another in an electric insulator factory. It is still too early to say whether or not the method will be successful (McLaughlin, 1953). In this work on the antidotal dusts there is a danger that, as a result of the attention given to them, efforts to control the dust clouds in the work-places might be relaxed. Generally, it is better to spend money in suppressing the dust at its source than in providing an antidote after it has got into the lungs.



FIG. 388.—Patients with Silicosis at Timmins, Ontario, breathing Aluminium Powder from an Experimental Treatment Chamber
(By courtesy of Dr. H. W. Jacob)

ASBESTOS AND OTHER FIBROUS SILICATES

The question as to whether silicates are toxic when inhaled has been much debated since Jones (1933) put forward the view that sericite, a potassium aluminium silicate, is the cause of much industrial silicosis. His view has received but little support. Certain silicates are noxious, and the chief of these is the group of fibrous mineral silicates known as asbestos. To a less extent talc produces a fibrous reaction in the lungs; sillimanite and glass wool seem to be harmless.

(*a*) Asbestos

Asbestos is the name given to a series of minerals composed of silicates of magnesium and iron. It is sometimes referred to as earth flax, stone flax or mountain cork. It is silky and fibrous in form, and this fibrous structure determines many of the characteristics of *asbestosis*, the disease caused by inhalation of the dust. Asbestosis is a more serious disease even than silicosis, but it is far less prevalent (Stewart, 1947).

History of Asbestos

On a small scale, asbestos was made into cloth at least 2,000 years ago. Herodotus (450 B.C.) relates how the Romans mined it in the Italian Alps and Ural Mountains, using it for enshrouding corpses before cremation to

permit of easy collection of the ashes for burial. They named it *amianthus*, meaning without miasma, undefiled or incorruptible. Pliny (A.D. 50) refers not only to difficulties in the weaving of asbestos, but also to the use of respirators to avoid the inhalation of dust. Strabo (30 B.C.) and Plutarch (A.D. 70) refer to the wicks of the lamps of the vestal virgins as *asbesta*, the unquenchable, inextinguishable or inconsumable. It is stated that Charlemagne possessed a tablecloth made of it, which was cleansed by passage through fire. However, although the occurrence of asbestos is widespread throughout the world, it is only since about 1890 that it has been used for industrial purposes. In 1880 the annual world production was only 500 tons, a figure which had risen to 1,300,000 tons by 1951.

Sources and Varieties

Canada produces 65 per cent of the world's asbestos. The remainder comes from Italy, Cyprus, Finland, Russia, South Africa, Australia, New Zealand, the United States of America and Peru. The term asbestos is a collective name applied in commerce to a variety of silicate minerals which differ from each other in chemical composition and physical properties, but resemble one another in their finely fibrous nature and flexibility. Their value depends on the facility with which they can be split into long and short flexible fibres, their resistance to acids and their insulating properties with respect to heat and electricity. Most of the asbestos in commerce is fibrous serpentine, especially chrysotile, $3MgO.2SiO_2.2H_2O$. This has the longest and strongest fibres and can be spun. Asbestos is derived also from the hornblende (amphibole) group of minerals, of which the most important are crocidolite and amosite. These consist of fibres too short for spinning, but more stable chemically and more resistant to acids and heat than chrysotile. The hornblende varieties contain less magnesium and usually more aluminium and iron, crocidolite being mainly a silicate of iron, $NaFe(SiO_3)_2FeSiO_3$. It is also known as *blue asbestos*, in allusion to its prevalent colour, and as *Cape blue*, because formerly it was mined only in Cape Province, South Africa. Amosite, $(MgFe)O.SiO_2$, which occurs in workable quantities only in the Union of South Africa, is named from the initial letters of Asbestos Mine of South Africa, the title of the mine where it was first worked. Other varieties are actinolite, $CaO.3(MgFe)O.4SiO_2$, and tremolite, $CaO.3MgO.4SiO_2$.

Uses

Capable of being spun into yarn and woven into cloth or of mixing with cement, rubber, resin, plastics or graphite, it provides the engineering, electrical, building, chemical and other trades with a variety of useful products. These include a wide range of insulation products, as well as flat and corrugated cement sheets for building work, and also roofing shingles, cements, tiles and fireproof paints. In brake linings and clutch facings the asbestos is usually reinforced with wire threads. Asbestos is made into acid-resisting filtering cloth, linings for chemical pans, valve packings,

gaskets, jointings and lagging material for steam pipes, and is used for coating the bulkheads of ships and marine piers. Long-fibred asbestos, which can be spun and woven into textiles, is used for fire-fighting suits, firemen's helmets, sheets and ropes, theatre safety curtains, boiler mattresses and for the coverings of welding rods where it acts as a flux.

Mining, Crushing and Disintegrating

Asbestos is mined both in open pits and by underground methods. After blasting, the longest asbestos fibres are separated from the mother rock by cobbers. These men are exposed to much dust because they do



FIG. 389.—Stripping an Asbestos-carding Machine before a modern type of Exhaust Ventilation was applied locally
(By courtesy of British Belting and Asbestos, Ltd.)

their work by hand with a cobbing hammer. Raw asbestos in crude and fibre form is imported into Great Britain and stored in sacks in open sheds. The sacks are carried to a rotary mill and opened, two or three at a time, by hand. This process is dusty. The material then passes by vacuum suction into a closed-in machine which delivers flocculent, soft, fibrous material, free from grit.

Carding and Card Cleaning

For the manufacture of yarn and cloth, cotton is often incorporated with the fibre before carding. The carding machine consists of large, fast-running cylinders, covered with innumerable minute wire points to ensure

that the fibres are laid parallel. From the end of this machine the material is received as a sliver or rope. Stripping the carding machines (fig. 389) and cleaning the cylinders with a wire brush is a very dusty occupation.

Spinning and Weaving

Spinning, doubling and weaving follow the process of carding. Dry weaving is usually practised, for wet weaving has the disadvantage of rusting certain fittings on the machinery. For non-textiles, such as millboards, a wet process is employed. Fibred asbestos is mixed with clay, cement and water, and run through a series of sedimentation tanks to secure the finest of the pulp. This passes along the machine as an emulsion, becoming drier and more doughy on its way, until it is eventually rolled out upon a cylinder. From this it is cut, removed and dried in the drying-room. Subsequent operations, such as sawing, grinding, turning and polishing, give rise to much dust. Insulating mattresses are made almost entirely by hand from lengths of asbestos cloth, and are filled with fibred asbestos by hand, scoop or shovel, levelled by beating and finished by sewing.

Historical Summary

The dangers of asbestos remained for long unsuspected. They came to light with a somewhat disconcerting suddenness. The first case of asbestosis known in Great Britain was observed in 1900 and described by Murray in 1907. The man had worked on a carding machine for fourteen years and was the last survivor of ten men who had worked in the card room, the others all having died about the age of thirty with lung disease. He died in Charing Cross Hospital at the age of thirty-four, and necropsy revealed extensive diffuse pulmonary fibrosis. Although the lungs were found to contain asbestosis bodies, a diagnosis was made of typical fibroid phthisis. Difficulties of certification delayed further action and cases of asbestosis continued to occur. Real attention was drawn to the disease by Cooke (1924). But it was the case published by Seiler in 1928 which seemed to establish an unequivocal relationship between asbestos and pulmonary fibrosis which precipitated the inquiry leading to the Report of Merewether and Price (1930). Reports of a heavy incidence of asbestosis have been made in some German factories, especially in Dresden (Krüger and others, 1931) and Berlin (Gerbis and Ucko, 1932).

Thirty Cases in One Factory

Between 1930 and 1934 thirty-seven workers in asbestos attended the Out-patient Department of the London Hospital; twenty-five were women and twelve were men. Of these, thirty were judged to be suffering from asbestosis. Seven of these died; and in four necropsy showed that the asbestosis was complicated by pulmonary tuberculosis. Many had worked in more than one department of the same factory, but the principal occupations in order of frequency were as follows: spinner, weaver, dis-

integrator, mixer, mattress maker, card cleaner, storekeeper, slab maker, presser and machinist. The average length of exposure before the onset of symptoms was seven years. Of the patients who died, one was a weaver who had been exposed for eighteen months and another a storekeeper who had been at work for nineteen years. In many cases there was a long interval between removal from exposure and the onset of symptoms: the longest such interval was eight years.

Symptoms

The presenting symptom was dyspnœa. This was early, conspicuous and always out of proportion to the signs found in the chest. It was nearly always associated with non-productive cough, as many as eighteen cases having little or no sputum. Hæmoptysis never occurred in the absence of pulmonary tuberculosis: it was found in three cases. In sixteen cases pain occurred between the shoulders, under the scapula or behind the sternum; it was never more than slight. In two cases a diagnosis of acute pleurisy had been made during the period of employment in asbestos, but in no case had a pleural effusion occurred. A history of warts was obtained in thirteen cases, of laryngitis in three and of conjunctivitis in two.

Physical Signs

The patients were usually well nourished. Clubbing of the fingers was present in six cases. Cyanosis was occasionally noticed, but was only slight. Examination of the chest showed limitation of the respiratory excursion on both sides. The percussion note was never seriously impaired, but slight dullness was sometimes found at the bases. The breath sounds were vesicular, and auscultation sometimes revealed scattered, medium, crepitant râles, often with a metallic tone, and situated especially at the bases. Added sounds were absent on repeated examination in ten cases. For obvious reasons the condition is not unilateral, and therefore displacement of the heart and other mediastinal structures was not found.

Asbestosis Bodies in the Sputum

Thirteen cases showed asbestosis bodies in the sputum. These are golden-yellow structures of elongated bead-like form, often with bulbous ends (Cooke and Hill, 1927). The appearance of fully formed bodies has been compared to beads on a necklace. The beads vary in size and make up an irregularly segmented body. These bodies have been found to vary in length from 20 to over 200 microns. An asbestos fibre forms the central core in each body (Gloyne, 1929). They are best seen with an oil immersion lens, and show up clearly without staining as golden-yellow structures (fig. 390). They can be stained by hæmatoxylin. The tubercle bacillus was found in the sputum of five of the above thirty cases, and four of these died.

Radiographic Appearances

The fibrosis in asbestosis is much more diffuse than that due to silica. In silicosis it occurs in the form of numerous more or less isolated foci, but



FIG. 390.—Asbestosis Bodies in the Sputum magnified 1,000 diameters

in asbestosis as a fine network throughout the lungs. In radiographs it is so fine as to resemble ground glass or fine cobweb (plate XVI). Homogeneous opacities of this nature are seen at both bases, and may later become more obvious as a fine punctate stippling. The inter-lobar pleura is usually thickened, and there is a shaggy outline to the heart shadow. Ultimately the fine punctate mottling can be observed to have spread beyond the limits of the lower zone to the middle and even the upper zone, but the apices usually remain free. This fine, diffuse, punctate mottling stands out in definite contrast to the nodulation seen in silicosis. In fact, in advanced asbestosis the degree of shadowing present would have little significance if seen in silicosis (Timmermans, 1931).

Prognosis

Broadly speaking, the length of time which may elapse between exposure to the dust and a fatal termination is only one-half of that in silicosis. In other words, in asbestosis the fibrosis develops more rapidly. With continued exposure to high concentrations of dust, the fibrosis may be

fully developed in seven years, and may cause death after about twelve years' exposure as compared with thirty-five years for all cases of silicosis. The shortest length of exposure to asbestos dust which has ultimately caused death is eighteen months. It must be remembered that the existence of a clinically recognizable degree of asbestos fibrosis in any individual is an additional adverse factor in the prognosis of any acute infection of the lungs such as pneumonia or bronchopneumonia.

Pathology of Asbestosis

At necropsy pleural adhesions are extensive and dense, and there are often thick subpleural plaques of fibrous tissue. Large areas of the lung are tough owing to the presence of fibrous tissue; emphysema is extensive but usually localized to the lower and apical parts of the lungs. There are two views regarding the mode of development of asbestosis. According to the one, the action of the dust is chemical; the other view supposes that the asbestos fibres act mechanically. Experiments carried out at the Saranac laboratories do not support the chemical theory, since aluminium hydroxide which neutralizes the toxic action of quartz does not prevent asbestosis. Moreover, asbestos fibres shorter than 20 microns were found to be relatively innocuous, whereas if the action were chemical an increase in potency would have been expected with reduction in particle size below 5 microns, as in the case of silica. A comparison of the effect of asbestos fibres 15 microns long with those 2·5 microns long (King and others, 1946) showed that rabbits receiving the long fibres developed a nodular reticulin formation comparable with experimental silicotic nodules; animals receiving the short fibres developed a diffuse interstitial reticulin formation.

Effect of the Size of the Fibre

The localization of the asbestos lesions appeared to depend on the size of the fibres; with long fibres, too big for removal by phagocytes, the reaction resulted in the formation of connective tissue within the alveoli; with shorter fibres capable of being taken into the alveolar walls, there was diffuse interstitial fibrosis. These workers suggest that if the fibres were shorter than 2·5 microns they would be completely removed from the alveolar walls and fail to produce fibrosis. Johnstone (1948) concluded from clinical investigation that workers exposed to high concentrations of extremely fine asbestos dust did not suffer from asbestosis. The Saranac workers conclude that asbestosis is caused by mechanical irritation from the asbestos fibres during the movements of respiration, and that this peculiarity is related to the flexibility of the fibres not possessed by other foreign bodies. The view that movement of the lungs is a factor is supported by the absence of reaction to asbestos in organs other than the lung. Wyers (1949) points out that if the fibrous character is absent, as in the case of serpentine, the chemical equivalent of asbestos, the dust is inert.

Incidence of Associated Pulmonary Tuberculosis

Precise information as to the incidence of pulmonary tuberculosis amongst asbestos workers cannot be obtained, but the Factory Department figures for 1935 suggest a less close association between asbestosis and tuberculosis than is the case with silicosis and tuberculosis. Thus among 702 deaths from silicosis the disease was accompanied by tuberculosis in 56 per cent, whereas among eighty-two deaths from asbestosis only 36 per cent showed associated tuberculosis.

Association with Carcinoma of the Lung

In an analysis of 235 cases of asbestosis by the Factory Department (1947), cancer of the lung or pleura was found to occur in thirty-one (13·2 per cent, average age 52·1 years). These figures were contrasted with the much lower incidence of cancer of the lung in 6,884 cases of silicosis, of whom 1·32 per cent had carcinoma (average age 59·4 years). Usually the carcinoma appears many years after exposure to asbestos has ceased (see case 38, p. 882).

Preventive Treatment

In asbestos mining wet rock drilling is imperative. In Great Britain legislation has been effective in controlling the disease. In factories and textile mills the principle of locally applied exhaust ventilation must be enforced to prevent the escape of asbestos dust into the air of any room in which work is done. Hand cleaning of the cylinders of carding machines is prohibited while any other person is present, nor may it be done by hand strickles or other hand tools. Those cleaning cylinders, filling insulating mattresses and entering chambers containing loose asbestos should be provided with an efficient apparatus by means of which they can breathe air free from dust. Suppression of dust in the cleaning of carding machines is best ensured by the use of a revolving brush fitted with a cover and connected to a portable vacuum cleaner (fig. 391). By the use of closed-in machines, handling should be reduced to a minimum. In one factory, spinning frames have been totally enclosed in *perspex* with an ingenious device which permits easy access to bobbins (Wyers, 1949). Education of the worker must aim at a high standard of good housekeeping.

Case 37. *Asbestos spinner—exposure to asbestos dust for five years—asbestosis.*

A. R., a woman, aged 27, had worked from 1924 to 1929 in the spinning department of an asbestos works where the protection from dust was poor. In 1928 she suffered from a dry cough, and in 1930 from shortness of breath and occasional pain in the right side of the chest on breathing.

On examination: five warts on fingers, the largest 3 by 1 mm. No clubbing of fingers. Dyspnœa, but no cyanosis. Apex beat not displaced, and heart sounds normal. There was diminished movement of the base of the right lung, with slight dullness. diminished breath

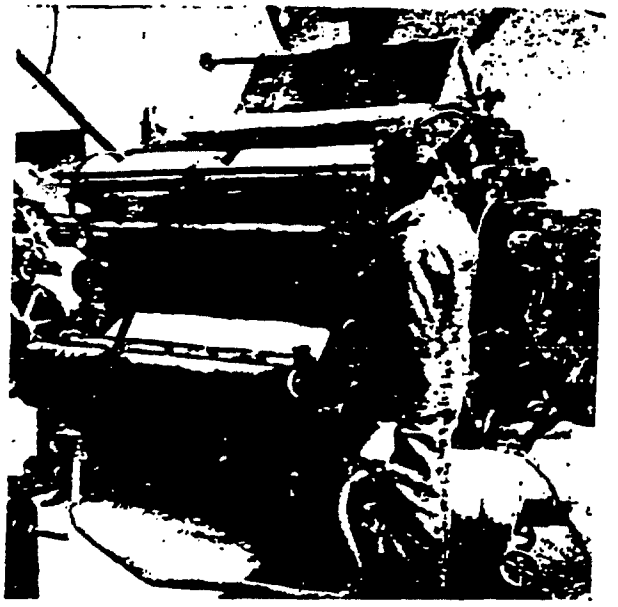

FIG. 391.—Portable Vacuum Apparatus for cleaning Asbestos Carding Machine. Note the Revolving Brush fitted with a Cover and connected to a Vacuum Cleaner
(*Report on Conferences concerning Methods for Suppressing Dust in Asbestos Textile Factories, 1931*)

sounds and extensive creaking pleuritic sounds. A few crepitations at both bases. No sputum. X-ray showed the heart to be normally situated, and there were fine homogeneous opacities resembling ground glass at the bases of both lungs. In 1935 the shortness of breath was worse; there was a dry cough. On examination there were no warts on the fingers, and the physical signs in the chest were unchanged.

Case 38. *Asbestos carder and weaver—exposure to asbestos dust for three years—latent interval of twenty-five years—death from compression paraplegia, secondary carcinoma of spine; primary carcinoma of lung; asbestosis.*

M. C., a married woman, aged 43, L.H. Reg. No. 41149/1947.

1919–22: between the ages of fifteen and eighteen she worked as carder and weaver in an asbestos factory. It is not known whether she cleaned the carding machines with a steel handbrush, but this method was in use at the time. She gave up the work owing to shortness of breath.

25 years: she had frequent brief attacks of breathlessness sometimes with cough but was not disabled.

16 years: at the age of twenty-seven she married, and later had one miscarriage but no further pregnancy. She remained active, doing all the duties of a housewife.

Four months: sudden onset of aching pain in the back of the neck without any apparent precipitating factor. The pain radiated to both shoulders and to both sides of the chest, was continuous and severe enough to interfere with sleep. It remained for some time practically unchanged, but had gradually increased in severity during six weeks.

Three weeks: she felt unsteady on getting out of bed in the morning and since then she had become progressively weaker and more unsteady on her legs. There were occasional involuntary movements of the legs, usually flexion of the knees with plantar flexion of the ankles. Occasional paræsthesiæ of the legs. Hesitancy of micturition. Bowels open regularly.

On examination: temperature 99° F. Pulse 112. Respiration 22. A nervous distressed woman, in considerable pain. Furred moist tongue. No pallor. No enlarged lymph glands or œdema. Regular pulse. Apex beat 1 inch lateral to mid-clavicular line. Blood-pressure 120/90. Heart sounds normal. There was some impairment of resonance and diminution of breath sounds with coarse crepitations at the bases of both lungs. The abdomen, including digital examination of rectum and vagina, showed nothing abnormal. Nervous system: an anxious but co-operative lady. Cranial nerves normal. Upper limbs normal. Lower limbs—approximately symmetrical spastic paralysis with brisk reflexes, ankle clonus and extensor plantar responses. Abdominal reflexes absent, and some weakness of lower trunk musculature. Loss of sensation to pin-prick, postural sense and vibration up to the level of D6.

Investigations: catheter urine: trace of albumin; deposit; calcium oxalate crystals; culture sterile. Blood count: hæmoglobin 89 per cent, red blood cells 4,720,000 per c.mm., colour index 0·94, platelets 255,000 per c.mm., white blood cells and differential count normal. Cerebrospinal fluid: clear, colourless fluid, under normal pressure, rising unevenly on jugular compression to 300 mm., and falling similarly; no increase in cells, protein 500 mgm. per cent, Lange 111,211, Wassermann reaction negative. X-ray of chest: lung markings heavy throughout, excessive fine interlacing shadows at bases, no nodulation. X-ray of spine—the body of D4 is smaller than its neighbours, especially in vertical height, and is mottled. The pedicles are not visible.

Progress: while in hospital the pain continued without much alteration. In the middle of the second week she suddenly developed severe pain in the left chest, became dyspnœic, collapsed, pale and cyanosed. Some relief was obtained from morphine and oxygen. This was followed by fever up to 101·8° F., with rapid respiration rate and two further attacks of chest pain. Electrocardiogram at this time showed absent Q and inverted T in lead 3 with right axis deviation, the rate being 180. She became steadily weaker and died 2½ weeks after admission.

Necropsy: Professor Dorothy Russell made the necropsy (L.H.P.M. 282/1947), of which the following is a summary.

Secondary carcinoma of thoracic vertebræ invading epidural space. Primary carcinoma of lung. Asbestosis.

Dark greyish-black reticular fibrosis throughout both lungs, especially lower lobes and right middle lobe, with conspicuous subpleural fibrosis (0·5 cm. deep). Area (3 × 3 × 2 cm.) of pale grey carcinoma beneath pleura of mesial aspect of left upper lobe. Clear yellow pleural effusion, left (3 oz.). A few fibrous pleural adhesions over posterior surface of left lung and mesial aspect of right upper lobe. Secondary carcinoma of hilar glands, most marked right, and one in superior mediastinum. Secondary carcinoma of greater part of bodies of 2nd to 4th thoracic vertebræ. Collar of growth encircling dura over 4th and 5th thoracic segments. Patchy softening of spinal cord at this level. Nodule (1 cm. diam.) of secondary carcinoma in mesial surface of left parietal cortex. Secondary carcinoma in supraclavicular glands. Swollen red pulp in spleen. A well-nourished, edentulous woman.

Microscopic. Lungs: conspicuous anastomosing bands of dense fibrosis containing numerous asbestosis bodies, often in clumps, and associated with foreign body giant-cells. Similar zone beneath pleura of lower lobes; less marked and interrupted over middle lobe. Congestion and emphysema of residual lung tissue; many macrophages and occasional asbestosis bodies in alveolar spaces. *Left upper lobe:* similar changes, together with ill-defined, infiltrating tubular, columnar-celled and polygonal trabecular carcinoma beneath mesial pleura, apparently arising from bronchus. *Gland from right hilum:* focus of secondary polygonal-celled carcinoma; anthracosis and a few collections of asbestosis bodies. *Right and left supraclavicular glands:* extensive secondary carcinoma. *Fifth thoracic segment with dura:* extensive secondary carcinoma of epidural tissues, not penetrating dura. Permeation of many veins. Engorgement of pial veins. Ill-defined wedges of ischæmic softening in both lateral columns of spinal cord extending into crossed pyramidal tracts, and a few smaller similar foci in anterior and posterior columns. *Thoracic vertebræ 2–3 with intervertebral disc:* extensive osteoclastic and osteoplastic secondary carcinoma of spongiosa and periosteum. Considerable necrosis of medullary tissue. *Left parietal cerebrum:* nodule of secondary carcinoma in superficial cortex. *Spleen:* slight myeloid reaction in pulp.

(*b*) Talc

Talc is a hydrated magnesium silicate, $3MgO.4SiO_2.H_2O$. The purest deposits occurs in association with dolomite and magnesian limestone, where the talc owes its origin to the hydration of the magnesium silicate minerals which were formed during the earlier metamorphism of the carbonate

rocks. Talc mining is carried out mainly in the United States of America and Manchuria. The largest deposit in Europe is on the northern slope of the Pyrenees.

Uses

Talc is the first mineral man encounters upon entering the world, for it is the main constituent of talcum powder and of toilet powders in general. It is soft and has a greasy feel, and on that account the massive impure substance has been called *steatite* or *soapstone*. This is the *French chalk* used by tailors for marking cloth. Perhaps 90 per cent of all the talc produced is marketed in the form of an impalpable powder. Nearly half the total output is used in the manufacture of paint and distemper. In ceramics, talc is used for the manufacture of tiles, electrical porcelain, table-ware and refractories. Nearly 25 per cent of talc production goes into the manufacture of paper in which the mineral serves for filling and glazing better than kaolin. It is widely used as a dusting agent for rubber tyres, rubber gloves and for packing parachutes to prevent rotting from moisture.

Pneumoconiosis in Talc Mining and Milling

In New York State, disabling pneumoconiosis occurred in 14·5 per cent of the workers in the talc mining and milling industry. The dust contained fine, straight, needle-like fibres. The talc fibrosis was associated with an increased susceptibility to tuberculosis (Siegal and others, 1943). Marked radiological changes were found in nine out of thirty-four men employed in a talc mill in Norway for periods lasting from seven to twenty-four years; there was less disablement than would have been expected from the X-ray appearances (Bruusgaard and Skjelbred-Knudsen, 1949).

A Case proved by Necropsy

The first case of this type proved by necropsy to occur in Great Britain was published by McLaughlin and others in 1949. The man was fifty-one years of age and had worked in a rubber-tyre factory since he left school at fourteen years of age. The only industrial dust to which he was exposed was that of talc. For two years he had complained of increasing dyspnœa, which was partly due to heart failure arising from rheumatic carditis with aortic incompetence. The X-ray film showed nodular shadows distributed over both lung fields. At necropsy, throughout the lungs there were grey nodules which had coalesced in some places, especially in the lower lobes. Microscopically the nodules showed whorling, but this had not the typical appearance of silicosis. Within the nodules were fibre-like structures from 10 to 90 microns in length, arranged singly and in clumps; the non-fibrous particles apparently had been eliminated. Some of the fibre-like structures were similar to asbestos bodies, and the appearances suggested that talc pneumoconiosis and asbestosis are similar diseases and that the former may be caused only by the fibrous varieties of talc. Asbestos is more actively fibrogenic than talc, and this might be due to the higher proportion of fibres in asbestos.

(*c*) Sericite

Sericite is of geological rather than commercial importance. It is a variety of the mica known as muscovite, $K_2O.3Al_2O_3.6SiO_2.2H_2O$. It occurs as minute shreds or in very thin veins and is a secondary mineral formed by hydrothermal alteration of silicates such as the felspars. It forms acicular crystals and has been suggested as a cause of pulmonary disease by Mavrogordato (1926), Badham (1927) and Jones (1933). Since it occurs in many rocks in association with quartz it is not surprising that it has been found in the lungs of miners with silicosis. It is a residual material only and has not by itself been found to cause progressive fibrosis of the lungs, possibly because the fibrous particles are too small to have the effect caused by asbestos.

(*d*) Sillimanite

Until about 1920 the minerals of the sillimanite group were almost unknown outside the geological world, but now they are in great demand for making high-grade refractories. They are all aluminium silicates capable of withstanding high temperatures. Andalusite comes from California and the Transvaal; kyanite and sillimanite are mined in India. Porcelain made from them is characterized by its high melting-point, low coefficient of expansion, resistance to shock and low electrical conductivity, properties which render the porcelain eminently suitable for sparking-plugs, laboratory ware, thermo-couple tubing and special refractory bricks used in electric and forging furnaces and cement kilns.

Sillimanite Pneumoconiosis

Sillimanite presents a fibrous crystalline form in the raw state and when calcined, but, unlike asbestos, the fibrous form is lost when it is crushed. When fused in a furnace at 1,545° C., sillimanite is converted partly or wholly into mullite. Gärtner (1947) suggested that this fact may be related to the histological changes, with the formation of small irregular nodules, found in a portion of lung of a furnace worker, and reported by Gärtner and van Marwyck (1947). In the examination of sixteen workers exposed to dust of calcined sillimanite, Henry and Middleton (1936) found radiographic changes but no disability, and no signs or symptoms which could be related to the dust exposure.

(*e*) Glass Wool

Fibrous glass, *fibre glass* or *glass wool*, is a fibrous silicate manufactured in industry. In his experiments Gardner (1942) found that owing to the high electrostatic charge on the fibres it was impossible to maintain an appreciable concentration in the atmosphere, and few fibres, 3 microns or less in diameter and 10 to 12 microns in length, penetrated to the alveoli. Unlike asbestos fibres, glass fibres are not retained in the respiratory bronchioles and they do not produce fibrosis. Gardner considered that this

material lacks the basic physico-chemical properties required to produce fibrosis.

Non-fibrous Silicates

In the absence of quartz and of fibrous structure, silicate minerals appear to produce radiographic changes accompanied by symptoms which are less severe than might be expected from the radiographic signs. The amount of change depends on the concentration of dust and the period of exposure.

(*a*) Mica

Micas are complex aluminium silicates combined with either potassium, magnesium or iron. The more common are muscovite, $K_2O.3Al_2O_3.6SiO_2.2H_2O$, biotite, $(HK)_2(MgFe)_2Al_2(SiO_4)_3$, and phlogopite, $KH_2Mg_3Al(SiO_4)_3$.

Sources

Muscovite, or white mica, is transparent and colourless in thin sheets, and is distinguished by its highly perfect cleavage. It is not decomposed by sulphuric acid. The name is derived from Muscovy in Russia, where the mineral was in early use as window glass. In commercial deposits the crystals vary from a few inches to a foot or so in diameter and up to two inches in thickness. They are referred to as *books* of mica because they can be cleaved into sheets almost as easily as separating the pages of a book. India supplies 70 per cent of the world total of sheet mica. Large quantities of muscovite are mined in the United States of America, but only a small percentage is of sheet-quality, the rest being used as ground mica. Phlogopite, or amber mica, is mined in Canada and Madagascar. It is difficult to differentiate from muscovite, except that it is decomposed by sulphuric acid. Biotite varies in colour, being either brown, black or dark green, and even in thin sheets, in contrast to muscovite, it has a smoky colour and is spotted with impurities. For this reason it is rarely used in sheet form but is employed as ground mica.

Uses

Owing to its transparency and heat resistance, muscovite is used for the chimneys of oil-lamps, for peep-holes in stove doors, ovens, furnaces and optical lanterns, and is then often termed isinglass stone, after isinglass, which is a semi-transparent form of gelatin. No other mineral has better cleavage, flexibility or elasticity; it is possible to roll a sheet of muscovite a thousandth of an inch in thickness into a cylinder a quarter of an inch in diameter, and its elasticity would enable the sheet to flatten out again quite easily. Its high resistance to the passage of electricity is so great that no substitute, artificial or natural, has proved equally satisfactory, so that in the electrical industry it is as important as copper, and now ranks as one of the essential minerals in modern civilization.

Mica is employed in industry either as sheet or as powdered mica. It

has been estimated that 90 per cent of the production of sheet mica is used in the electrical industry. For condensers, as insulating material between the segments of commutators of generators and dynamos and for telephones, only the highest-quality mica is employed; and the increased demand since about 1925 for use in wireless apparatus and in motor-car and aeroplane engines has been spectacular. Phlogopite is more suitable than muscovite for separating the segments of a commutator, because it wears more evenly and at about the same rate as the metal bars; and the best grades of phlogopite, such as those produced in Canada, are used extensively in aeroplane sparking-plugs.

Mica which is unsuitable for use as sheets, and the parts of sheets trimmed off, is marketed as scrap or ground mica. About two-thirds of all such mica is utilized in the roofing trade as a backing for rolled asphalt roofing and shingles to prevent sticking, and to a lesser extent as a decorative surface for these materials. Some scrap mica is mixed with shellac and moulded for use in electrical insulation. Approximately 10 per cent of ground mica is employed to give lustre to decorated wallpaper and to produce the glistening *Jack Frost* effect on Christmas trees and Christmas cards. It is also used in paints, as a filler in rubber and as a dusting medium to prevent rubber tyres and rubber goods from adhering to the moulds in which they are shaped. Among miscellaneous uses for ground mica are: to give a finish to stucco and concrete; in the making of artificial stone slabs and blocks; for lubrication in axle grease and as a dry lubricant.

Mica Pneumoconiosis

In an examination of fifty-seven men exposed to mica dust for periods lasting from eighteen to forty-six years Dreesen and others (1940) found ten cases of pneumoconiosis. The dust was generated by grinding hand-sorted mica and contained practically no free silica. Radiological examination of 456 men who had spent more than half their working life in mica dust showed changes in the lungs in 9·4 per cent of cases, a figure almost double that in a comparable group with no exposure to mica. Vestal and others (1943) found similar X-ray changes in seventy-nine workers handling mica containing no free silica.

(b) Fuller's Earth

Fuller's earth is so named from its use at one time as an absorbent of the grease in wool and cloth, a process called *fulling*. It is a non-plastic clay which disintegrates to a fine powder when placed in water. The composition varies; it usually contains up to 90 per cent of montmorillonite, an aluminium silicate, together with traces of quartz.

Sources

It is found at Nutfield, Surrey, at Midford near Bath and at Bala, North Wales, and also in Saxony, in California and in eight other states of

the United States of America. It is either mined or dug in the open according to local circumstances. It is then ground to a fine powder and made into a slurry with water which is allowed to carry off the coarser particles. After consolidation the finer particles are dried, broken into lumps and packed for transport.

Uses

Although the use of fuller's earth for cleansing wool and cloth has greatly decreased, the demand for the material is greater than ever. It is now used for the filtration of mineral oils, for soap, cleansing preparations, pigments for wallpapers and toilet requisites.

Hazard to the Worker

Radiographic examination of five men exposed to the dust of fuller's earth for periods from four to thirty-nine years showed in three men, employed from four to nineteen years, films within normal limits; two others, employed thirty-five and thirty-nine years respectively, had signs of fine punctate mottling and some coalescence. One of these men died, at the age of fifty-six, after chest symptoms for some years and disablement for eighteen months. A necropsy was made by Gloyne (Campbell and Gloyne, 1942), who described the condition found as soft patchy pneumoconiosis without massive fibrosis and with none of the hard nodular character associated with silicosis. From microscopic examination of a portion of one of the lungs Belt described small black rubbery lesions, each of which appeared to represent the cut end of a small blood vessel or bronchus; there was no silicotic nodulation or massive fibrosis, and no tuberculosis (Middleton, 1940). The persistence of the pulmonary changes which follow impregnation by dust is shown in a case reported by Tonning (1949). This patient, when a young man, had been employed for about fifteen years in the same fuller's-earth works as that in which the above cases occurred; he had gone to Canada and, after various employments, with no exposure to dust, died there at the age of seventy-nine of pneumonia following an operation for cancer of the lip. The pathological changes found in the lungs resembled those found in Belt's English case (Middleton, 1940).

(c) Kaolin

China clay is so called because samples of it were originally obtained in the eighteenth century from China. Kaolin is a corruption of the Chinese word *Kau-ling*, the *high ridge* to the east of *King-te-chen* which for centuries has been the chief centre in China for the manufacture of pottery.

Sources

All the china-clay deposits of the world are the product of the decomposition of feldspars in granitic rocks. Feldspar is sodium or potassium aluminium silicate, $K_2O.Al_2O_3.6SiO_2$, and the potash is leached out by

downward percolating waters containing carbon dioxide and other substances, especially organic acids. The residue is china clay, $Al_2O_3.SiO_2.2H_2O$, an hydrated aluminium silicate, together with quartz, mica and other minerals from the granite. The china clays of Cornwall and Devonshire have the advantage of being pure white. Those of Czechoslovakia, Germany, Japan and the United States of America may have a yellowish tinge, and this colour has to be disguised by the addition of blue dyes which then interfere with the usefulness of subsequent products. It is not surprising, therefore, that of all the raw materials exported by Great Britain china clay ranks second in tonnage after coal.

Uses

In the manufacture of paper, china clay is the chief filler in the pulp, producing the body of the paper and the smooth surface. Newsprint contains 10 per cent of its weight of china clay. Used in ceramics, it is an essential constituent of the bodies and glazes of cups, saucers and plates. It is used as a filler in the rubber industry, for paints, as a stiffener for textile fabrics, for wall-plasters and in white Portland cement. For pharmaceutical purposes *Kaolinum Leve, B.P.* is used in the treatment of cholera and food poisoning to adsorb the toxins of the cholera vibrio and of Salmonella organisms. *Cataplasma Kaolini, B.P.* is often used in place of the linseed poultice.

The Worker remains in Good Health

Men quarrying and mining kaolin suffer no harm. Experiments have been carried out to test its toxicity. Two types of kaolin, from coal measures and china-clay deposits, were tested by intratracheal insufflation in rats by King, Harrison and Nagelschmidt (1948). The kaolins produced only a very mild reticulin reaction in the lungs; a specimen of ignited kaolin which contained amorphous silica in the form of a glass produced slightly more reticulinosis but not fibrous silicosis.

Pneumoconiosis of Coal Miners

We have seen (p. 853) that only a small percentage of coal miners develop classical silicosis, and that the mining occupations involving exposure to silica dust include ripping, brushing, driving a hard heading and driving a cross-measure drift. But there is a very much commoner disease of coal miners working at the coal face resulting from the inhalation of a mixed dust of coal together with a relatively small proportion of free silica. This pneumoconiosis of coal workers occurs in all countries where coal is mined, and it is found also in trimmers of coal cargoes in ships. In Great Britain it accounts for more deaths than do all other forms of pneumoconiosis combined, and since 1930 it has constituted the greatest medical and social problem in all industry. The distribution of the disease in Great Britain shows a preponderance in the South Wales coalfield.

Historical Summary

The history of lung diseases of coal miners in Great Britain from 1800 to 1952 has been reviewed in detail by Meiklejohn (1951, 1952). According to Bremner (1869) the earliest documents in which coal mining in Great Britain is mentioned are *The Saxon Chronicle of Peterborough* (852), and Bishop Pudsey's *Boldon Book* (1180). Newcastle coal is first alluded to in a charter granted to the inhabitants of that town in 1234, and the first mention of coal in Scotland occurs in a charter granted in 1291 to the Abbot and Convent of Dunfermline. We have seen (p. 52) how a shortage of wood about the year 1578 during the reign of Elizabeth I led to an expansion of coal mining, with the corresponding alteration of domestic hearths and furnace fires to suit sea coal.

Coal Miners' Consumptions and Phthisicks

It was not long before chest disease in coal miners gave rise to comment. In his *Fumifugium, or the inconvenience of the Aer and Smoak of London dissipated* (1661) John Evelyn wrote:

> Newcastle Cole as an expert Physician affirms, causeth consumptions, phthisicks and the indisposition of the lungs, not only by the suffocating aboundance of smoak, but also by its virulency, for all subterrany fuel hath a kind of virulent or arsenical vapour rising from it; which, as it speedily destroys those who dig it in the mines, so does it, by little and little, those who use it here above them.

Melanosis of the Lungs

Early in the nineteenth century the morbid anatomists of Paris described melanosis of the lungs. In his *Traité de L'Auscultation Médiate* (1819) Laennec clearly differentiates secondary melanotic carcinoma of the lungs and the black pulmonary matter deposited in coal-miners' lungs. But the first author to record in print that the disease arises out of employment in coal mines was Gregory (1831); his patient was a Dalkeith collier who had worked in the coalpits for twelve years. Various opinions were at that time held as to the causation of the disease, but in the main it was attributed either to the effects of blasting with gunpowder or to the inhalation of lamp-black or soot from the oil-lamps of the miners.

Black Spit and Phthisis Melanotica

In 1833 Marshall of Cambuslang near Glasgow published details of three men, all of whom had worked about fifty years as coal miners. In two of the patients the chest symptoms culminated in the expectoration of material as black as printers' ink. This *black spit* corresponded to a necropsy picture of lungs with jet-black nodules and cavities, the expectoration increasing in quantity as the destruction of the lung advanced. Marshall rejected all previous views as to the origin of the black material and stated that the inhalation of fine coal dust and its deposition in the substance of

the lung was the cause of *spurious melanosis* or *phthisis melanotica*. He considered that though there was an individual predisposition to the disease the main factor in its causation was the excessive dustiness of certain mines where there were hard dry seams and much pick work.

Anthracosis of Colliers

The word *anthracosis* was first used for the disease in colliers by Thomas Stratton of North Shields in 1837. It was at this time that the Thomsons of Edinburgh, father and son (1837, 1838), published their classic inquiry into the subject. The tubercle bacillus was unknown to them, but they clearly differentiated between phthisis and tuberculous phthisis and between those patients who died with anasarca and those with hectic fever. They had, of course, no X-rays, but their descriptions of pathological changes were minute and accurate and closely correlated with the clinical findings. They recognized the nodulation of the lungs of stone workers as opposed to the massive black agglomerations in the lungs of men who worked at the coal face. From necropsies on men killed in accidents they learned that lungs which were universally jet black with coal were compatible with health and full work.

Supposed Decline in the Disease

The generic name *pneumonokoniosis* was introduced by Zenker in 1866, and in 1874 Proust invented the shorter form *pneumoconiosis anthracosica*. The idea that coal dust may possess the property of hindering the development of pulmonary tuberculosis and of arresting its progress originated with Hirt (1871) of Breslau. Arlidge (1892) did not subscribe to this view; he believed that large fibrotic masses could break down without the presence of tuberculosis. Meanwhile, the discovery by Koch in 1882 of the tubercle bacillus had enabled medical men to distinguish with certainty between phthisis and tuberculous phthisis. As the nineteenth century drew to its close doctors all over Great Britain had satisfied themselves that anthracosis of colliers had for all practical purposes ceased to exist as a medical problem. This happy state of affairs, so they thought, had been achieved in fifty years by improved ventilation and improved sanitary conditions in coal mines together with shorter working hours. In the Milroy Lectures of 1915 Collis said—

> Miners' asthma, common though it used to be, has passed unobserved from our midst, and conjectures as to its character and causation are idle.

Mechanization in Coal Mines

Whether such opinions were correct or not we cannot now be sure, but mechanical methods of mining were soon to change people's views of the effects of coal dust inhaled into the lungs. Late in the nineteenth century many patents were granted for coal-cutting machines. In 1913 only 8 per cent of coal in Great Britain was machine-cut (fig. 392); by 1950 the



FIG. 392.—Miner hand-cleaning at the Coal Face
(By courtesy of the National Coal Board)



FIG. 393.—Chain Coal Cutter middle-cutting Coal-seam
(By courtesy of Professor I. C. F. Statham)

figures had increased to 80 per cent. The earliest machine worked like a circular saw and was called a *disc machine*. Since 1920 the most widely used type of machine is the *chain machine*. It depends for its action upon a traversing band saw represented by the chain which carries a series of cutter picks. In 1938 only 140 disc machines were in use in our coal mines, whereas 6,005 chain machines were operating. Since that year 95 per cent of the coal-cutters have been of the chain type (fig. 393). With the mechanical cutting and conveying of coal there was a great increase of dust, and by 1930 the lung diseases of coal miners were again compelling attention, particularly in South Wales.

John Scott Haldane (1860–1936)

By his important studies on the chemical regulation of respiration J. S. Haldane of Edinburgh gained an international reputation as a physiologist (fig. 394). He devised the standard apparatus and methods commonly used in gas analysis, gasometry of the blood and estimation of the basal rate of metabolism. In addition to his academic appointment as professor of physiology in Oxford, he acted as honorary director of the Mining Research Laboratory of the University of Birmingham. His investigations of factory ventilation and mine explosions, his work on the physics and physiology of deep diving and his discovery of hookworm in the Cornish tin mines (p. 657) have been of great moment to industrial hygiene. He was mainly responsible for the introduction of stone-dusting in coal mines as a means of controlling underground explosions. His work among metalliferous miners, particularly Cornish tin miners, led him to study the cell-storage and expulsion of dusts and the retention in the lungs of siliceous dusts. His work on silicosis in miners in metalliferous mines emphasized the particular liability of such men to die of tuberculosis.



FIG. 394.—John Scott Haldane, 1860–1936
(By courtesy of Mr. A. Shaw)

Ill-informed Complacency of Medical Men

But coal miners show a low death-rate from pulmonary tuberculosis, and this fact led to the acceptance of the view that silicosis was rare among them. The declaration by Haldane (1923) that—

> the inhalation of coal dust causes no danger to life but on the contrary gives even protection against the development of tuberculosis,

was largely responsible for the fact that mining engineers made no attempt to reduce the concentration of air-borne coal dust at a time when the introduction of mechanized methods of mining was causing an increased concentration of coal dust. Of this situation Fletcher (1948) has said—

> It must be admitted that medical men by their ill-informed complacency have a heavy load of responsibility to bear for the present high incidence of pneumoconiosis among coal miners.

Improved Methods of Chest Radiography

This period of complacency was closed chiefly by the increasing use of radiography in the diagnosis of chest disease. Cases of definite silicosis in coal miners who had carried out drilling in rock were first reported in South Wales in 1926 by Tattersall. Soon after this, in South Wales, tuberculosis officers of the Welsh National Memorial Association and private radiologists, in particular Harper (1934) in Ammanford, began to observe radiological pictures similar to silicosis in coal miners who would have been diagnosed clinically as cases of chronic bronchitis, and even in apparently healthy miners. In 1930 Cummins and Sladden in a classical paper published a detailed account of the pathology of such cases, showing that they had a kind of silicosis modified by what was regarded as an accumulation of coal dust due to lymphatic blockage by silicotic fibrosis. It was at this time that the technique of chest radiography became perfected as we know it today. In consequence, from 1930 onwards, knowledge of the dust diseases of the lungs was acquired at a greatly accelerated pace.

Exposure of Anthracite Miners in Spakes

Haldane (1935) maintained that coal miners developed bronchitis because of the excessive pulmonary ventilation occasioned by their strenuous work, and that consequently inhaled coal dust was not normally eliminated from their lungs and accumulated to cause a benign type of fibrosis which was not silicotic. Jones (1936) added the suggestion that bronchitis was especially common among anthracite miners because of the method used to bring them to the surface. Entry to and exit from many anthracite pits in South Wales is by drift, a slope negotiated in trams of open cars called *spakes*. In winter the ride is cold and long drawn out, giving maximum exposure to cold air of men who had become hot from heavy muscular work. Examined by X-rays, these men had lung shadows which in Johannesburg gold miners would have meant a sentence of death. And so with the advent of modern chest radiography Haldane found himself in violent collision with clinicians and radiologists.

Mounting Number of Anomalous Cases in South Wales

Meanwhile the Registrar-General's figures for the occupational mortality statistics for the years 1930–2 were published. They showed that

respiratory diseases were on the increase in coal miners; they were not benefiting from public health measures to the same extent as other groups. By 1935 certifications among coal miners by the Silicosis Medical Board focused attention on the progressively mounting number of cases occurring almost entirely in South Wales, especially in the anthracite area around Swansea. In 1936 Dr. Charles L. Sutherland, Chief Medical Officer of the Silicosis Medical Board, directed the attention of the Home Secretary and of the members of the Industrial Pulmonary Diseases Committee of the Medical Research Council to the fact that, in the previous three years, claims for compensation on account of silicosis by coal miners had increased by 70 per cent and these were almost entirely in South Wales. There was also a vast number of men whose cases were of anomalous types and who failed to obtain compensation.

Pathological Significance of Coal Dust

The situation was a most unhappy one and called for immediate scientific investigation. In 1936 the Medical Research Council undertook to investigate the problem of chronic pulmonary disease among coal miners, with particular reference to conditions in the South Wales coalfield. The inquiry which was set up extended over the years 1936–42 and consisted of a medical survey and pathological and environmental studies. Hart and Aslett (1942) confirmed that hard-heading workers driving through siliceous rock were subject to silicosis, but that they were few and provided only a small fraction of all the cases of lung disease. The bulk of cases occurred in colliers working at the coal face and to a less extent among other underground workers. Similar lung changes were found amongst surface workers on the screens and coal trimmers at the docks. It was this observation which underlined the importance of coal dust and not rock dust as the causative agent in pneumoconiosis of coal workers.

Reticulation the Earliest Sign in X-rays

According to Hart and Aslett (1942) the pathological changes in the lungs were reflected by radiographic appearances which they classified as (*a*) reticulation, (*b*) nodulation, (*c*) coalescent nodulation, (*d*) massive shadows and (*e*) multiple fluffy shadows. Nodulation and massive shadows were already well recognized as features of silicosis. They introduced the term *reticulation* to denote the radiographic appearances in which the lung fields were diffusely altered by delicate shadows like a net of very fine mesh. It was the earliest radiological sign of any abnormal changes in the lungs and was the commonest X-ray abnormality amongst the younger colliers in all mines whatever the type of coal.

Pneumoconiosis of Coal Workers

Even when reticulation was not accompanied by clinical disability, its presence still implied the risk of the later progression to consolidation with

its more serious accompaniments. Yet this condition could not rightly be designated silicosis. Thus Hart and Aslett had defined a new concept, which led to fresh legal definitions and demanded a new name. They suggested *pneumokoniosis of coal miners*, which the Committee on Industrial Pulmonary Disease later altered to *pneumokoniosis of coal workers* so as to include workers engaged in any operation underground in coal mines, in screen workers at collieries and in coal trimmers at docks.

The Cardiff Pneumoconiosis Research Unit

The next step in the investigation of this complex problem was the establishment in Cardiff in 1945 of the Pneumoconiosis Research Unit under the direction of Dr. Charles M. Fletcher. As a result of extensive researches Fletcher and his colleagues have made many useful contributions to the subject. One of many lines of investigation they have pursued concerns the study of disability in the miner by physiological methods. Apart from its intrinsic interest, this study has two important practical aspects derived from the fact that the degree of radiological abnormality in any case does not give an accurate indication of the severity of the associated respiratory disability: first, there is the need to provide some objective method of disability assessment for purposes of compensation and job selection; secondly, there is need for a means of diagnosis in certain cases in whose X-ray films focal emphysema may have obscured the underlying coal nodulation.

Radiographic Appearances

The Unit have agreed that it is impossible radiographically to identify either the presence or the extent of focal emphysema (see p. 900). They have concluded that the radiographic appearances may be divided into two main forms: simple pneumoconiosis and progressive massive fibrosis, corresponding with the pathological distinction made by Gough. Fletcher (1948) points out the confusion which has arisen from the use of the term *reticulation* and from the description of its appearance as being that of a network.

> Whatever may be the merits of the word *reticulation* to describe the characteristic pattern of early coal-miners' pneumoconiosis, there is no doubt that in practice its use may give rise to some misunderstanding. Hart and Aslett were forced by circumstances to use a portable X-ray set for their radiography and were therefore unable to achieve a high degree of definition in their films. In the Pneumoconiosis Research Unit we have been fortunate in having a fine-focus rotating anode tube, and we have used ordinary films and high-definition screens. With this technique we find that the most characteristic and almost universal change in the disease is a fine mottling whose main components are usually less than 1 mm. in diameter but may reach twice that size.

Pin-head Mottling

For this appearance we have, for the time being, used the term *pin-head mottling*. In the earliest stages it appears alongside and between exaggerated lung markings, but later it may become so intense that it obscures the normal markings completely. It is uncommon for this pin-head mottling to remain in a pure form. Frequently it is associated with a coarser mottling, through which it can still be distinguished, so that a film is often seen which appears nodular when viewed at a distance of a few feet but in which, on closer scrutiny, the nodules can be seen to be composed of aggregates of pin-head mottling. These granular nodules may be distinguished from a more homogeneous type of nodule which we see particularly in cases resembling classical silicosis with a history of heavy exposure to rock dust or in cases where we suspect a complicating tuberculosis.

Coarser Nodular Shadows

The commonest type of film is one in which there is a mixture of pin-head mottling with rather coarser nodular shadows of irregular shape and size. We are not inclined to use the word *network* to describe any of these appearances. We do not suggest that at present the term *reticulation* should be abandoned. It has proved useful in emphasizing the differences between the early stages of coal-miners' pneumoconiosis and those of classical silicosis, and it can only be supplanted by some better term as a result of wide agreement among workers in the field of pneumoconiosis. Moreover, it may be that, with further advances in radiographic technique, further modifications in terminology may become necessary.

Progressive Massive Fibrosis

This nodulation in due course may coalesce so that conglomerate shadows develop. These may be symmetrical, resembling *angel's wings*, or they may be massive, homogeneous, smooth, rounded shadows, well-defined, and commonly in the upper zones (plate XV). Following melanoptysis a central translucent area develops within a massive shadow indicating the presence of a cavity which may show a fluid level. They are accompanied by some degree of simple pneumoconiosis and by basal emphysema. The combination of simple pneumoconiosis with progressive massive fibrosis has been called *complicated pneumoconiosis*.

Clinical Varieties of the Disease

Coal-miners' pneumoconiosis presents three different clinical pictures. There is the pure coal-dust disease, non-progressive and benign unless focal emphysema develops, when it may be fatal. In this respect it is a more serious condition than the second disease, the classical non-infective silicosis of rock workers, in which focal emphysema is much less prominent.

In coal miners these two diseases are often indistinguishably combined. Then there is a third disease process, due to the interaction between some infective process, often tuberculosis, and the dust ensnared in the lung. The fundamental similarity of coal-miners' pneumoconiosis to pure silicosis is close. Fletcher (1948) believes the differences must be due, as Cummins (1934) maintained, to some action of the coal dust in the lung in subduing the toxic effects of tuberculosis both generally and locally, so that it is not clinically apparent, and so that the bacilli only occasionally escape from their fibrous prison to spread throughout the lung or appear in the sputum. The clinical differences between coal-miners' pneumoconiosis and silicosis are chiefly that the coal miner with his focal emphysema and his anthraco-silico-tuberculosis is more breathless than ill and his death is like that of a chronic bronchitic with emphysema and right heart failure. He has, as Cummins has emphasized, miners' dyspnœa or miners' asthma. On the other hand, the silicotic with silico-tuberculosis is more often ill as well as dyspnœic. He has miners' phthisis, and dies of clinically recognizable tuberculosis.

Clinical Course

The clinical course of the illness may cover many years. Sometimes the patient has no symptoms, although routine X-rays show abnormalities in the lungs. And then for a long time the only prominent symptom is dyspnœa. As the disease progresses this gets worse and cough may appear. *Melanoptysis*, the coughing up of coal-black sputum, sometimes occurs and continues on and off for years. The chest becomes barrel-shaped and there may be clubbing of the fingers. Hyper-resonance appears at the bases, and here the breath sounds become diminished. When bronchitis supervenes there will be coarse crepitations throughout the lungs. The vital capacity and maximum ventilatory capacity are below normal and the residual air is increased. The progress of the disease is often accompanied by a rising erythrocyte sedimentation-rate. The ultimate clinical picture depends upon whether right heart failure or silico-tuberculosis supervenes.

Pathology of Simple Pneumoconiosis

In the simple state the dust is found throughout the lungs in the form of black foci measuring up to about 5 millimetres in diameter. The dust collects around the small bronchioles and their accompanying arteries, having been brought there from the alveoli by phagocytes. For the most part the dust remains within these cells, the general shape of which is preserved, although the nuclei are obscured. Reticulin fibres develop in the foci of dust (Belt and Ferris, 1942). Fibrosis may not proceed beyond this stage or there may be the development of collagen. The latter does not develop to the same extent as in classical silicosis, nor does it have the concentric disposition, but runs irregularly or radially. The foci have a crenated edge with the processes extending into the unaffected tissues.

Pathology of Focal Emphysema

In and around the coal foci the air-spaces become dilated, giving a characteristic appearance described as focal emphysema. This emphysema has also been described in classical silicosis, but it is very much more severe in thecoal worker, and in the latter the emphysematous spaces may enlarge and become confluent (Gough, 1947, 1949). They can be demonstrated to perfection by the new technique of large tissue sections (fig. 395). The focal emphysema appears to be due to some mechanical disturbance within the secondary lobules of the lung, as in these units the emphysema starts

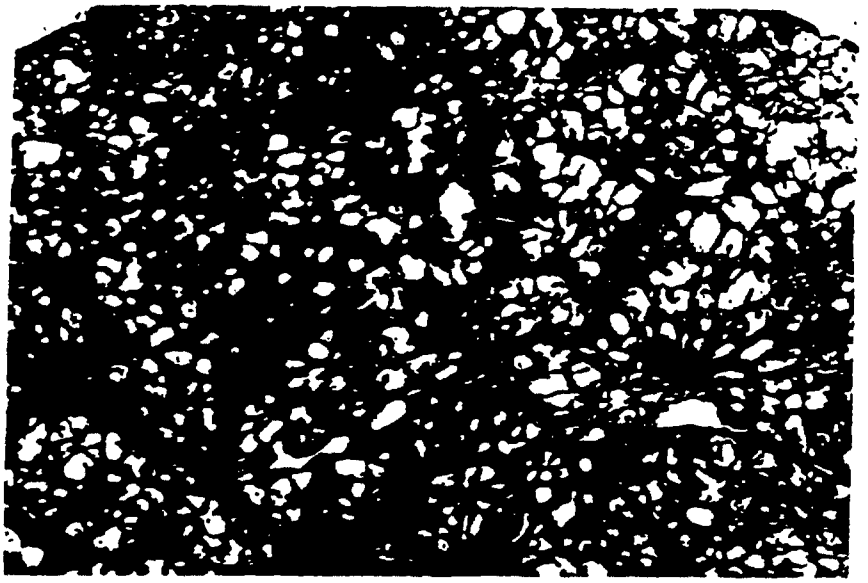

FIG. 395.—Large Tissue Section of Lung, natural size, showing the Multifocal Emphysema of Coal Workers' Pneumoconiosis

around the bronchioles and extends outward towards the interlobular septa. In focal emphysema there are no bullæ projecting from the surface of the lung, so that the condition can be distinguished from ordinary bullous emphysema. Williams (1944) observed that there was interference with the lumen of the terminal bronchioles, causing partial obstruction with trapping of air and consequent development of emphysema. Heppleston (1947) considers that the development of focal emphysema in coal workers can be explained by the accumulated dust acting mechanically, the foci of coal dust interfering with the function of the respiratory bronchioles. He also considers that shrinkage of the dust foci contributes to the development of surrounding emphysema.

Relationship to the Rank of Coal

King and Nagelschmidt (1945) regard coal-miners' pneumoconiosis as a form of silicosis and they contend that the small amount of quartz in the

dust is the pathogenic agent, although their chemical analysis of coal-miners' lungs does not give conclusive support to this view. King (1945) found that the dust from the Welsh coal mines has a low silica solubility, and Hicks and Nagelschmidt (1943) failed to find any marked relationship between the silica content of the dust in the various Welsh coal mines and the incidence of the disease. Reverting to the hypothesis of Hart and Aslett (1942) they suggest that the latter may be related to the rank of coal in the different mines; by *rank* is meant the amount of volatile material present. In the South Wales coalfield the character and rank of coal change gradually from north-west to south-east. The highest rank of anthracite with about 5 per cent of volatile matter is found in the north-west, and, proceeding towards the south-east, the volatile content steadily increases, the coal changing in character to lower-rank anthracite, steam coal, and finally in the south-eastern and eastern parts of the coalfield to bituminous coal with up to 36 per cent of volatile matter. The incidence of lung disease is higher in the anthracite and steam-coal areas than in the bituminous areas.

Harmful Effect of Coal not restricted to Silica Content

The opposing theories of mechanical and chemical action of the dust in coal-miners' lungs may be reconciled by accepting that each is in part correct, as is suggested by the following experiments. King and his co-workers (1948), treating rats by intratracheal injections of coal mixed with quartz, found that the mixtures produced more fibrosis than quartz alone, and they conclude that a small amount of quartz in the presence of a large amount of coal will bring about greater pathological results than the probable sum of the two components used separately. No difference was found between bituminous and anthracite coals, either when given alone or when mixed with quartz, and it is suggested that perhaps the coal dust blocks the lymphatic channels so that the quartz remains longer in the lung. These results support the conclusion that the harmful effect of coal is not restricted to its silica content. The practical result of such a view is that all dusts in the coal mine should be regarded as harmful and should be suppressed.

Identical Lung Disease in Coal Trimmers working in Ships

We have seen that Hart and Aslett found pneumoconiosis in coal trimmers who worked only in ships (fig. 396). In 1940 Gough investigated the pathology of this condition and demonstrated focal emphysema identical with that in men working at the coal face. This proves conclusively that the dust of commercial coal is noxious. In the past it has been argued that the respiratory disease in coal miners may be due to nitrogen-dioxide fumes from the explosives used in getting the coal, or to severe chilling when leaving the working-place on the long journeys underground against the incoming air. The occurrence of an identical form of the disease in men loading coal into ships shows that cold and nitrogen dioxide can be at the



FIG. 396.—Mechanical loading of a Collier in a Dockyard. The men who level the Cargo in the Holds of the Ship are called Coal Trimmers
(By courtesy of the Sunday Graphic)

most only contributory factors and that the essential cause is the inhalation of dust.

Infective Variety of Coal-workers' Pneumoconiosis

The condition described above as simple pneumoconiosis seems to be due to the action of dust alone, but superimposed upon it there is frequently a fibrosis which appears to be due to the combined action of tuberculosis and dust. This fibrosis occurs in the form of circumscribed masses most frequently in the upper and posterior parts of the lungs, sometimes unilateral, usually bilateral. The fibrous masses are black and may be several inches in diameter, and are firm and rubbery in consistency (fig. 397). The centres of the masses often contain black inky fluid in which are crystals of cholesterol; in some cases the fluid is expectorated, leaving ragged cavities. These cavities appear to be formed by necrosis due to obliterative endarteritis.

Massive Fibrosis due to Tuberculosis

In about 40 per cent of cases of massive fibrosis, tubercle bacilli can be demonstrated in the lesions post-mortem by culture or animal inoculation, and in those cases where no bacilli are found it is assumed that the infection has died out. In the past, tuberculosis has been regarded as of low incidence in coal workers, but the form of tuberculous reaction which occurs does not usually give rise to caseation or to the usual symptoms of open tubercu-

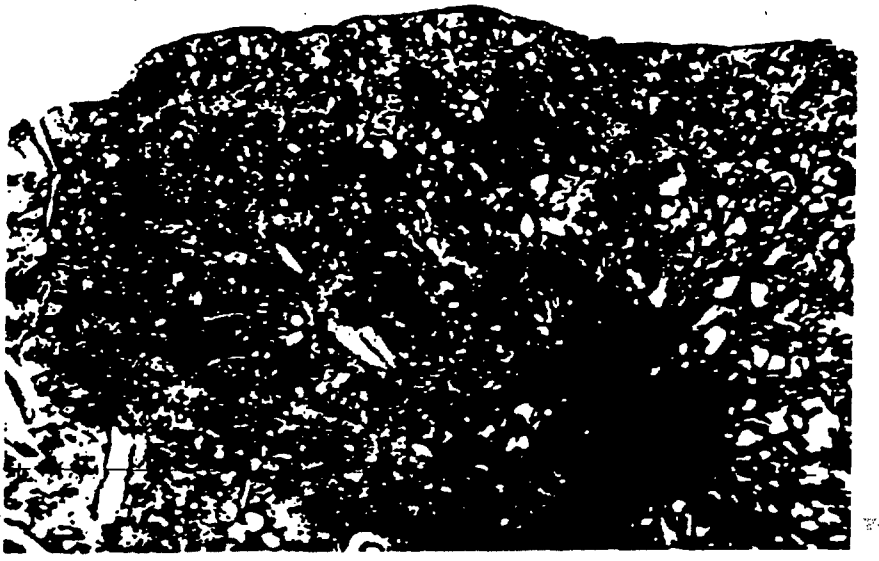

FIG. 397.—Large Tissue Section of Lung, natural size, showing the Black Areas of Massive Fibrosis in Coal Workers' Pneumoconiosis

losis. There is greater formation of fibrous tissue, the progress is very slow and the ill effects are principally on the pulmonary circulation, causing right heart hypertrophy and death from congestive heart failure. In a smaller percentage of cases the tuberculosis spreads more acutely, and in these there is caseation and bronchopneumonic spread of the infection. Another cause of death is pulmonary arterial thrombosis. This begins in a branch of the artery in relation to an area of massive fibrosis, and the thrombus spreads towards the hilum of the lung.

Hazard of Right Heart Failure

In summarizing the outstanding differences between the pathology of coal-workers' pneumoconiosis and classical silicosis, emphasis is placed first on the more severe focal emphysema in the coal-worker's lung, and secondly on the somewhat different effects of tuberculosis in the two conditions. In classical silicosis the open type of tuberculosis is a common terminal event, although heart failure from massive fibrosis also occurs; in coal workers, massive fibrosis leading to heart failure is the usual occurrence, whereas open tuberculosis is a less common complication.

Periodic Radiographic Examination of Coal Workers

The course of the disease under various environmental conditions is of considerable importance in prognosis and in deciding whether or not a patient may safely continue in his occupation. On the average, young men with quite advanced simple pneumoconiosis are not seriously disabled. Men with progressive massive fibrosis on the other hand are usually much

disabled and their disability increases both with age and the radiological degree of the progressive massive fibrosis. Simple pneumoconiosis, judged radiologically, does not progress as such in the absence of further inhalation of coal dust, but once massive fibrosis has started it is nearly always progressive whether or not dust exposure ceases. It has therefore been argued that if men could be removed from the danger before the critical stage is reached, their future health would be safeguarded. To fulfil this desirable purpose, a system of periodic radiographic examinations of coal workers was advocated (Fletcher, 1948; Cochrane and others, 1951).

Coal Mines Medical Service

Under the National Coal Board there is a comprehensive full-time occupational health service covering every coalpit in Great Britain (p. 189). This service is assisted by a staff of nurses (p. 189) and collaborates with H.M. Inspectors of Mines (p. 188), who ensure the observance of mining regulations. Under the *Coal Mines (Medical Examinations) General Regulations, 1952*, it is provided that all persons entering employment in or about coal mines shall be medically examined (p. 189).

Neglect of Airborne Coal Dust

After the passing of the *Coal Mines Act* in 1911 the use of some device to prevent the escape of rock dust into the air during drilling became obligatory, but no attempts were made to suppress coal dust. In 1938 the Royal Commission for Safety in Coal Mines discussed suppression of coal dust only as a means of reducing the danger from explosions. Research into the problem was, however, advised, and the British Coalowners' Research Associations and the Safety in Mines Research Board began to investigate the problem. In South Wales, as the results of the Medical Research Council's investigations came to be known, serious attention began to be paid to the prevention and suppression of coal dust underground, by the Mines Inspectorate, the Monmouthshire and South Wales Coal Owners' Association and by the Amalgamated Anthracite Company, who set up their own Silicosis Research Committee.

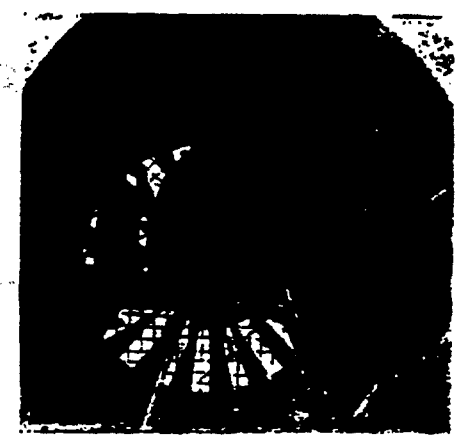

FIG. 398.—Centrifugal Fan in a Coal Mine. It can circulate up to 400,000 cubic feet of Air per minute

Methods of Dust Suppression

Six main methods are used to protect the coal miner against airborne dust.

(i) *Ventilation* is essential in reducing dust concentration (fig. 398). It has been particularly important in the South Wales anthracite mines, where, owing to the methods of working the coal in common use and owing to the rather haphazard development of the mines from shallow surface workings, poor ventilation was formerly one of the main reasons for the high dust concentrations.

(ii) *Wet Cutting* is achieved by directing two jets of water on the chain of the coal-cutter at the points where it enters and leaves the coal (fig. 399).

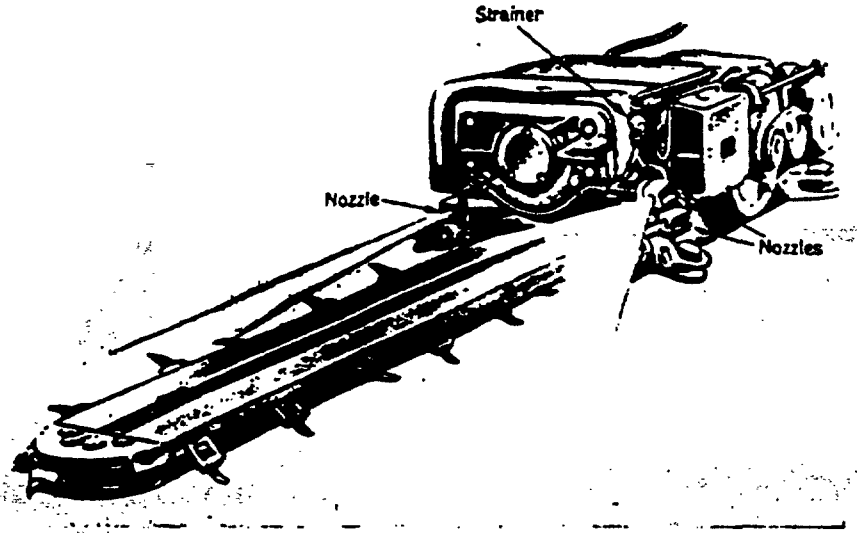

FIG. 399.—Jeffrey 35B Shortwall Mining Machine with Spray Equipment to allay Dust during undercutting
(By courtesy of Professor I. C. F. Statham)

The usual quantity of water is 5 gallons per yard of advance on the coal face. Of course, this treatment has a very beneficial effect during the subsequent loading on to conveyers. Dry cutting should be forbidden.

(iii) *Wet Drilling* is carried out with compressed-air drills, in which the water passes under pressure through a hollow drill-steel and is directed on to its cutting edge. Dry drilling should be forbidden (fig. 400).

(iv) *Water Infusion* is carried out on coal faces which are not undercut. At intervals of 12 feet along the face a series of 7-foot holes is drilled. A tube with an expansible rubber collar is inserted into each hole and 15 gallons of water at 100 lb. pressure are forced in, a small meter being used to indicate the quantity. The water spreads through the cleavage planes in the coal and wets the loose dust which is always present in the joints and seams.

(v) *Hand Spraying* is the wetting down with water of the coal face before the coal is pulled down and of the loose coal before it is loaded or

before it is transferred from one conveyer to another or from a conveyer into trams.

(vi) *Dust Masks.* There are certain mining methods in which the production of heavy dust clouds will be unavoidable—for instance, shot-firing. It will then be necessary for men to wear respirators. Unfortunately, no efficient respirator has yet been devised which men are willing to wear consistently. In the design of respirators, filtration efficiency has been given



FIG. 400.—Drilling Shot-holes at the Coal Face
(By courtesy of the National Coal Board)

priority over wearability, despite the obvious fact that an efficient mask in the pocket is of much less use than an inefficient mask on the face. In this field of investigation the biological approach is needed, giving first attention to the convenience of the man, if necessary at the expense of perfect filtration efficiency (Fletcher, 1948).

The rest of England is still behind South Wales in the application of these methods. Dust Prevention Committees have been set up in every division of the British coalfields and the fruit of their labours is anxiously awaited.

Need for fully trained Dust-suppression Officers

Fletcher (1948) emphasizes that it is the doctor's rôle not to ask over how many miles of coal face measures of dust prevention are officially being applied; he must ask, rather, how far dust levels are in fact being reduced.

A further rôle for the doctor as a human biologist is to insist that dust-suppression measures should not only be mechanically and physically effective but that the men who have to operate them should find them convenient and acceptable in use and that they should by a policy of education come fully to appreciate their need. Sprays that are ill-adjusted may wet the men more than the coal and will be turned off. Excessive use of water in any method may cause unpleasant working conditions. Men are always unwilling to accept measures for their own safety that are inconvenient and require care and attention. Perhaps the most urgent needs today in the field of dust suppression are that more attention should be paid to the human factor, that in every piece of mining machinery automatic dust-suppression devices should be incorporated by the manufacturers, and that there should be *fully trained* dust-suppression officers in every pit.

Non-pathogenic Inorganic Dusts

The dusts of the compounds of certain metals, among them calcium, iron, tin and barium, seem to be inert when inhaled. The resulting conditions are sometimes referred to as the ***benign pneumoconioses***. The particles, provided they are small enough to enter the alveoli, are engulfed in phagocytes, some of which are coughed up, while others are deposited in the aggregations of lymphoid tissue at the bifurcations of the bronchioles. They are capable of lying there for years without inducing any deposition of fibrous tissue or other reaction, but because they are relatively opaque to X-rays they may produce changes in the X-ray film which then must be distinguished from those of silicosis. The density of the X-ray shadows increases with the atomic weight of the element in the compound concerned as follows: calcium 40·08, iron 55·85, tin 118·70 and barium 137·36.

(*a*) Calcium

We have seen (p. 317) that the dusts of limestone, marble, lime, cement and gypsum are harmless to the lungs, producing at the most non-progressive arborescent shadows in X-rays of the chest but no symptoms or disability.

(*b*) Iron

In 1866 Zenker ascribed fibrosis of the lungs to the inhalation of iron dust and named the condition ***siderosis***. For many years this was considered to be the first of the pneumoconioses. But both the cases he had examined had pulmonary tuberculosis and such fibrosis as he found in the lungs may well have resulted from infection with the tubercle bacillus. We now know that inhalation of iron or iron oxide causes no fibrous reaction in the lung tissue. The disease at first called siderosis in knife grinders and needle finishers is, in fact, ***sidero-silicosis***, and is produced not by the iron in the

tissues, for this is inert, but by the free silica arising as a dust from the sandstone wheel used for grinding.

Welders' Siderosis or Iron-oxide Lung

Today opportunities for inhaling iron-oxide dust occur in many occupations, including welding and silver finishing. The heat of the electric arc or the oxy-acetylene torch melts and boils the metal being welded, iron coming off as a blue-grey fume and oxidizing almost immediately to iron oxide in minute particles. Welders who have spent years at this work, particularly if they have performed substantial amounts of welding inside boilers, tanks and other ill-ventilated spaces, may be found on X-ray examination to show reticulation or nodulation. But this does not necessarily mean fibrosis of the lungs nor, indeed, disease of any sort, and many a worker has passed through a very unhappy period after being told he was suffering from silicosis when, in fact, he was merely exhibiting a dusting or tattooing of his lungs with iron oxide.

The Worker remains in Good Health

The condition called *welders' siderosis* or *iron-oxide lung* was first described by Doig and McLaughlin in 1936. They emphasized that the X-ray changes occurred in men who were apparently in good health. Further experience by clinical examinations of some hundreds of welders has shown that the condition is benign and unaccompanied by symptoms, abnormal physical signs or diminished capacity for work. Some welders with dust reticulation have remained well and continued at work without disability up to fourteen years. The X-ray changes are not associated with dyspnœa or clinical evidence of fibrosis. Chest expansion is good and tolerance for exercise is normal. These findings have been confirmed by numerous investigators, not only in welders (Lanza, 1945) but also in other workers exposed to iron oxide, such as silver finishers (Barrie and Harding, 1947).

Non-progressive X-ray Changes

The X-ray picture may be identical with that of silicosis, and examination of a single X-ray film does not supply evidence of the inert nature of the condition. Serial X-rays taken over a period of years, particularly after exposure to welding has ceased, may, however, give supporting evidence. X-ray changes in silicosis are permanent; if they change it is for the worse, indicating a progression of the process, and such a change is unfortunately common. Improvement in the X-ray appearances has not been described nor is it to be expected in a disease characterized by the laying down of a considerable amount of fibrous tissue. On the other hand, Doig and McLaughlin (1948) have described two welders in whom the X-ray appearances changed for the better, and, indeed, in one case the picture returned to normal some years after giving up welding. Such an observation is only

compatible with a condition in which the dust lies amongst normal tissue and is not shut in by fibrous tissue or associated with other changes of a permanent nature.

No Fibrosis of Lungs at Necropsy

There is only one report in the literature of a post-mortem examination of a welder who showed X-ray changes of the dust-inhalation type during life (Enzer and Sander, 1938). This is illustrated by excellent photomicrographs showing no fibrosis round the collections of dust, which were found to give the Prussian-blue reaction. The man's death was not related to his occupation or his lung condition, but was the result of an accident. In successive papers McLaughlin and others (1945), Barrie and Harding (1947) and Harding (1948) describe the post-mortem findings in five silver finishers using as an abrasive *jewellers' rouge* which is finely divided ferric oxide, Fe_2O_3. The X-rays of these men had shown the typical changes of dust-deposition in the lungs. In the first four cases there was no fibrosis round the deposits of iron oxide. In the fifth case there was a minimal amount of fibrosis of the reticulation type, not at all like that produced by silica. Harding thinks that in this case individual susceptibility may have been of importance, but there is always the possibility that the iron dust at some period in the forty years of the man's working life may have contained silica or some other constituent capable of producing fibrosis. Indeed, it is not impossible that he may have used sand as the polishing medium at one time (Doig, 1949). Histological examination disclosed iron oxide and silver in the lungs; the former was in phagocytic cells along the course of the lymphatics and had produced no interstitial fibrosis. The silver dust was deposited in the elastic tissue of the alveolar walls and the small pulmonary vessels. In two cases there was marked emphysema, but the authors were not prepared to associate this with the silver impregnation of the alveolar walls.

Experimental and Statistical Evidence

Further evidence of the inert behaviour of iron oxide has been obtained from animal experiments. Harding, Grout and Lloyd Davies (1947) produced X-ray changes in the lungs of rats exposed to iron-oxide dust, but examination of the tissues showed no fibrotic or other reaction. Finally, there is statistical evidence. The Registrar-General's *Supplementary Report on Occupational Mortality*, 1931, shows that of the 11,542 welders in England and Wales in that year there were only 123 deaths compared with 161 expected on the basis of age-rates for all males. In welders in the Kaiser shipyards in Oregon, Collen (1947) describes the incidence of pneumonia in welders, showing that the incidence, death-rates and case fatality-rates were similar to those of all other shipyard workers and that there was no difference in the severity, the number of days required for treatment or the incidence of complications. It seems clear that iron oxide, inhaled into the lungs in amounts sufficient to give rise to changes in the

X-ray film, lies inert in the tissues for years without giving rise to pulmonary fibrosis or other permanent change.

(*c*) Tin

Tin is much more radio-opaque than iron and therefore deposits in the lungs cause particularly dense shadows, a condition known as *stannosis*. Pendergrass and Pryde (1948) reproduce an excellent X-ray of this type, the subject being a man aged forty-five years who had bagged tin oxide for fifteen years. He had no disability, the X-ray abnormality being discovered during a routine survey of all the work-people. Barták and Tomečka (1948) also consider that tin oxide, although producing radiographic changes, does not produce ill-effects clinically.

These authors in 1939 examined one worker in an enamel factory who consulted a doctor on account of asthma which had suddenly become worse. He was forty-nine years of age and for eighteen years had had charge of a furnace in which pure tin was burned to tin oxide. The atmosphere in which he worked was loaded with the dust of the oxide. There were no abnormal physical signs, but the X-ray picture of the lungs showed scattered dense punctate shadows. Of sixteen co-workers at the same factory, six showed the same X-ray picture. These men had been exposed to the dust of tin oxide for periods from six months to twenty-five years. The remaining ten workers had been employed at the furnace only occasionally; their real work was in the same room as the furnace, but 6 metres away from it. None of them had symptoms, and X-ray pictures of their chests were normal. A year later (1940) the patient with stannosis was found to have carcinoma of the stomach, of which he died in 1942.

(*d*) Barium

Baritosis is a type of benign pneumoconiosis first described by Arrigoni (1933) among the baryta miners of Valsassina in Italy. It occurs particularly in the grinders and packers, and results from the inhalation of finely ground particles of barium sulphate. In the men concerned there was no respiratory incapacity, and abnormal physical signs were slight or absent. The only evidence of its presence was the demonstration by X-rays of small sharply circumscribed nodules evenly distributed throughout the lung fields. In 1953 Pendergrass and Greening described a case of baritosis in a man who had worked for twenty years in the grinding department of a lithopone plant. Analysis of the ground material showed 97 per cent barium sulphate and 3 per cent silica. Analysis of the air-floated dust sample revealed barium 49·1 per cent and silica 2·1 per cent. Wearing a respirator, the man had been exposed for one and a half hours a day to dust of which the particle count was 6,000,000,000 particles per cubic foot of air. At the age of seventy-one, and twelve years after the detection of the baritosis, he had a sudden severe attack of retrosternal pain and died of a large infarct of the heart. At necropsy the lungs showed uniformly scattered black firm fibrous nodules from 1 to 3 millimetres in diameter. Barium sulphate was detected

in the lung by chemical analysis, spectrographic examination and X-ray diffraction. There were about twenty other workers exposed to the same hazard, but of the group only this man and one other showed changes in the lungs.

Pathogenic Inorganic Dusts

Various non-siliceous dusts are capable of producing pulmonary lesions, the most important of these being aluminium, beryllium and silicon carbide. We have seen already how manganese (p. 413), cadmium (p. 400) and vanadium (p. 440) can give rise to chemical pneumonitis.

(*a*) Aluminium

In spite of the fact that aluminium has been found to reduce the toxic action of silica, exposure to the inhalation of aluminium dust in Germany in the Second World War gave rise to a condition known as *aluminosis* or *aluminium lung*. Examination of material from the German cases shows that there was undoubtedly a pneumoconiosis, so that aluminium must be regarded as a toxic substance under certain conditions of exposure. In the Second World War powdered metallic aluminium was used extensively in making explosives, incendiary mixtures and paints. The powder was made by two processes: either it was blown, when the particles were covered with a thin film of alumina, or it was stamped, when they were covered with a thin film of stearine. The fact that German workers were affected in this industry rather than those of other countries may have been due to the omission of lubricants in the stamping process or perhaps to the increase in the amount of dust in the atmosphere in consequence of war conditions.

Symptoms and Signs

Describing the investigation of 700 workers in Germany, Goralewski (1939, 1940, 1941 and 1943) gives details of various types of disease attributed to aluminium. The aluminium powder to which the workers had been exposed contained about four milliards of particles per grain of dust, but the size was not stated. The development of the illness was rapid and appeared to bear no relation to the length of exposure to aluminium. They 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. In 78 per cent of the cases the sedimentation-rate was within normal limits, and the vital capacity was decreased in twenty-seven instances. 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.

A Colloidal Aluminium-hydroxide Complex

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 jagged outline from carbon particles. Jager and Jager (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 chlor ions are allowed to diffuse away, and if protein is also present it is co-precipitated round the partly dissolved aluminium particles. Jager and Jager think it is this complex which causes the lung changes. Koelsch (1942), however, believes that the disease was a consequence of the unsatisfactory ventilation of workrooms resulting from the blackout imposed by the war.

Inhalation of Aluminium Powder in the Treatment of Silicosis

Crombie, Blaisdell and McPherson (1944) investigated 125 workers employed in the Pittsburgh stamp mills of the Aluminum Company of America. The workers here had been exposed to aluminium dust for periods ranging from six to twenty-three years, and their health was found to be as good as 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 thirty-four 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 thirty minutes (fig. 388). Some of the men received 300 treatments, but the majority only 200. Out of the thirty-four cases thus treated, clinical improvement in nineteen was manifested by lessening or disappearance of shortness of breath, cough, pain in the chest and fatigue. In fifteen 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. Obviously this treatment cannot be regarded as a cure for silicosis, since it cannot restore lung tissue which has undergone fibrotic change, but Crombie and his colleagues believed that the inhalation of finely particulate aluminium powder is not harmful to human lungs. Denny, Robson and Irwin (1939) exposed eight rabbits to metallic aluminium dust of particle size below 5 microns 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 round them.

Exposure in Men grinding Duralumin Propellers

Hunter, Milton and Perry (1944) endeavoured to throw further light on this problem by studying the effect of dust on the lungs of grinders of duralumin aeroplane propellers. The grinders were exposed to a mixture of aluminium dust arising from the propellers, and alumina, Al_2O_3, from

the alundum grinding wheels (fig. 401). The average concentration of aluminium dust close to the operator's mouth was found to be 3 milligrams per cubic metre of air, of which the particle size in 1 milligram was between 2 and 7 microns, the remaining 2 milligrams consisting of larger particles. The alumina particles were mostly of less than 1 micron diameter.

Propeller Grinders remain Healthy

The health of these workers, as shown by their records for sick absence during the preceding year, was better than that of those in a machine shop

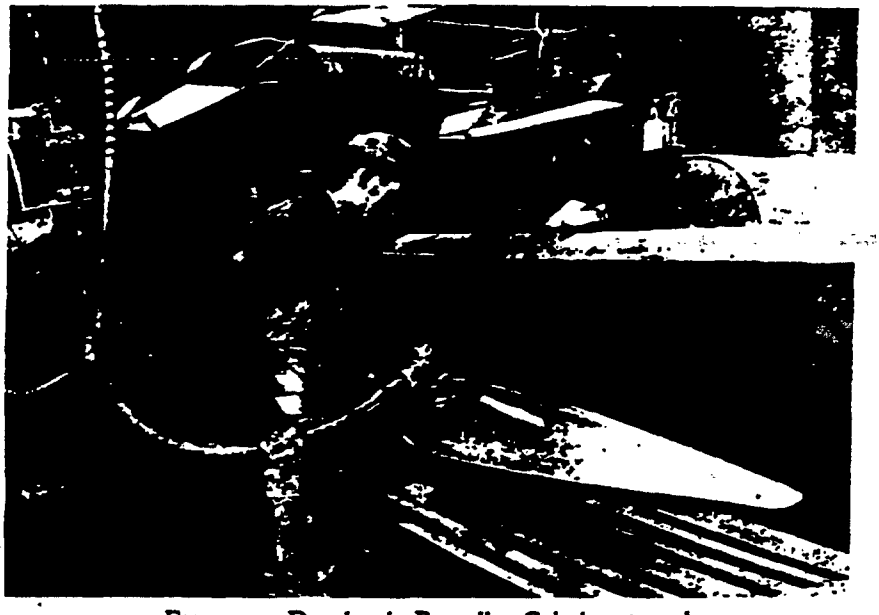

FIG. 401.—Duralumin Propeller Grinder at work
(By courtesy of the De Havilland Aircraft Co.)

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 ninety-two workers; in seven instances there were shadows in the peripheral part 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 forty, and thirty over forty: twenty-eight of them had been on the job for more than seven years, a further nine for more than five years, and thirty-one for more than two years. In the German cases the changes were not related to length of time on the job.

Pneumoconiosis in Bauxite Smelters (Shaver's Disease)

In connexion with aluminium, another form of pneumoconiosis was described in Canada (Shaver and Riddell, 1947; Shaver, 1948) as occurring in men exposed to the fumes arising in the process of making the abrasive *corundum*, Al_2O_3, a form of artificial emery. This is done in a works at Niagara by heating bauxite, an ore containing approximately 80 per cent Al_2O_3 and up to 7 per cent silica, with iron and coke in the electric arc at 2,000° C. The men affected were bin men, furnace feeders and overhead crane men. Between 1941 and 1943 visible fume came off from the kettles in which the bauxite was being fused. In 1943, production was accelerated, thus giving rise to a greater dose of fume per man. There were 13 deaths from pneumothorax. One man had 8 pneumothoraces and he is the only man seriously affected who still lives (Riddell, 1954). Silicotic nodules did not develop, but there was diffuse fibrosis and the formation of emphysematous bullæ which often ruptured and sometimes fused together to form larger cavities. The ætiology of the fibrosis of the lung is uncertain, as the noxious atmosphere contained 32·3 per cent silicon dioxide and 56 per cent aluminium oxide, the silica being almost entirely in amorphous form, Pratt (1950). The particles were from 0·02 to 0·5 microns in diameter, a particle size which has in the past been regarded as of little significance, Hatch (1950). In order to render silica insoluble by means of alumina the latter must be of a type which readily takes up water and becomes adsorbed on the silica particles. The alumina in the fume at the Niagara works was not of that type, Irwin (1950). Meanwhile, tuberculosis has not developed in the lungs of the survivors, and Riddell (1954) believes that the disease is aluminosis and not silicosis.

(*b*) Beryllium

The toxicity of beryllium and its compounds is discussed on p. 381. Since the inhalation of certain beryllium compounds can produce pulmonary lesions, these must be regarded as within the definition of pneumoconiosis. Two types of pulmonary disease are described. The first is acute chemical pneumonitis, with onset during exposure to the dust, and is characterized by cough, blood-stained sputum, retrosternal pain, dyspnœa, cyanosis, anorexia, loss of weight and prostration; resolution may occur within a few weeks or months or, rarely, chronic granulomatosis may follow. In the chronic type, anorexia, loss of weight and weakness are early symptoms; cough, extreme dyspnœa, œdema of extremities and cardiac failure develop; mortality is high, and many of those patients who survive suffer permanently from pulmonary distress.

Beryllium has been found in many organs and tissues, suggesting that the disease is systemic. It is of some interest to mention an analogy with silica; besides pulmonary lesions, certain compounds of beryllium cause chronic cutaneous lesions when implanted in the tissues (see p. 385); similarly, a condition called *pseudotuberculoma silicoticum* has been described by Shattock (1917), and by Faulds (1935), who described two such

cases, in which quartz-containing material was introduced into the skin and subcutaneous tissue by accidental injury.

(c) Silicon Carbide

Silicon carbide (carborundum) has been regarded as harmless since the classical experiments of Gardner (1923) showed that it produced no fibrosis of the lungs in experimental animals. Since carborundum is harder than quartz this work refutes rather convincingly the mechanical theory of silicosis—namely, that it is the hardness or sharpness of particles that causes damage in the lung. It would be ironical if it were now to be found that carborundum produces pneumoconiosis in man. Yet Smith and Perina (1948) found nodular shadows by X-ray examination in workers exposed to the dust of silicon carbide and alumina. Bruusgaard (1949) found definite radiographic changes in ten out of thirty-two workers exposed exclusively to the dust of silicon carbide. These authors suggest that the question of pathogenicity of this substance will need to be reconsidered, and further investigation, including post-mortem studies, will be necessary before the position can be clarified. It may be mentioned, however, that Gardner (1938) found in experimental animals that silicon carbide, together with tuberculous infection, could produce a fibrotic reaction, and this may perhaps explain the human cases.

Dusts of Vegetable Origin

Vegetable dusts, including pollen and fungus spores, are often large enough to be trapped in the nose and upper respiratory passages (see p. 838). Many of these produce asthma, hay fever and urticaria. Dusts of particle size small enough to reach the alveoli sometimes produce acute and chronic bronchitis, chronic emphysema, and even bronchopneumonia, fibroid lung and bronchiectasis. They include the dusts of bagasse, cotton, derris, flax, flour, grain, gum arabic, hay, hemp, jute, linseed, malt, nuts, paprika, seeds, sisal, straw, tea, tobacco and wood. The study of the effects of these substances has been neglected partly because the chronic bronchitis which may result from their prolonged inhalation is unaccompanied by any characteristic X-ray picture indicative of dust inhalation as is the case with pneumoconiosis due to mineral dusts. The chronic effects of vegetable dusts are thus less easily distinguished from non-occupational bronchitis.

Absence of Silica Hazard

In many cases it is not known whether the noxious agent lies in the vegetable matter itself—for instance, some protein, which is toxic—or whether it lies in the impurities. In the case of chronic disease silica has been considered, for free silica derived from soil is a universal contaminant of vegetable material. The amount present, however, is small, generally about 2 per cent or less, and particle size so great that it is most unlikely that silica plays any part in the production of lung disease from vegetable dust.

Mechanical and Allergic Effects

McNair and Middleton (1924) showed that the handling of grain in bulk causes liberation of an irritating dust consisting of the hairs from the *brush* of wheat and rye grains, and from the paleæ and awns of barley and oats; the dust also contains mould spores, débris and mineral matter. Another possible explanation is that dry vegetable matter on inhalation readily swells and this might result in small areas of atelectasis prone to become secondarily infected. It seems that vegetable dusts more than mineral dusts have a common type of action. Thus the dusts from grain, hemp, flax and cotton cause asthma or mill fever, or else result, after years of exposure, in chronic bronchitis and emphysema. In such cases the signs and symptoms are alike, no matter which dust is involved.

Bacteria and Fungi

The biological contaminants are more important. Vegetable matter, especially if it is rotting, may contain a high content of bacteria, and spores and mycelia of fungi, some of which are known to be pathogenic. Thus the mycological examination of cotton dust has revealed fungi of the genera *Alternaria*, *Mucor*, *Rhizopus*, *Fusarium*, *Sporotrichum*, *Aspergillus*, *Gladosporium* and *Penicillium*. Viable counts of such fungi may reach figures as high as 400 millions per gram (Furness and Maitland, 1952). When such masses of fungi exist in the dust inhaled, they naturally appear in the sputum, but this is no proof that the lung disease under consideration is of mycotic origin.

Fungus Allergens

In certain occupations inhalant allergic symptoms occur from sensitization to fungus spores. The men affected are dock workers unloading grain, lumbermen debarking trees infested with *Coniosporium corticola*, dealers in second-hand furniture, and gardeners handling tomato plants infested with *Cladosporium*. Sensitivity varies in an unpredictable way and the workmen involved may be isolated persons. New quantitative techniques are available for the study of this problem. Automatic spore traps are used in which a measured sample of air impinges on a sticky glass slide. By this method the number and different types of fungal spores can be measured over a given period of exposure.

(*a*) Cotton

Four conditions affecting the respiratory system are described in workers exposed to cotton dust in different parts of the world. These are mill fever, byssinosis, weavers' cough and an acute illness occurring among people who handle low-grade stained cotton. Of these, byssinosis is the most serious. It is principally a disease of Lancashire, where the cotton-spinning

industry is concentrated in towns like Oldham, Rochdale, Bury and Bolton. It is a progressive condition resulting in total disablement if exposure to cotton dust continues after symptoms first appear. No specific cause has been found for mill fever, byssinosis or weavers' cough. The endotoxin of *Aerobacter cloacæ* has been held responsible for the symptoms in an acute illness of workers handling contaminated cotton in the United States of America.

Processes in the Cotton Textile Industry

The processes used in the manufacture of cotton yarn and cloth are ginning, baling, bale breaking, bale opening, scutching, carding, spinning, weaving and finishing.

Cotton Ginning

Cotton ginning, or separation of the lint from the seed, is performed in the field in order to save unnecessary transport of the seed, which forms about two-thirds of the weight of the harvested crop. The modern ginneries in America are almost automatic, the whole operation being carried out by pneumatic suction so that the cotton is hardly touched by hand. From the gin the lint is carried pneumatically into a chamber where it is compressed into bales, which are then bound with steel strips.

Processes in the Cotton Mill

Cotton is imported into England chiefly from Egypt, India and the United States of America. Arrived in Lancashire, the bales are taken to the cotton mills. The functions of the processes in the mill are as follows:

(i) to divide up and clean the compressed cotton from the bale;

(ii) to form the fibres into a rope or sliver which can be attentuated until it is thin enough to form threads;

(iii) to comb out the fibres and set them parallel by drawing them between carding cylinders;

(iv) to insert sufficient twist into the material to make a firm thread;

(v) to finish and prepare the yarn;

(vi) to divide the warp or horizontal threads of the fabric so that the weft or cross-threads may be interwoven between them;

(vii) to insert the weft or cross-threads;

(viii) to beat up the last inserted weft thread to the fabric proper, and

(ix) to finish the cloth by smoothing, stiffening and glazing.

The Hazard of Dust Exposure

From what follows it will be seen that although the processes in the cotton chamber, the blowing room and the card room are dusty, the subsequent processes of spinning, weaving and finishing are free from dust. To understand how respiratory disorders arise in the industry, one must concentrate upon what happens in the card room.

The Hopper Bale Opener

The raw cotton arrives in bales, tightly compressed and containing particles of leaf and seed coat, cotton hairs, fragments of mould and fine sand. In the process of cleaning it passes through the cotton chamber, the blowing room and the card room. In the cotton chambers the bales are broken and the tightly compressed cotton is pulled off in layers and hand-fed by the cotton feeder into a machine known as a hopper bale opener. It then passes into the blowing room, where it is dealt with successively by hopper openers, hopper feeders, porcupine openers and finisher scutchers.

The Blowing Room

The purpose of all these machines is to open out the compressed cotton, to spread it well and, by a beating action aided by powerful currents of air, to free it from a high percentage of its impurities and at the same time ensure adequate mixing of the different qualities of cotton. With the exception of the cotton feeder tending the hopper bale opener, all machines are enclosed in dust-proof covers. The machines, however, have to be hand-cleaned and, as this is a particularly dusty job, the operatives engaged on the work are suitably clothed and wear respirators. Operatives in the cotton chamber and blowing room do not show a high incidence of respiratory disorders.

The Card Room

Emerging from the blowing room, the cotton passes to the card room. Not only does the carding process arrange the cotton fibres in parallel order, but also it carries out the final cleansing process by eliminating such impurities as have survived the previous operations. The name *carding* comes from the Latin, *carduus*, a thistle, since the dried bracts of thistles were originally used for the job. The hooked prickles of *Dipsacus fullonum*, the fuller's teasel, are still used for dressing cloth, their action being to raise a smooth nap on the material treated. For this purpose the dried teasel heads are mounted close together on a wooden frame known as a *teasel frame*. In cotton carding at a later date the thistle heads were replaced by wire brushes operated by hand. Today the combing action is mechanized.

The Carding Engine

The carding engine consists of a horizontal metal cylinder fitted with sharp-pointed steel-wire teeth, 300 or so to the square inch. The rotating cylinder carries the cotton fibres upwards until they meet the flats. These are iron bars 2 inches wide, covered with wire teeth similar to those on the cylinder but pointing the opposite way. The flats move in the same direction as the carding cylinder but at a much slower rate. By this means the fibres are combed into line with each other in a wide mat or lap and short lengths are removed. Women working in the card room, the engine-head tenters, slubber-tenters and intermediate tenters are rarely affected by respiratory

disorders. The strippers and grinders, the operatives in charge of the carding engines, are the principal sufferers. In men over thirty-five years of age working in the Lancashire cotton industry, chronic bronchitis and emphysema are found in 20 per cent of weavers, but in 80 per cent of strippers and grinders (Schilling and Goodman, 1951).

Stripping and Grinding

Usually each stripper and grinder is in charge of sixteen carding engines. Each engine is cleaned four times a day by a vacuum process, and twice



FIG. 402.—Cotton-carding Engine, showing Dust Cloud from stripping the Cylinder in the days before Exhaust Ventilation was applied locally to the Stripping-brush

(From Legge, T. M., Industrial Maladies, Oxford Univ. Press, 1934).

weekly it is brush-stripped. This is done by another form of metallic combing cylinder or brush. The brush-stripping is necessary because the vacuum process fails to clean sufficiently deeply between the teeth of the machine, and the evidence is very strong that the atmosphere in the immediate vicinity of the carding engine contains the injurious substance causing byssinosis (Gill, 1947). In time the teeth of the carding cylinder become blunted and distorted in shape and have to be cleaned by a process known as stripping, and then reshaped and sharpened by grinding them by means of a revolving cylinder covered with emery. The men employed at this work are known as strippers and grinders. In the past the stripping of carding engines was a very dusty job and can still be so if proper pre-

cautions are not taken (fig. 402). It is not surprising that chronic lung disease in these men became known as *trippers' and grinders' asthma.*

Mill Fever

Almost all workers suffer from *mill fever*, or *cotton cold*, when they begin to work in the cotton mills. The term *mill fever* was first used by Arlidge in 1892 to describe a condition occurring on exposure to flax dust. This curious illness has been given many names, according to the dust producing it—*hackling fever* and *flax fever* in flax mills, *combers' fever* in hemp mills, *grain fever* in grain porters and *malt fever* in maltmen. It is uncertain whether an attack caused by one dust confers immunity to some other vegetable dusts, but Doig (1949) records that he knew several workers who had experienced mill fever from jute and who suffered no illness or discomfort on later working in flax mills. *Mill fever* differs from *Monday fever*, with which it is sometimes confused. The latter condition occurs only after exposure of more than ten years to cotton dust, and is the first stage of byssinosis.

Mill fever occurs among cotton workers who have not previously been exposed to the dust. Tolerance is developed usually within a few days, though symptoms may reappear following an absence from work for a time as short as two to four weeks. Oliver (1902) wrote:

> Workers when exposed to the dust for the first time complain of dryness of the throat and suffer from what is called *mill fever*. The indisposition lasts for two or three days; the patients complain of headache, with a sense of feeling ill all over, accompanied by a slight rise of temperature.

A short, slight, dry cough is common and attacks of sneezing sometimes occur. In a few cases an urticarial rash appears on the forearms, especially in women operatives (Gill, 1947). A visitor to the card room frequently has an attack of *mill fever* within the next twelve hours. The new employee may develop symptoms during the first working day, but more commonly the symptoms develop after he gets home in the evening. The temperature may rise as high as 100° to 103° F., but it is invariably normal again by next morning. Headache, malaise and exhaustion accompany the fever, and occasionally nose-bleeding, nausea and vomiting occur. These symptoms may reappear each night for the first few days, or even weeks, of exposure, but most workers become acclimatized within the first month. Severe cases and complications are unknown. Exposure to a higher concentration of dust than that to which the worker has become accustomed may result in a recurrence of *mill fever*.

Byssinosis

The word *byssinosis* is derived from the Greek word βύσσος, meaning *fine linen* or *fine flax*. The disease has been known to exist among cotton workers for nearly a hundred years. It was, and still is, known in the cotton

trade as *strippers'* or *grinders' asthma*, or the *cotton card-room asthma*. It was mentioned by Greenhow in 1860 in a report to the Privy Council in London, and it was described in detail in 1863 by Jesse Leach, factory surgeon, who found it prevalent in the mills of Heywood and district. Three stages are recognized in the development of the disease.

The First Stage

The first of these is commonly called *Monday feeling* or *Monday fever* by card-room operatives (Prausnitz, 1936). The symptoms begin after long exposure to cotton dust, and commonly occur in workers who suffered from *mill fever* on beginning work in the cotton industry. The worker may have been quite well for ten years or more except for slight cough. Suddenly the cough becomes aggravated and exceedingly irritating, or the worker has attacks of tightness in the chest and breathlessness. These attacks usually happen on Monday, and the man is well for the rest of the week. This condition may persist without getting any worse, until the worker leaves the industry.

The Second Stage

In the second stage the symptoms extend over more days in the week and finally become permanent. Removal from exposure to cotton dust during the first stage of byssinosis will result in complete cure, but symptoms recur suddenly and with great severity if work at the mill is resumed. If exposure continues, attacks of bronchitis or asthma follow. These are in no way different from the bronchitis and asthma of the general population. Intervals of absence from work become necessary, and these increase in frequency and duration. Even at this stage recovery can occur, at least to the extent of making the individual fit for some job, if exposure to cotton dust is ended.

The Third Stage

But many men who reach the second stage do not leave the industry, and pass into the third stage of disabling byssinosis. The symptoms of tightness of the chest and dyspnœa are so distressing that the worker has to leave the cotton industry. Though he will have some relief when he is away from cotton dust, the dyspnœa remains as a permanent disability. The disease progresses to chronic bronchitis with emphysema. Cough with muco-purulent sputum is present, but there is no fibrosis and therefore characteristic X-ray findings are absent. Post-mortem examination reveals advanced emphysema (Shaw Dunn and Sheehan, 1932). Thus, there are no changes which distinguish byssinosis from the chronic bronchitis and emphysema occurring in persons not exposed to cotton dust.

Diagnosis and Prognosis

The rate at which the disease progresses depends upon the amount of dust inhaled and on the susceptibility of the individual. Dust-control methods are helping to reduce the incidence and severity of the illness.

Diagnosis depends on a history of long exposure to cotton dust, together with the symptoms and signs of chronic bronchitis and emphysema.

Ætiology of Byssinosis

The exact mode of action of the cotton dust is not known. The natural history of the disease suggests that there is an allergen in cotton dust which sensitizes the bronchial mucous membrane and produces bronchospasm and possibly swelling of the mucous membrane. After prolonged exposure, this leads to emphysema. But the work of Furness and Maitland (1952) does not confirm this hypothesis. They prepared different extracts of cotton dust and did skin tests on men with mild and severe symptoms of byssinosis and on men who had worked in cotton card rooms without symptoms, and on others who had never worked in a cotton mill. Those who had a wheal and flare type of reaction were equally distributed between all these groups. This reaction probably indicates hypersensitivity to cotton dust, but it does not indicate a hypersensitivity specific to byssinosis.

The Morbidity and Mortality Rates

The morbidity and mortality rates of men working in the cotton industry emphasize how serious the effects of exposure to fine cotton dust can be. Bradford Hill (1930) compared the sickness experience of strippers and grinders with that of warehousemen and ring-room spinners in the period 1923–7. He showed that after the age of thirty sickness due to respiratory disease was for the strippers and grinders three times as high as for those working in the same industry but not in the dusty rooms. Death-rates from respiratory disease in the triennium 1930–2 were equally striking. For strippers and grinders over fifty-five the respiratory mortality was at least three times as great as for men working in other parts of the cotton mill. But the respiratory mortality of strippers and grinders declined appreciably between 1920 and 1930. This was no doubt to some extent due to the recession in the trade at this time and the consequent reduction in the time spent in the mills; but in the previous decade attempts had been made with some success to enclose the stripping brush and to remove the dust by exhaust ventilation. This, no doubt, also had a beneficial effect on the death-rate.

Weavers' Cough

From time to time outbreaks of an acute illness called *weavers' cough* have occured in cotton-weaving sheds (Collis, 1913; Bridge, 1928). These outbreaks have always been associated with the handling of mildewed yarn, usually the warp which is more heavily sized than the weft. Size consists of flour, tallow, china clay and water, and constitutes an excellent medium for the growth of moulds.

Ætiology

The association of the illness with the handling of mildewed yarn and its absence in workers even in the same factory who handled non-mildewed

yarn points very strongly to a mildew as the cause. The air over the looms using the affected yarn was found to contain many fragments of mycelia and conidia, and examination of the dust and of the yarn revealed the presence of various fungi, including penicillium, aspergillus, mucor and others.

Symptoms and Signs

The first sensation experienced is one of constriction of the chest beneath the sternum; this feeling extends up towards the larynx, and the act of breathing becomes an effort. Next, breathing becomes rapid and rather shallow, with occasional deep sighs, and a persistent irritating cough develops. This cough is associated with expectoration of thick yellow or yellowish-green sputum and a sweetish taste in the mouth. Later, the difficulty in breathing progresses until it resembles asthma and may prevent the sufferer from sleeping, or arouse him from a disturbed rest fighting for breath and drive him to throw open the bedroom window for air. Food may be rejected in a spasm of coughing, epistaxis may occur and the fauces are found red and inflamed. The individual suffers from general malaise, aching limbs and back, severe headache which is usually frontal, and shivering, ending in perspiration; the temperature may reach 100° to 102° F. in the evening. As the symptoms persist, the individual cannot hurry when walking, finds an ordinary slope difficult to climb and tires rapidly; the appetite becomes affected and weight is lost. Pronounced cases have been deprived of sleep for a week and have been confined to bed, but those less affected have managed to remain at work (Middleton, 1926).

Prognosis

The illness attacks both acclimatized and new workers, and immunity does not develop. Thus Middleton (1926) describes the case of a man affected in an outbreak in Burnley in 1926 who had also suffered in an outbreak at another mill in the same town in 1911. Sometimes a chronic condition is established with persistent cough, though rarely any sputum. Examination shows scattered rhonchi in the chest. Such a man may be unable to return to work without a relapse of symptoms although he remains well in a different job.

Acute Respiratory Illness from Contaminated Cotton

In 1941 an acute illness among people handling dusty, low-grade, stained cotton broke out in the United States of America (Neal, Schneiter and Caminita, 1942). This illness is a distinct clinical entity not connected with byssinosis. Sudden outbreaks occurred among rural mattress makers and among workers in one cotton mill and several cotton-seed processing plants. Schemes for rural mattress making were designed to reduce the cotton surplus and to benefit farm workers. Poor families who wished to make mattresses for home use were supplied with 50 lb. of cotton and 10 yards of cotton ticking for each mattress. They gathered at a central depot

to make their mattresses, and contact with the cotton was limited to the time allowed each family to make its quota of mattresses.

Symptoms and Signs

The illness occurred among workers exposed to high dust concentrations. The dust was produced in opening the bales, carding the cotton, filling and closing the mattresses, and finally beating them. Dust concentrations were particularly heavy when low-grade stained cotton was used. Symptoms began within the first six hours of exposure, with fatigue and generalized aching. Anorexia, headache, nausea and vomiting followed. The vomiting persisted for from six to nine hours. Rigors, with a temperature of 102° F., and headache occurred. Many people complained of abdominal pain or cramp and a sense of retrosternal discomfort which prevented them from taking a deep breath. Symptoms lasted from two to five days.

Ætiology

This disease has been attributed to the effects of the endotoxin of *Aerobacter cloacæ* (Neal and others, 1942; Schneiter and others, 1942). Samples of cotton from all sources where outbreaks occurred were heavily contaminated with this organism almost to the exclusion of other bacteria. Its abundance in this cotton was associated with particular climatic circumstances and undue exposure of the cotton to contamination from the soil. The clinical symptoms were reproduced experimentally in human beings by the inhalation of dust from the cotton and by the inhalation of a mist from cell-free culture filtrates of *A. cloacæ*. Although the clinical picture is similar to that of weavers' cough in Lancashire, there is no proof that these two conditions are related nor can a relationship with mill fever, byssinosis or bagassosis be established.

Control of Dust in Cotton Mills

It was not until 1908 that attempts to control the dust in cotton mills were made. Obviously the ideal method is to ensure that the cotton dust does not escape into the atmosphere. This has not yet been found possible. Considerable improvements have been achieved by the introduction of dust covers and exhaust fans for machines in the cotton chamber and blowing rooms. Unfortunately, even in the best equipped mills, at least one-third of the trash is carried into the card room in the laps; it is this third which is eliminated in the carding process. The sliver emerging from the carding engine is almost dust-free.

Oiling the Cotton

In spite of constant experimental work designed to make the atmosphere free from dust, particularly in the vicinity of the carding engines, no completely satisfactory method of dust suppression has yet been evolved. In some mills 1 per cent mineral oil is sprayed on the cotton in the hopper

bale breaker. This treatment helps to reduce the dust and does not interfere with the quality of the yarn, since most of the oil is absorbed before this stage is reached. The modern flat carding engine is not totally enclosed. Attempts to do this have been made by completely hooding the whole of the top of the carding engine and exhausting by means of a fan. The air, drawn through the hood, is then filtered before re-entering the room.

Exhaust Suction and Air Conditioning

The under-space of the carding engine is completely sealed off, and to clean under the engine the stripper and grinder has only to pull a small lever; the entire collection of dust is then swept away by the creation of a vacuum. According to Gill (1947) this arrangement has greatly reduced the amount of dust in the atmosphere; 3·66 lb. was extracted from the hoods of twelve carding engines, through which 11,400 lb. of cotton had passed in forty-four hours. These figures do not include the weight of debris from underneath the carding engine. Many mills have insufficient air space, with the machinery crowded into too small an area. Greater air space means a greater dilution of the dust. Improved air conditioning, in conjunction with improved dust extraction, is necessary. Air conditioning must include a correctly controlled temperature, the right humidity and adequate air movement. These are also the best conditions for the cotton fibre. But the engineering problems involved are complex and the British Cotton Industry Research Association at the Shirley Institute, Didsbury, Lancashire, is continuously employed in researches on the subject.

Need for Periodical Medical Examination

A careful selection of operatives for the card room, and regular examination of existing workers, offer the best protection. All intending card-room operatives should be medically examined before engagement, and every effort should be made to exclude those likely to be susceptible to the disease. Any operative who gives a personal or a family history of asthma, bronchitis, hay fever, eczema or recurrent urticarial attacks should be excluded from the card room. The general build and shape of the thoracic cage should be noted, and also the type of respiration. Mouth breathers are bad subjects. Malformation of the nasal bones, deflected septum, nasal polypi, enlarged tonsils and extensive adenoid vegetations are all contra-indications for work in the card room. A chest expansion below 3 inches is suspect. All operatives are urged to practise nasal inspiration and to wear protective respirators.

Need for Improved Engineering Control

In spite of all the improvements introduced, a substantial number of men employed in the more dusty mills spinning the coarser grades of cotton were found by Schilling and others (1952) to be suffering from byssinosis. Of 131 men over the age of thirty-five with at least ten years'

exposure in card and blowing rooms, 45 had no symptoms, 52 were in the first stage of the disease, and 16 and 15 in the second and third stages respectively. Schilling (1952) points out that there could be a more effective control of the disease if medical supervision and engineering control of dust were combined. There is at present no system of periodical medical examination of card-room workers. If there were, the incidence and severity of the disease found would indicate both the mills where improved methods of dust control were needed, and the men who were getting progressively worse and who should be encouraged to leave the industry or work in the less dusty mills.

(*b*) Hay

Agriculture is one of the largest industries in Great Britain, employing about 800,000 persons. In rural areas, especially where there is a high rainfall, the workers are liable to lung disease arising from the handling of mouldy hay and straw. The condition which has been called *farmers' lung* was first mentioned by Campbell (1932) and has been clearly described by Fawcett (1938) in Westmorland and Cumberland, but it exists in other agricultural districts, including Lancashire, Wales, Lincolnshire and Devonshire. Fuller (1953) collected thirty-two cases from the Devonshire area in eight years.

Farmers' Lung

The relation of farmers' lung to the inhalation of mouldy hay dust is unquestioned, but the commonly held view that it is a true pulmonary mycosis is based only on the occupational history of the patient and the cultivation of moulds from his sputum. It is noteworthy that the single necropsy in the series of cases described by Fawcett (1936) revealed no sign of pulmonary mycosis. An analogous condition has been reported from Scandinavia, where nine cases of *threshers' lung* were described in workers exposed to the dust of mouldy wheat (Törnell, 1946). This author reported marked improvement after treatment with potassium iodide. Hoffman (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 day before threshing. They were infected with a fungus of the genus *Ascomycetes*.

Exposure Hazard

Hay becomes mouldy when it has lain wet in the field after cutting. The extra moisture encourages the growth of various moulds, and when the hay is handled, large amounts of spore-bearing dust may be inhaled. The cowman puts out fresh hay for the cattle each day, and when this is being done it is impossible to see across the cowshed for dust. The same thing happens during the threshing of ricks of oats, wheat and barley which have been harvested in bad weather. The greatest risk is to members of mobile threshing teams, who may be repeatedly exposed.

Symptoms and Signs

The usual history is that a farmer after working continuously for a time with mouldy hay develops a short, dry cough, with little or no expectoration. He may also suffer from general malaise, weakness and slight fever, but the outstanding feature is distressing dyspnœa provoked by any physical exertion. Moist sounds may be heard in the lungs, but radiographs of the chest are normal. The acute illness is so short that the patient does not always consult a doctor. Removal from exposure to the dust of mouldy hay is followed by rapid recovery, which is complete and permanent if further exposure is avoided.

Subacute Phase

The subacute phase, however, is more incapacitating and often brings the patient to hospital or chest clinic. The man gives a history of repeated exposure to mouldy hay or straw during the previous two or three months. He complains of increasing breathlessness, with a short, dry cough and bouts of fever in the evenings after exposure. Often he is cyanosed, but there is no bronchospasm or other evidence of asthma. Physical signs include patches of dullness and crepitations. The erythrocyte sedimentation-rate is increased. X-ray examination of the chest shows a fine alveolar mottling, with evidence of compensatory emphysema. The symptoms clear up in a month or two after the patient has been removed from contact with the dust, but the radiographic appearances remain abnormal for three or four months.

Chronic Phase

With successive seasonal recurrences the disease tends to become chronic. Fortunately, such cases are rare. The cough becomes more productive, the sputum is muco-purulent and slight hæmoptysis may occur. Physical weakness is marked, and so great is the dyspnœa, even after slight exertion, that the man becomes incapacitated for work. The dullness to percussion is now more extensive and added sounds are found over wider areas. Radiographs show increase in the density of the mottling, patches of opacity due to coalescent areas of fibrosis, increase in the hilar and perihilar shadows, marked restriction of the movements of the diaphragm and considerable areas of emphysema. This stage of pulmonary fibrosis with emphysema and sometimes bronchiectasis is irreversible. It is indistinguishable from what is seen in the final stages of byssinosis and bagassosis.

Ætiology and Pathology

Duncan (1945) examined specimens of sputum from cases of supposed farmers' lung and found mould spores often present. From one specimen alone he cultivated six different species of *Aspergillus*, as well as other moulds. He is in no doubt that the fungal elements in the sputum represent recently inhaled matter, and he finds it significant that fungal conidia are

scarce or absent in the sputum of patients who have been removed for even a short time from exposure to hay dust. He points out that the rapid clinical improvement brought about by a change from the dust-laden atmosphere does not suggest a progressive mycosis. Undoubtedly the fungus is an essential factor in the ætiology of farmers' lung, but its rôle has not been determined. Duncan poses the questions—(*a*) Does it cause a true pulmonary mycosis? (*b*) Does it behave as an allergen? (*c*) Does it produce, in its growth on the hay, some noxious substance which causes the symptoms and pathological changes? (*d*) Does it, by breaking down the hay fibre, produce a sufficiently fine vegetable dust to cause by inhalation a disorder analogous to byssinosis? The suggestion conveyed in the last question seems to be the most plausible, but there is need for much further research.

Preventive Treatment

In cowsheds and in places where threshing machines are in use, a systematic plan using wet methods for dust suppression should be carried out. Schemes for mechanization of farm work should embody the means to minimize the escape of vegetable dust into the working environment. The risk to the farm worker of dust disease of the lungs is greatly reduced by the use of the combine-harvester, when the corn is threshed as soon as it is cut and is therefore free from moulds and spores.

Broken Wind of Horses

Stall-fed horses are exposed to a much greater degree than farm workers to inhalation of dust of mouldy hay. In consequence of this they develop a disease known as *heaves*, or *broken wind of horses*. The ætiology of this condition is disputed, but it seems not to occur in pastured animals. It is an affection of horses in advanced and middle age. It is rare before the age of five years and its frequency increases with age. It frequently follows some kind of pneumonia.

Symptoms and Signs

It may be preceded by chronic cough, an annoying defect which may disappear in the summer and reappear in the winter months. The symptoms consist of wheezing, a peculiar hollow cough and the fact of being too easily fatigued. The symptoms are aggravated by heavy exertion, inclement weather and bad working conditions. The principal physical sign is a double expiratory movement of the flanks brought about by the use of the abdominal muscles in an effort to overcome the effect of loss of elasticity of the lungs. A horse is tested for broken wind by pulling it up suddenly after galloping, when a broken-winded horse will be recognized by the cough and double respiration (Lovell, 1952).

Morbid Anatomy

In broken wind of horses the main pathological change is hypertrophic vesicular emphysema. At necropsy, on opening the thorax the lungs are

over-distended and marked on the surface by deep imprints of the ribs. Air bullæ under the pleura are often present where the breakdown of alveolar walls is extensive. Right heart hypertrophy occurs in advanced cases. Although chronic bronchiolitis is a constant finding, no adequate mycological or bacteriological studies have been made of the disease. Presumably it is of the same nature as farmers' lung.

Preventive Treatment

Broken wind of horses has become prevalent only since timothy hay, red clover, alfalfa and cultivated grasses have been introduced as feeding stuffs. The claim is made that it can be prevented if the hay is wetted sufficiently to suppress dust from rising and being inhaled.

(*c*) Grain

Many observers have from time to time reported a high incidence of respiratory disease among men who handle grain; for example, grain porters, dock labourers, threshers, millers and maltsters, all of whom may be exposed to high concentrations of grain dust during their work.

Grain Sifting

In his description entitled *Diseases of Sifters and Measurers of Grain*, Ramazzini (1713) states:

> All kinds of grain, and in particular wheat, whether they are stored in pits and trenches as in Tuscany, or in granaries and barns with roofs as is done in the whole country on both sides of the Po, always have mixed in with them a very fine dust; not only the dust that they pick up from the threshing-floor, in the threshing, but also another less innocent sort which is shed from the grain itself when it is kept for long. Inasmuch as the seeds of cereals are full of volatile salt, and so much of it that unless they are well dried in the summer sun before being stored they become overheated and very soon crumble to dust, it follows that fine particles are continually dropping from the skin with which the grain is coated; besides these there is the residual dust and decay caused by the grubs, borers and weevils that consume the grain and by other such corn pests and their excrements. Hence, whenever it is necessary to sift wheat and barley or other kinds of grain to be ground in the mill, or to measure it when corn-merchants convey it hither and thither, the men who sift and measure are so plagued by this kind of dust that when the work is finished they heap a thousand curses on their calling. The throat, lungs, and eyes are keenly aware of serious damage; the throat is choked and dried up with dust, the pulmonary passages become coated with crust formed by dust, and the result is a dry and obstinate cough; the eyes are much inflamed and watery; and almost all who make a living by sifting or measuring grain are short of breath and cachectic and rarely reach old age; in fact, they are very liable to lapse into orthopnœa and finally

dropsy. The dust moreover is so irritating that it excites intense itching over the whole body, of the sort that is sometimes observed in nettlerash.

Dusty Cargoes

Today large seaports with modern equipment handle cargoes of grain by suction methods. Smaller ports are not so equipped, and in any case suction is not suitable for every type of grain cargo. In many seaports dock labourers are called upon to spend at least a third to half of their time working grain and seed ships under conditions creating incredible amounts of dust. The dustiness of a grain cargo will vary not only with the particular grain or seed being handled, but also with its place of origin. Thus dockers in Antwerp complain that grain from the Black Sea is the most dusty. The port of Hull imports wheat from Canada, Australia and India, maize from South Africa, South America, Australia and Russia, barley from Turkey, Morocco and Russia, oats from Russia, millet from India, linseed from South America and Russia, rape from Africa and India and cotton seed from Egypt (West, 1951).

Filthy Working Conditions

Usually cargoes of grain or seed are carried loose in the holds in bulk. Only occasionally is the grain found bagged on arrival, and then no dust problem arises. The apparatus found to cause the greatest amount of dust is the elevator fitted with hoppers. Very often unloading is carried out by the age-old method of shovelling the grain or seed into wicker baskets which are then hoisted over the sides of the ships into trucks or lighters. Sometimes a grain ship is unloaded by the more modern method of buckets passing on an endless belt into a silo. With the method of shovel and basket the dockers find themselves working farther and farther down in the hold as unloading proceeds. Here the conditions are very dusty indeed and the men become filthy and caked with dust. The grain works into their clothes and boots and the dust hangs on their eyebrows and eyelashes. Various methods of keeping the dust from the ears and nostrils are used, such as an old stocking worn round the nose and mouth.

Symptoms and Signs

A person who is not used to it, on going down into the hold of a ship where grain is being unloaded immediately experiences irritation of the upper respiratory tract with sneezing, cough and a burning sensation in the mucous membranes of nose and pharynx. This lasts all the time he is in the dust-laden atmosphere and even persists after he gets back into the fresh air, sometimes for several hours. As to the docker, the presenting symptom is shortness of breath while he is actually on the job. Only rarely does he complain of cough and expectoration. But nothing is known as to the physical signs, if any, which may occur in these men. Do they suffer unduly from bronchitis, asthma or chronic emphysema? What radiological

changes, if any, are to be found in their lungs? Although they are no longer a casual labour force, we have only a vague picture of their health and we do not know how it compares with that of other sections of the working community. A survey of this problem by clinical and radiological methods badly needs to be done.

Ætiology and Pathology

As long ago as 285 B.C. in his *Historia Plantarum*, the earliest extant Greek herbal, Theophrastus of Eresos raises the question why the dust from wheat is more irritating than that from other kinds of grain. Grain dust consists of husk particles, cellulose hairs and spikes, starch granules, moulds and 5 per cent of mineral particles.

The hairs differ according to the grain concerned. They are generally thick, strong and sharp, and therefore they can penetrate into the mucosa of the upper air passages. Those from wheat are long and stout and, according to the dockers themselves, wheat dust seems to be the most irritating even when the grain is clean and healthy. Maize has finer hairs, and maize cargoes cause less severe symptoms. McNair and Middleton (1928) investigated grain dusts, making forty-three dust counts with Owens' dust counter. Of these, twenty-four were found to contain from 190 to 3,880 hairs per 500 millilitres of air with an average of 932. The most usual figure was 1,050 hairs in wheat dust and 1,147 in barley dust.

In the wheat-flour mills of Kansas City, asthma occurs in some of the men exposed to the dusts arising from the first cleaning of the wheat grain. The minute hairs of the grain have been held responsible. Injection of infinitesimal amounts of wheat-hair extract gives rise to severe local reactions and to asthma in sensitive millers (Duke, 1935).

From time to time fungi growing on mouldy grain have been found in the sputum of workmen. Thus in 1928 *Aspergillus fumigatus* and *Mucor mucedo* were held responsible for a small outbreak of respiratory disease in men who shovelled barley in a French malting-house. Such outbreaks raise the old question as to whether the fungi are pathogenic or merely saprophytic. The need for systematic mycological studies in the occupations where grain dusts are encountered is apparent.

(*d*) Bagasse

Bagasse is a word of French origin indicating the cellulose fibre of sugar-cane after the sugar has been extracted. It is used for making boarding for the interior decoration of buildings and for thermal insulation. The sugar-cane which is used for this purpose is grown in Louisiana and the bagasse is shipped to Great Britain in bales. Inhalation of dust from certain consignments has caused a disease of the respiratory system known as *bagassosis*.

Fungi of Twenty Species

When samples of bagasse are examined, innumerable fungi are found to be present and the dust arising from bagasse in a badly conducted

factory may contain as many as 240 million fungal spores per gram. These consist of the uncultivable teliospores of fungi of the order *Pucciniales* (rusts), as well as the conidia of many species of saprophytic fungi. About twenty different species have been isolated in culture, including *Pæcilomyces varioti*, *Aspergillus fumigatus*, *A. niger*, *A. terreus*, *A. candida*, *Trichoderma lignorum*, *Monilia sitophilia*, and species of *Penicillium*, *Mucor* and *Rhizopus*. Amorphous silica forms only 1 to 2 per cent of the bagasse, and quartz 0·1 per cent, many of the quartz particles being from 20 to 30 microns in diameter. It is most improbable therefore that exposure to bagasse dust could lead to silicosis.

Dust Hazard from Shredding Machine

A febrile illness affecting the lungs of workers exposed to the dust of bagasse has been described in the United States of America, England and Mexico. In England the disease arose in workers on a machine called the shredder, which broke up the bales of bagasse (Hunter and Perry, 1946). In England before 1940 the bales were broken in a soak pit by a wet process causing no dust. During the Second World War in order to save shipping space the material in the bales was packed more tightly. In order to open such bales it became necessary to use the shredder, which produced heavy clouds of dust. In the factory concerned the shredder was housed in a separate building and no hazard existed in the main buildings away from this machine.

Symptoms and Signs

Out of twenty-one men exposed to the dust in a period of fifteen months, ten (47·5 per cent) developed a characteristic acute respiratory 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 twenty-five days' illness, but unfortunately no necropsy was performed. These cases afford strong evidence that a specific disease, manifesting itself by acute bronchiolitis, collapse and broncho-pneumonia, has occurred in the lungs of men handling bagasse.

Complete Occupational History

In 1943 all the workers in the factory were interviewed and occupational histories taken. It was found that out of 163 men employed, twenty-two had worked in coal mines, and of these, nineteen 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 in his chest the radiographic changes already described. He had a cough and was short of breath.

Ætiology and Pathology

The ætiology of bagassosis remains unknown. Between bagassosis, farmers' lung, broken wind of horses and byssinosis certain resemblances can be picked out, but we must bear in mind that the pathology of these diseases has not been fully elucidated. It is important to emphasize that the disease produced by inhaling bagasse dust shows none of the appearances of silicosis. Schneiter and others (1948) hold the view that it is due to the endotoxin of *Aerobacter cloacæ*. But it seems more 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 even bronchopneumonia result. In two of the patients described by Hunter and Perry (1946) the condition did not resolve, and they developed a fibrosis of the lungs with cough and sputum and great shortness of breath. Radiographs showed thick bands of fibrous tissue traversing the lung fields so as to simulate cavities. One of these men died, and at necropsy he was found to have chronic bronchiolitis and bronchiectasis, but no large cavities were found.

Absence of Evidence of Pulmonary Mycosis

The disease is not a true pulmonary mycosis, though it is possible that fungi play a rôle in breaking down the bagasse fibre into a very fine vegetable dust. *Aspergillus* is present in all specimens of bagasse dust and it can be grown from the sputum of men exposed to this dust. This fungus had been present in the sputum of the man who died, but, of course, this is no proof that it was the cause of his illness. Indeed, the post-mortem material from the lung showed no evidence of *Aspergillus*, and histological examination in no way suggested a disease of fungal origin. On the other hand, administration of bagasse to rabbits produced pneumonic lesions from which *Aspergillus* was isolated, whereas bagasse which had been autoclaved caused only a foreign-body giant-cell reaction and slight fibrosis (Gerstl and others, 1949).

Preventive Treatment

Resumption of wet methods of working prevented further outbreaks of the disease. When the bales were again broken in the soak pit no appreciable dust was produced.

(e) Paprika

The red-pepper industry of Eastern Europe dates back to the sixteenth century, when the Turkish invaders introduced the paprika plant which they grew for medicinal purposes. In modern times the species *Capsicum annuum longum* has been cultivated in the region of Szeged, and this town is

the centre of the Hungarian red-pepper industry. Other centres along the Danubian plain include Horgoš in Yugoslavia. The fruit of the plant when dry is deep red and is four inches long, one inch broad at the stalk and tapers to a point.

Exposure Hazard

Women called splitters cut open the fruit with a knife and strip out a series of thin pale ribs which line it longitudinally. The ribs are discarded before grinding because they contain too much of the pungent principle capsaicin. Fruits picked late in the season are often infested with the mould *Mucor stolonifer*, the colonies of which are removed along with the ribs by the women splitters. The air of the workroom is then clouded by spores and mycelia which settle everywhere as a fine dust. Workers grinding the split fruits and those handling the finished pepper do not suffer.



FIG. 403.—Bronchogram showing Bronchiectasis in a Paprika Splitter
(*von Kovàts, F. (1937), Acta med. Szeged, 8, 1*)

Symptoms and Signs

A proportion of the splitters, especially those who have been many years at the job, suffer from loss of weight, poor appetite, poor sleep and even hæmoptysis. There is slight fever, and signs of bronchitis develop. In its chronic form the disease presents with cough, fetid expectoration, and apical dullness and cavities in the lungs. X-rays confirm the presence of consolidation and the cavities show in bronchograms (fig. 403).

Ætiology and Pathology

Necropsy shows extensive fibrosis of the lungs with bronchiectasis, and right heart failure. It is not clear what part the fungus plays in the ætiology

of the disease, but, of course, the presence of its spores in the sputum and in the bronchi after death does not prove it is more than a saprophyte. Although generations of women had followed this occupation, using the same method for eighty years, neither employers, workers nor doctors became aware of its existence until 1931.

Preventive Treatment

It was at first sought to prevent the disease by the use of cotton-wool respirators, but these had little success. Ultimately the chief botanist of the Paprika Research Institute in Szeged succeeded in cultivating a variety of the plant in which the fruit had ribs containing much less capsaicin. Since it is possible to obtain first-class red pepper by grinding this fruit whole, there is now no risk to the worker of inhaling the fungus spores for, indeed, the paprika splitter is no longer needed (Von Kováts, 1937). This example of the application of research in horticulture to the protection of the worker is unique.

(*f*) Tobacco

In factories, tobacco dust affects especially beginners, causing irritation of the eyelids and conjunctiva with lachrymation. The dust or juice from the leaves may cause dermatitis on the back of the hands and face. The genitalia may become affected where the dust is conveyed by unwashed hands. Acute nicotine poisoning with insomnia, headache, watering of the eyes, nausea, vomiting and tachycardia may occur. One fatal case took place in a workman employed in steeping tobacco in water. Gardeners and vine growers have been affected after using insecticidal sprays containing nicotine. Where a solution containing nicotine comes in contact with the skin, the use of hot water to remove it must be avoided, otherwise absorption will be facilitated. Chronic poisoning in tobacco workers is rare. It takes the form of tobacco amblyopia with colour scotomata in the visual fields due to degeneration of ganglion cells of the retina, especially in the macular area. Optic atrophy with partial blindness may follow. Its occurrence in non-smokers has been many times confirmed. The occurrence of attacks of asthma in cigarette cutters has been recognized for many years, but the condition is less common since the industry has been mechanized. At the necropsy on two women workers Zenker observed brown patches on the pulmonary parenchyma and on the hilar glands as well. He attributed these lesions to the action of tobacco and called the condition *tobaccosis*. In a study of the tobacco drying and stemming industry, McCormick, Smith and Marsh (1948) found that dust liberated in the processes contained from 22 to 58 per cent of free silica, and that the particles were of respirable size.

(*g*) Wood

Bronchial asthma due to allergy to a particular wood dust has often been noted. Dermatitis, as well as running of the eyes and nose, also may occur (see p. 686). Usually only those isolated workmen who are unduly

sensitive are affected, but sometimes small outbreaks occur. Thus in 1905 the use of South African boxwood (*Gonioma kamassi*) in shuttle making in Lancashire caused a great deal of disability. Out of 112 men seen, thirty-four experienced difficulty in breathing, but there were other toxic symptoms as well (see p. 687). In 1936 Bridge described the effects of bronchial spasm in men handling mansonia wood (*Sterculiacea altissima*). Doig (1949) recorded two small outbreaks of asthma in factories where Western red cedar (*Thuja plicata*) was handled. Thirteen men were known to have suffered from *cedar poisoning*; one of them was still working and still allergic to the wood after eleven years, another had been free from symptoms for two years since his retirement from work.

(*h*) Gum Acacia

Gums have come into increasing use in industry and, in the various reports on sensitivity to different gums, acacia, Karaya gum and tragacanth have been the three most common allergens, in the order listed (Brown and Crepea, 1947). Bohner and others (1941), in the United States of America, were the first to describe a group of printers with asthma due to sensitivity to gum acacia. They reported ten cases and reviewed the literature of isolated reports of sensitivity to inhaled acacia. In Canada, Sprague (1942) reported a further case of asthma in a printer sensitive to inhaled acacia. Fowler (1952) described thirty-two cases of *printers' asthma* in men exposed to a spray, made of gum acacia and isopropyl alcohol, which is used in colour-printing. This spray is thrown over each sheet and the gum forms a barrier between successive sheets on which the colour-print is still wet. Colour from one sheet is thus prevented from passing on to the back of the next sheet added to the pile. With many spray machines working, the mist can be seen spreading from one printing-machine to the next. The first patient attended a hospital complaining of asthma which he believed was due to the spray fluid to which he was exposed at work. A few weeks later thirty-one further printers were examined. They came in response to a letter sent to London printing chapels asking members who thought they were affected by the spray fluid to attend for examination. The average duration of exposure before asthma developed was 4·8 years, but the period of exposure was half as long for the six printers who had a past or family history of allergy. In one firm 19 per cent of the printers had asthma and a further 30 per cent had early symptoms of asthma due to sensitization to the spray. This industrial hazard can be eradicated by the substitution of dextrose for gum acacia in the spray fluid.

Dusts of Animal Origin

Reports as to the harmful effects of animal dusts have been very scanty and it is clear that such dusts must be accounted of minor importance in the production of respiratory disease. They include the dusts of bone, feathers, fur, hair, horn, ivory, leather, silk and wool. Although such dusts contain virtually no silica, it should be pointed out that both bone and horn

are sometimes shaped by the use of sandstone grinding wheels, the dust from which could in time produce silicosis.

Occasionally animal dusts may carry infections, especially anthrax. Pulmonary anthrax at one time occurred from inhaling the dust of infected wool (see p. 629). The crushing of bones from anthrax-infected animals may carry a similar risk when sun-dried bones are used (see p. 627).

(*a*) Feathers

In workshops handling feathers there seems to be a direct relationship between the dustiness of a process and complaints by workers of cough and a feeling of tightness in the chest (Bridge, 1934). This subject needs further investigation by clinical and radiological methods.

(*b*) Leather

The worker who dresses leather may inhale dust containing leather together with smaller quantities of china clay, carborundum and calcium carbonate. McLaughlin (1952) described such a man aged forty-six, who had been a leather dresser for thirty-two years. He had no cough or sputum, but he complained of slight dyspnœa on exertion. Clinical examination revealed little abnormality except that the percussion note was slightly impaired over both upper zones and that the breath sounds were diminished over the whole of the lung fields. His exercise tolerance test and cardiac response to effort were normal. X-rays of the lungs showed nodular shadows resembling those of silicosis. It is not known what are the histological changes in the lungs which cause these X-ray shadows. The occupational history precludes a diagnosis of silicosis because the man had not been exposed to the dust of free silica.

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