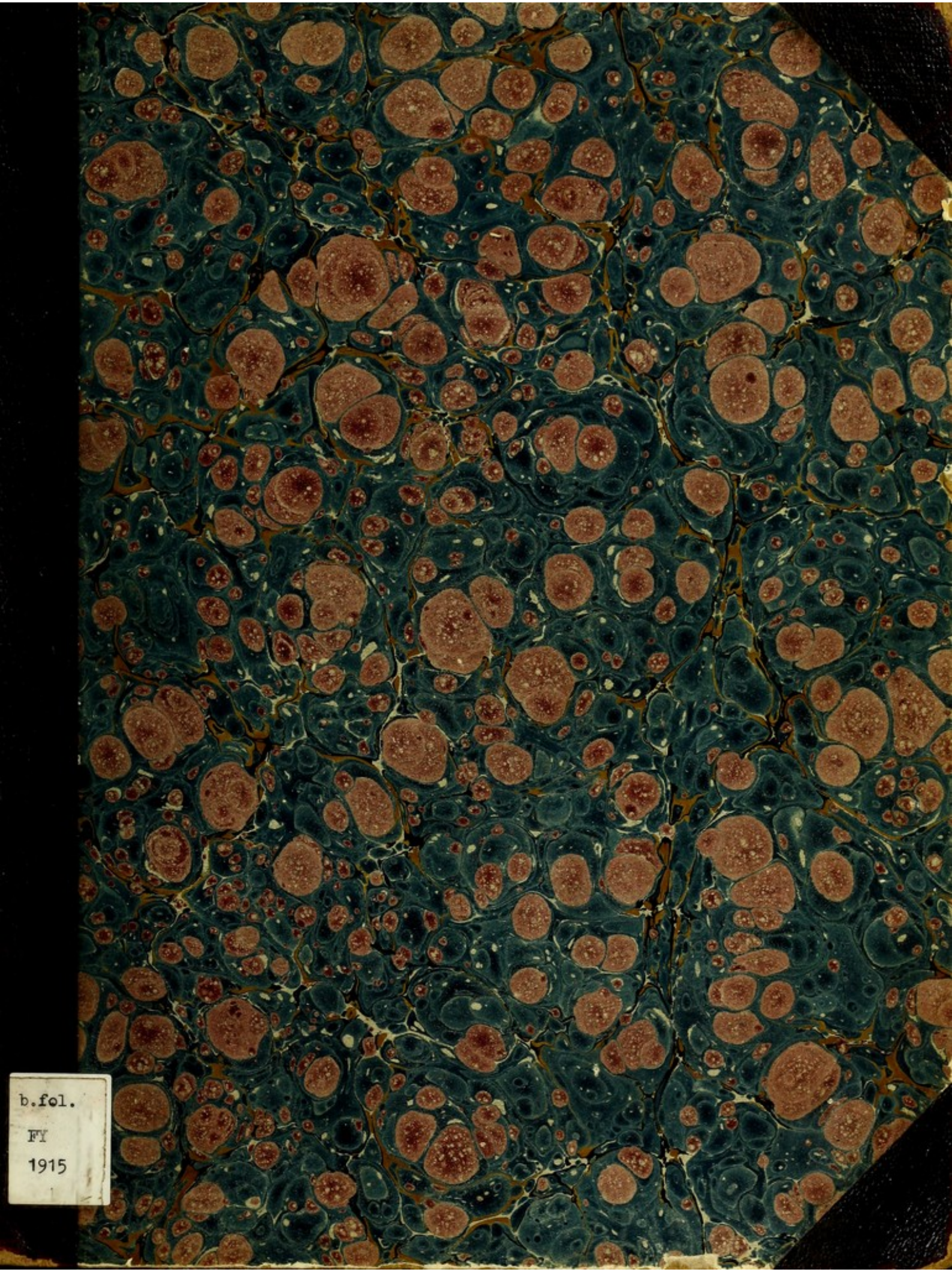
Industrial pneumonoconioses, with special reference to dust-phthisis / by Edgar L. Collis.



MILROY LECTURES

(1915).

INDUSTRIAL PNEUMONOCONIOSES,

WITH SPECIAL REFERENCE TO

DUST-PHTHISIS.

BY

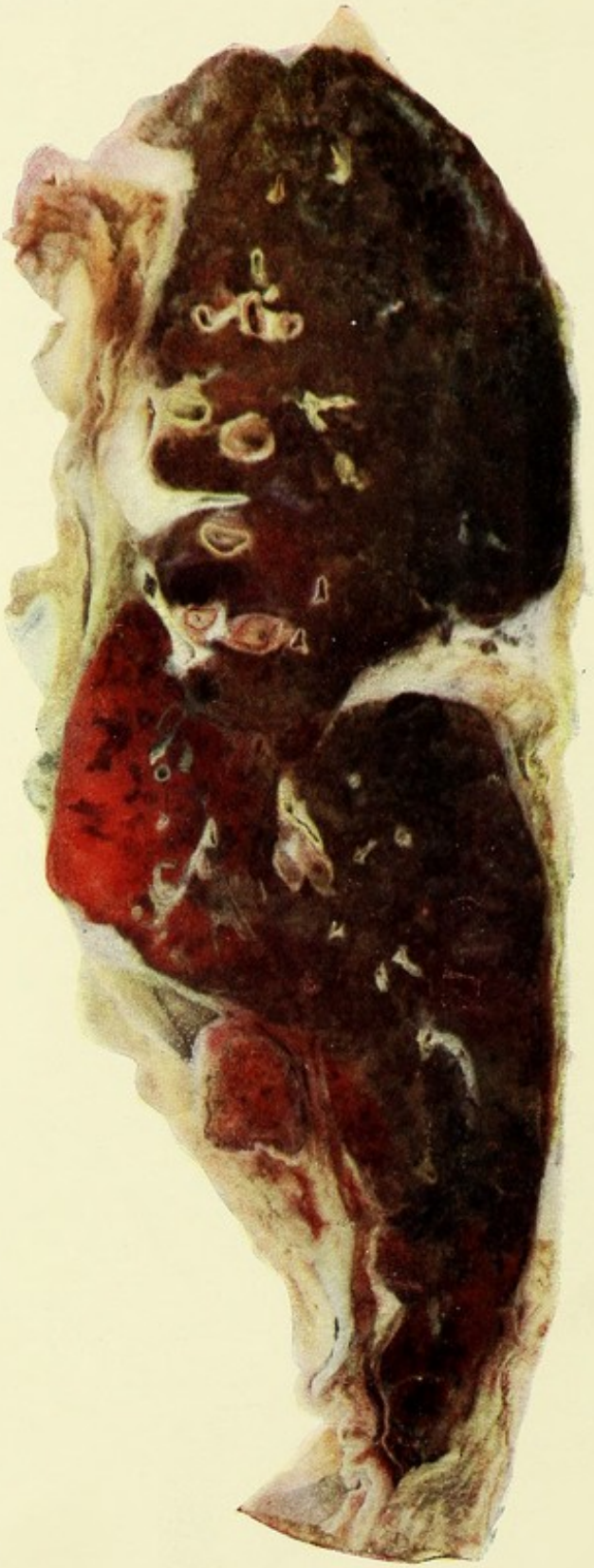
EDGAR L. COLLIS, M.B. (Oxon.),

H.M. Medical Inspector of Factories.

"Old things need not be therefore true,
O brother men, nor yet the new;
Ah! still awhile the old thought retain,
And yet—consider it again."

—*A. H. Clough.*

Reprinted from "Public Health," the Official Organ of the Society of Medical Officers of Health.

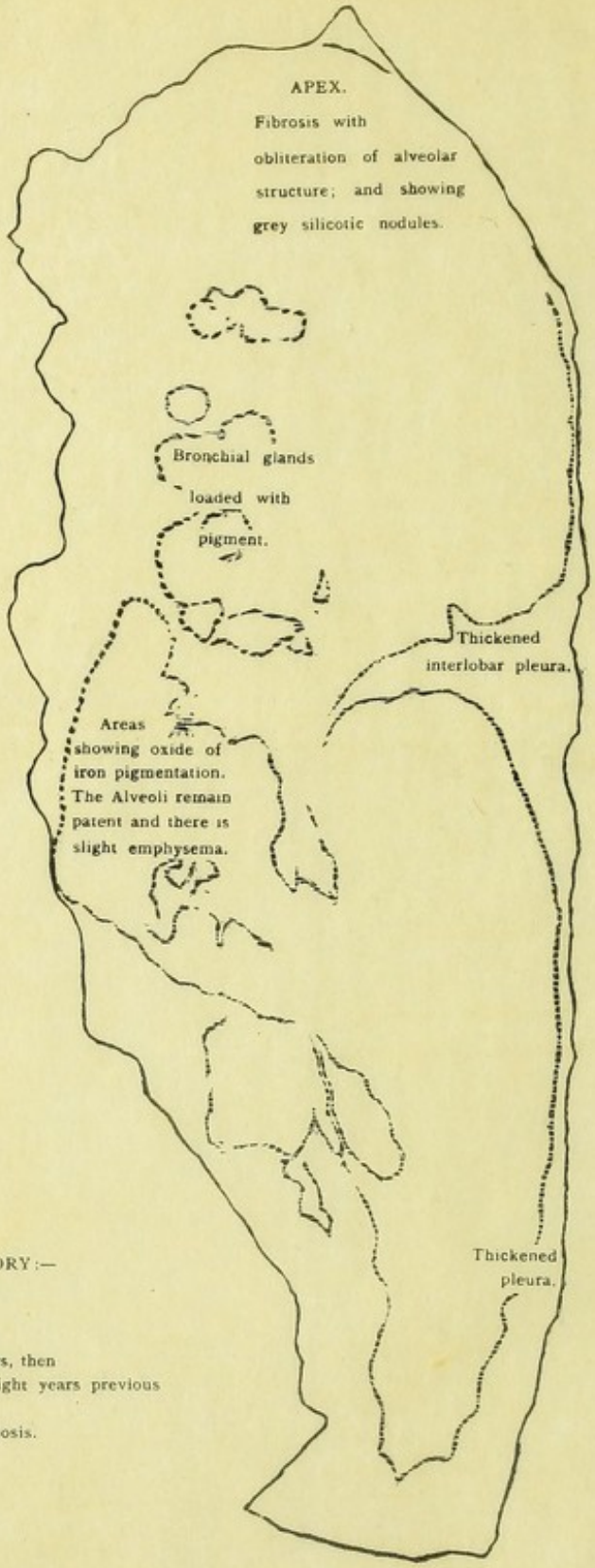


VERTICAL SECTION from Lung of Man who worked at IRONSTONE-MINING in England and GOLD-MINING in the Transvaal. *Natural Size and Natural Colour.*

OCCUPATIONAL HISTORY:—

(I) Ironstone Miner (England), then
(II) Goldminer (Transvaal), then
(III) Ironstone Miner (England), then
(IV) Goldminer (Transvaal) for 5 years, then
(V) Insurance Agent (England) for eight years previous to death.

Cause of Death:—Pulmonary Tuberculosis.



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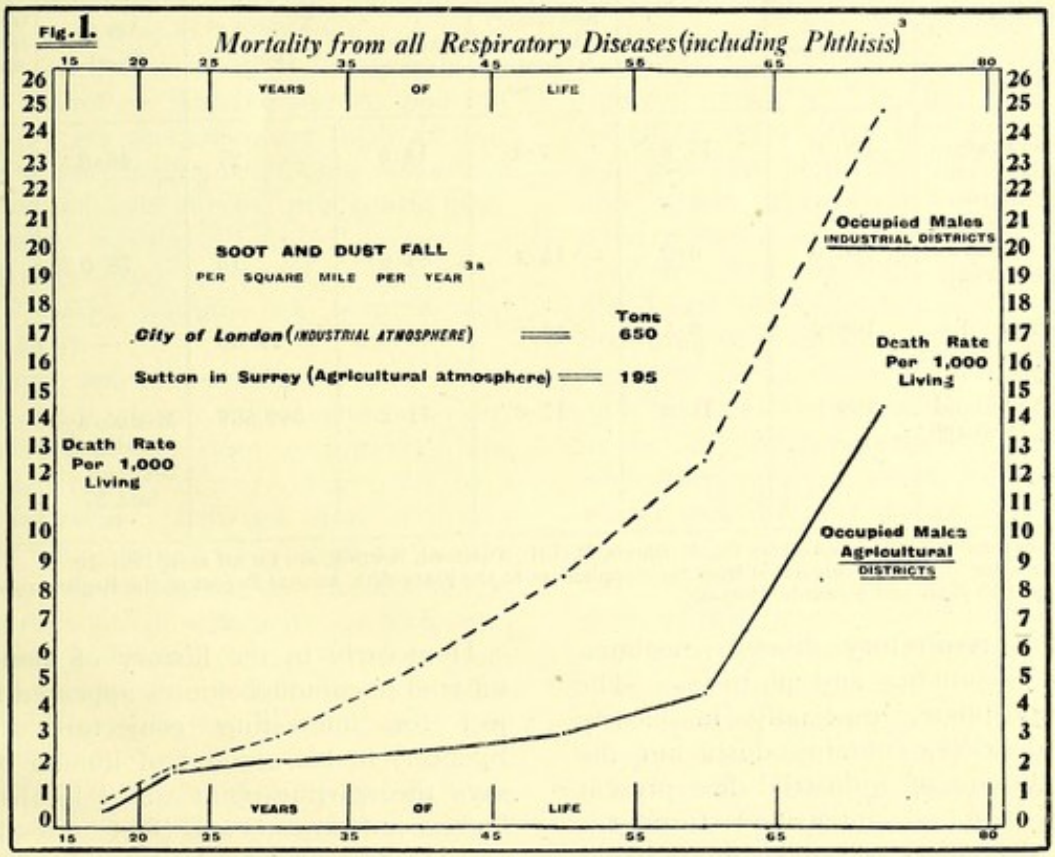
THE present time, when the recent report of the Royal Commission on Metalliferous Mines and Quarries (*1*), by drawing attention to some of the effects which follow upon the inhalation of dust, has stimulated inquiry into this subject, is opportune for reviewing our knowledge of dust diseases inherited from the past, and considering it again in the light of more recent investigations. The title chosen for the present course of lectures, Industrial Pneumonoconioses, or diseases of the lungs induced by dust inhaled during occupation, may seem somewhat tautologous, because the existence of pneumonoconioses (a term of Greek derivation, πνεύμων, lung, and κόνις, dust, introduced by Zenker) (*102*) apart from occupation is not generally recognised. The construction of our air passages, however, which are specially contrived to impede the entrance of dust; the amount of inorganic matter which nevertheless is found after death in our lungs—an amount which increases with age (*2*); and the excessive mortality from respiratory diseases experienced by dwellers in dusty atmospheres—an excess which increases with age (see fig. 1), and with the amount of dust present (see table 1); indicate



heaps of sand, that in running the knife through the pulmonary vesicles he thought he was cutting some sandy body"; and describes how stone-cutters "oftentimes suck in, by inspiration, the sharp, rough, and cornered small splinters or particles that fly off; so that they are usually troubled with a cough, and some of them turn asthmatic and consumptive" (*12*). The same author, quoting (*13*) from the Acta Hassniensia, describes pleurisy in a potter, "in whose dissected corps the right lobe of the lungs was found grown to the ribs and tending to a withered dryness and a phthisick; this indisposition of his lungs being attributed to the trade he had worked at"; and his description of the manufacture of earthenware, which is "first baked in a furnace, then covered with lead calcined, pounded with powdered flint and melted, and then put into the furnace again," shows that the potter of that day was exposed to the inhalation of flint dust, which has long been recognised in this country to be injurious. Thus a patent for grinding flints by a wet method granted in 1713 to Thomas Benson, of Newcastle-under-Lyme, states that previously flints were pounded dry, which process "proved very destructive to mankind insomuch that any person, ever so healthful and strong, working in that business, cannot possibly survive over two years, occasioned by the dust sucked into his body by the air he breathes" (*20*).

While these quotations display a recognition of a causal relationship between dust inhalation and consumption, that piquant observer, Ramazzini, who went personally to inspect the trades he speaks of, knew that some dusts, though injurious, did not tend to the development of consumption, for he tells (*14*) how there flies out of hemp and flax "a foul mischievous powder, that, entering the lungs by the mouth and throat, causes continual coughs, and gradually makes way for asthma"; and, after describing the process of combing silk cakes, he remarks (*14*) that "the poor people that comb these threads are usually troubled with a vehement cough, and a great difficulty of breathing, and few of them live to old age in that way of business. The virulence that gives rise to this tragedy is owing to the cadaverous particles of the silkworm that are mixed with these cakes." Note how he distinctly refuses to ascribe the trouble to silk dust (in which he is borne out by the much later work of Givre) (*27*), and also the omission of any reference to consumption in either of these trades. He is even clearer in speaking (*15*) of men who sift corn and who "are so plagued with this powder or dust, that when work is done they curse their trade with a thousand imprecations. The throat, the lungs, and the eyes sustain no small damage by it, for it stuffs and dries up the throat; it lines the pulmonary vessels with dusty matter that causes a dry and obstinate cough; . . . hence it is that almost all that live by that trade are short-breathed, and cachectick, and seldom live to be old; nay, they are apt to be seized with an orthopnœa, and at last with a dropsy." Surely an excellent clinical description of dust-bronchitis, emphysema, dilated heart, and failure of circulation.

These distinctions, implied or definitely stated, between the types of respiratory trouble which follow inhalation of different dusts, are the more notable, because even to-day pneumonoconioses are pigeon-holed in clinical teaching as a single entity, ascribed to exposure to any and every form of injurious dust, of which pulmonary fibrosis sums up the pathological findings and phthisis the morbid result.

Such generalisation has probably resulted from the publicity accorded to certain special inquiries into the prevalence of dust-phthisis, for example, that of Prof. Alison, who, when speaking (*21*) of "that modification of phthisis which occurs in middle and advanced life, . . . in those workmen who are much exposed to irritation of the lungs, particularly such as are in the constant habit of inhaling various fine powders into their lungs," says, "I have reason to believe that there is hardly an instance of a mason regularly employed in hewing stones in Edinburgh, living free from phthisical symptoms to the age of 50." The newer parts of Edinburgh were then being built out of Craigleith sandstone, the supply of which is now exhausted. This industrial disease was shown by Gulland to persist among Edinburgh masons in this century (*33*); and was deplored by that famous writer, geologist, and stone-mason, Hugh Miller,

who, when telling of his own narrow escape, writes (*22*) :—

"My general health, too, had become far from strong. As I had been almost entirely engaged in hewing for the two previous seasons the dust of the stone, inhaled at every breath, had exerted the usual weakening effects on the lungs — those effects under which the life of the stone-cutter is restricted to about forty-five years; but it was only now, when working day after day with wet feet in a water-logged ditch, that I began to be sensibly informed, by a dull pain in the chest, and a blood-stained mucoidal substance, expectorated with difficulty, that I had already caught harm from my employment, and that my term of life might fall short of the average one."

And later :—

"The dust of the stone which I had been hewing for the last two years had begun to affect my lungs, as they had been affected in the last autumn of my apprenticeship, but much more severely; and I was too palpably sinking in flesh and strength to render it safe for me to encounter the consequences of another season of hard work as a stone-cutter. From the stage of the malady at which I had already arrived, poor workmen, unable to do what I did, throw themselves loose from their employment, and sink in six or eight months into the grave—some at an earlier, some at a later period of life; but so general is the affection that few of our Edinburgh stone-cutters pass their fortieth year unscathed, and not one out of every fifty of their number ever reaches his forty-fifth year."

And again :—

"I remained for several months in delicate and somewhat precarious health. My lungs had received more serious injury than I had at first supposed; and it seemed at one time rather doubtful whether the severe mechanical irritation which had so fretted them that the air passages seemed overcharged with matter and stone dust, might not pass into the complaint it stimulated and become confirmed consumption."

That such generalisation, however, is not justified was claimed by Thackrah, who stated (*23*) that "dust of every kind irritates, but not in an equal degree"; and he quotes (*24*) the longevity of bricklayers and lime-workers, instancing the old adage, "bricklayers and plasterers' labourers, like asses, never die." He recognised that masons inhaling "particles of sand and dust which arise from chipping stone . . . are short-lived, dieing generally before they attain the age of forty" (*25*), and cites Patissier's account of phthisis among the stone-dressers of Saint-Roch (known as the disease of Saint-Roch) and Merat to support him; but he noted that "in the lead mines of the North of England, the men are injured by working ore in sandstone, but are sensible of no inconvenience when the ore is in limestone" (*26*). Thackrah also dwelt on the prevalence of phthisis among the metal grinders of Sheffield, and instances Dr. Knight's opinion that fork-grinding ought to be confined to criminals (*28*). A few years later Calvert Holland established statistically the sad phthisis mortality experienced by fork-grinders who worked on dry stones, "and the dust which is created, composed of fine particles of stone and metal, rises in clouds and pervades the atmosphere. The dust is thus every moment inhaled . . . and produces permanent disease of the lungs" (*29*). He portrays the lives of these workers, and then uses these vigorous words: "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. . . . 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" (*30*).

By this time the collection of national mortality statistics, inaugurated in 1832, was providing data for investigations; and Dr. Farr, the first superintendent of statistics, placed valuable information (*31*) as to the prevalence of respiratory diseases, and in particular of phthisis, among the tin-miners of Cornwall, and the lead-miners of the North of England, before the Royal Commissioners appointed in 1862 to inquire into the health of men employed in metalliferous mines. Dr. Peacock, physician to St. Thomas' Hospital, who, on account of the attention he had paid to respiratory diseases, and in particular because of his work on the phthisis mortality of millstone builders (*30*), a mortality ascribed by him to the dust generated in dressing buhrstone, was called in to assist these commissioners; and his careful report, a splendid clinical study still worth close attention, based on an examination of over 600 miners, together with evidence from local medical men,

established the existence of "miner's disease," thus giving the clue to the mortality figures of Dr. Farr. Peacock, in describing the disease (*32*) carefully distinguishes it from ordinary phthisis.

"The form of the disease in which there is local consolidation in some portion of the lungs, bears a close general resemblance to true consumption, and especially where, as often happens, the voice is husky and the patient expectorates blood. There are, however, features by which it is sufficiently distinguished from that disease. It usually occurs in persons who do not present any hereditary disposition to phthisis—their parents and other relatives often having attained advanced ages and being quite healthy. It commences at a later period of life than phthisis, indeed, in persons who have reached ages at which consumption is by no means of frequent occurrence. It is also much slower and less active in its progress, so that in persons who have been ill for several years the signs often do not indicate extensive or advanced disease. The quickness of pulse, the rapid and extreme emaciation, and the night perspirations so characteristic of true phthisis, are also generally absent or only slightly marked, and there is rarely diarrhœa, indeed, the bowels are often obstinately confined."

As a source of information the Report of the Commissioners is invaluable, but, notwithstanding the evidence of several witnesses, particularly the miners themselves, that the "stour" or dust was far worse than anything else they had to contend with, the conclusion arrived at, based, I may say, on practically unanimous medical opinion, that the influence of dust was subsidiary to the many other adverse conditions of ventilation, exposure to fumes of explosives, and variations of temperature, which at that time surrounded the mining industry, was unfortunate. At that time, however, all researches into the causation of phthisis, for example, the writings of Alison (*21*) and of Chateauneuf (*9*), were proving the importance of bad housing, poverty, and lack of ventilation, and, although the prevalence of phthisis in certain dusty industries was noted, the absence of the disease in other dusty industries obscured the issue, and Peacock, influenced by the thought of his day, may be forgiven for ascribing the prevalence of the disease to the imperfect hygienic conditions he saw, rather than to the then disputed point of dust inhalation. Fifty years later a Departmental Committee, of which Dr. J. S. Haldane was a member, was appointed in 1902 to reinvestigate the causation of the still persistent high phthisis mortality among Cornish tin-miners; and this committee brushed aside every other influence except dust inhalation, deciding (*34*) that—

"So far as the Cornish miners are concerned it seems evident enough that stone-dust which they inhale produces permanent injury of the lungs—gradually in the case of ordinary miners, and rapidly in the case of machine-drill men—and that this injury, while it is apparently capable of gradually producing by itself great impairment of the respiratory functions, and indirectly of the general health, also predisposes enormously to tuberculosis of the lungs, so that a large proportion of miners die from tubercular phthisis. That the primary injury to the lungs is due solely to inhalation of stone-dust would seem to be practically certain."

In the decade preceding the work of the Mines' Commissioners of 1862, organised study of public health had commenced; and we find H. Headlam Greenhow appointed to lecture on public health at St. Thomas' Hospital. In the elaborate statistical inquiry he carried out in preparing his lectures he immediately found that "one of the most evident facts brought to light . . . is the influence of occupation on health." With this Finlaison's conclusion, arrived at (*51*) in 1853, is in close accord, that "the real practical difference in the distribution of sickness seems to turn upon the amount of the expenditure of the physical force. The density of aggregation, described under terms of city, town, or rural districts, seems to exercise little or no real influence"; and this fundamental point was restated (*52*) in 1903 by Watson, who said, "The proportion of members sick during any year varies with occupation." Greenhow's investigation proved of such importance that Dr., later Sir, John Simon, then medical officer to the General Board of Health, brought it to the attention of the Board, by whom it was published. Shortly after Simon became medical officer to the Privy Council, and he entrusted to Greenhow the duty of pursuing the subject further by visiting the great industrial centres. The reports which followed in 1861

(*38*) and 1862 (*39*) have formed a mine of information from which later writers have freely drawn; they are the first example of State medical inspection of factories, and are a monument to Simon's sagacity in grasping the great influence exerted by occupation upon health, and to Greenhow's keen insight and powers of observation. Throughout these reports runs as a theme the influence of dust inhalation in causing pulmonary disease, whether among lead-miners in Yorkshire, tin-miners in Cornwall, needle-pointers in Alcester, cotton operatives in Lancashire, flax-hecklers in Pateley Bridge, metal grinders in Birmingham and in Sheffield, coal-miners in South Staffordshire and in South Wales, or stone-dressers in Stroud. Why work so well started was then allowed to lie dormant for so long, while other aspects of public health were being strenuously developed by medical officers of health with inspectors of nuisances appointed for every town and district, reinforced now by a battalion of tuberculosis officers, is astonishing.

Stimulated by what he had seen in industrial centres Greenhow obtained specimens of workers' lungs, and between 1860 and 1870 from time to time described the conditions he found before the Pathological Society, and his reports in the Transactions of those years have been relied upon by subsequent workers to establish the pathology of pneumonoconioses. The specimens themselves are still preserved in the museum of the Middlesex Hospital. At this time Virchow, Zenker, Knauff and other Continental workers were also dealing with the subject, but there is no record of personal inspection of industries like Greenhow's.

The period of Pasteur's marvellous work now followed, and, arising from it, Lister's methods of antiseptic surgery, and the discovery by Koch in 1882 of the tubercle bacillus. Soon after this Arnold (*95*) published a comprehensive monograph on the subject of dust inhalation, in which he pointed out that dust particles may be found in the liver, spleen, and bone marrow, as well as in the lungs; but for the moment the germ causation of disease diverted attention from the influence of dust: yet ten years later Arlidge wrote (*40*): "I doubt if these bacilli actually develop phthisis, unless there be some antecedent change in the vitality of the affected tissue; a change wrought by depressing causes connected with the mode of life, or with constitutional debility and inherited taint, or with occupation followed; of which contributory factors two or more may co-operate. And assuredly the breathing of dust may be reckoned as one such of no slight energy." The painstaking observations of this authority, however, and the productive labours of Oliver, need no mention here; they form the basis of our present-day knowledge, and are to be found in every text-book on the subject.

The researches just summarised compel us to recognise that respiratory diseases are influenced by dust inhalation; but some plan must be adopted in dealing further with the subject. Classification has been attempted by Hirt (*74*), Arlidge (*40*), and others on various plans, based usually on consideration of dusts, either by origin, animal, vegetable, or mineral (first suggested by Chateauneuf) (*9*), or by physical properties, hardness, sharpness, or the like; but such classifications have brought dusts which produce different pathological results into the same class. Recently, however, Heim and Agasse-Lafont have suggested (*41*) that the effects caused should be the basis of classification, and I intend adopting this suggestion, and considering first the relation of each of the main respiratory diseases to dusts, and then from that standpoint examining the properties of the dusts which are associated with an undue prevalence of each of these diseases.

Without, however, anticipating subsequent consideration of these properties, *size* may here be referred to. Dust particles to be inhaled must be sufficiently small to remain suspended in moving air, and so be carried into the air passages. Virchow, in the middle of last century, for a time doubted whether inhaled dust gains access to the lungs, and maintained that the pigment found in the lungs of city dwellers was derived from blood pigment, and not from inhaled carbon; but the work of Knauff (*35*), who, after exposing dogs to fumes of a smoky lamp for periods varying from one day to three months, showed that carbon was found in the lungs in amount varying directly with the length of exposure, and of Zenker, who demonstrated (*103*) the

presence of excessive amounts of oxide of iron in the reddened lungs of factory workers exposed to the fine powder of rouge, which is composed of this material, in making gold-leaf and in polishing mirrors, finally convinced him that inhaled dust reaches and is deposited in the lungs. [A portion of one of Zenker's original specimens is in the museum of St. Thomas' Hospital (No. 1840A); and there is in the Home Office collection, through the courtesy of Dr. J. S. Haldane, an even more convincing specimen taken from a man who worked in ironstone mines in this country, then went to the Transvaal gold-mines, and finally returned home to the ironstone mines. The period of gold-mining is indicated by the presence of grey fibrous tissue, and that of ironstone mining by bright red discoloration (*see frontispiece*).] Possibly a few particles, as suggested by Calmette (*42*), may reach the lungs through the lymphatic circulation after first entering the digestive tract; but no one has yet produced a condition suggestive of pneumonoconiosis by feeding animals on dust, and this channel of entry, as has been pointed out by Oliver (*94*), may be neglected for practical purposes; while Goadby's experiments (*43*) which show that lead poisoning is caused one hundred times more easily by inhaling lead dust than by eating it, have demonstrated that this toxic dust is chiefly dangerous because it definitely reaches and is absorbed by the lungs; and, further, W. Watkins-Pitchford's remarks on Transvaal gold-miners that "the lymph nodes in the gastro-hepatic and gastro-splenic omenta, and the mesenteric and retroperitoneal nodes are usually of normal appearance in fatal cases of silicosis" (*44*) and that he was unable to detect siliceous particles in them, conclusively show that the digestive tract is not the usual path of ingress.

Dust particles inhaled into the air passages may be divided into those which are too large to pass through the finer bronchioles and enter the alveoli, and those which are so small that they can pass through these passages; and upon this simple point may possibly depend differences found to exist between different dusts and the types of respiratory disease associated with their inhalation. The larger particles become entangled in mucus and are swept away by ciliary action; the smaller ones when they reach the alveoli are removed and carried into the lung tissue by phagocytes, probably through the pseudostomata. How these fine particles are carried into the alveoli is not quite clear. Lister, noticing that "in simple fracture of the ribs, if the lung be punctured by a fragment, the blood effused in the pleural cavity, though freely mixed with air, undergoes no decomposition" (*45*), concluded that inspired air must be "filtered of germs by the air passages, one of whose offices is to arrest particles of dust, and prevent them entering the air-cells"; and Tyndall, working with a beam of light, showed (*45a*) that the deeper air of the lungs is optically dark, *i.e.*, free from suspended particles of even ultra-microscopic size. Moreover, if aeration of the blood is carried on by diffusion of gases between the residual air of the alveoli and the inspired air, dust particles can hardly be carried into the alveoli by such diffusion; even though, as Watkins-Pitchford points out, "the alveolar sacs, being surrounded by elastic fibres, undergo a passive dilation and contraction synchronously with the movements of the thorax, and we conclude therefore that this process of 'diffusion' must at least be materially assisted by the rhythmical variations in the capacity of the air sacs. Some physiologists, however, have pointed out that, as the capacity of the trachea, bronchi, and bronchioles is about 140 c.c., and as about 500 c.c. of air pass in and out of the chest during each ordinary respiratory cycle, some 360 c.c. of this tidal air must flow directly into and out of the air sacs—in other words that some of the tidal air must strike right down to the alveoli at each ordinary inspiration. . . Quite large particles of siliceous mineral are often found in the pulmonary alveoli in early cases of miners' phthisis. Their size is often so great as to render it extremely improbable that they have escaped into the alveolus from the lung tissue, for the particles in the lung tissue are practically all of very small dimensions" (*44*). Probably we are at any rate justified in considering that during deep breaths, preliminary to a cough or sneeze, stimulated by the necessity for expelling dust-laden mucus, inspired air may carry with it

into the alveoli particles of dust sufficiently small to pass through the smallest bronchioles. No particles have been found (*46*) in the lung tissue, which exceed 10 μ in diameter, and these could have entered in this way; and definite evidence is wanting to show that particles ever pass into the lung tissue through the walls of the bronchi or larger bronchioles.

One further point, *solubility*. — Only particles which are insoluble in the fluids of the body when carried into the air passages remain as foreign bodies either to stimulate the ciliated epithelium to overaction for their expulsion, or, if they gain access to the lymph channels, to give rise to a proliferation of connective tissue; thus dusts of ivory, horn, bone, and other animal structures, and of calcium sulphate (plaster-of-Paris and alabaster), of limestone, and of oxide of iron are not associated with pneumonoconioses in the way that dusts of vegetable husks, emery, glass, sandstone and flint are. Generally speaking dusts are more injurious as their chemical composition differs from that of the human body, or from the elements of which the body is normally composed. Other distinctive properties of injurious dust will be referred to when the special respiratory diseases to which they give rise are discussed.

ASTHMA.

Asthma is a term used to indicate a train of symptoms rather than a disease, and *per se* seldom appears as a cause of death on death certificates; therefore, mortality statistics give no indication of its prevalence, indeed, asthma does not appear in the three years, 1910-12, as a cause of death among 195 deaths of cotton-strippers, who are known to suffer markedly from this complaint. That asthma occurs as a result of dust inhalation has already been mentioned. Agricola, in the quotation given (*18*), referred to its occurrence as the result of dust inhalation among Carpathian miners; Ramazzini refers (*14*) to it as resulting from the inhalation of flax and hemp dust; Diembroek mentions (*12*) its occurrence among stone-masons; and later inquiries have confirmed these earlier observations. Thus all writers upon the health of metalliferous miners describe the prevalence of attacks of shortness of breath, which they call asthma. Greenhow, when dealing with Cornish tin-miners, mentioned (*38a*) that "for the most part they become more or less asthmatical about the age of forty." Greenhow, however, it is only fair to say, remarks (*39a*) "the terms 'asthma' and 'asthmatical' are commonly used by various classes of operatives to designate any form of pulmonary disease attended by dyspnœa arising from their occupation. These terms are therefore employed . . . in their popular and not strictly in their pathological sense." Similarly the Committee on the Health of Cornish Miners noted (*34a*) among tin-miners that "shortness of breath appears to be almost always the first prominent symptom." Greenhow also said (*38b*) of lead-miners "as life advances, dyspnœa is added to the other symptoms, and at length most of the miners become asthmatical, are unable to move without more or less difficulty of breathing, and suffer habitually from cough and expectoration. . . The age at which miners for the most part become decidedly asthmatical is about the 45th year"; and a retired lead-miner recently told me that asthma still persists in the Durham lead mines. Cumpston (*48*) and Summons (*49*) speak of asthma among Australian gold-miners; and it is referred to by Watkins-Pitchford (*44*) and in all the numerous reports on Miners' Phthisis in the Transvaal. Arlidge states that "flint-millers . . .suffer sadly with asthma and interstitial pneumonia" (*40a*); and the same authority says "the features of potters are rather those of asthmatical subjects" (*40b*).

Personally I have not found asthma prominent either among grinders of metal in Sheffield or elsewhere, or among Aberdeen granite cutters, or among silica-brick makers, and, although some stone-masons have spoken of it, I have not found that asthma (as distinguished from attacks of air hunger) is common among them, although all suffer in excess from dust-phthisis; and West, when speaking (*47*) of the extreme shortness of breath exhibited by these workers, says "this is often spoken of loosely as asthma, but true spasmodic asthma is by no means common." On the other hand operatives exposed to certain dusts do suffer in a marked degree from true asthma; and I think that attacks of

dyspnœa, which occur in cases of dust-phthisis, are essentially different in causation and character from such cases of asthma. True dust-asthma, which I have seen in a pronounced form among cotton strippers, is associated with a physical configuration of the chest, and a type of breathing quite different from those seen among operatives who experience a heavy mortality from dust-phthisis. The course of the disease was far more distressing and more liable to invalidate the sufferer than the dyspnœa described among gold-miners and others.

Cotton-strippers are exposed, at one of the first processes of cotton spinning, to dust arising from cotton husk and débris which is thrown in a fine cloud into the air when the cylinders of cotton carding-machines are brushed out or "stripped." The amount of dust generated varies with the grade of cotton used, and is greatest from coarser grades, particularly from Surat and some American cottons, and least from finer grades, such as Egyptian. Each spinning mill usually deals only with material of special grades, so the effect of the different amounts of dust could be studied at different mills. Thus I found (*53*) among men working on coarse grades of cotton 91 per cent. more or less affected, on medium grades 72 per cent. and on fine grades 62 per cent.

Exposure to this dust has unpleasant effects for a chance visitor, who, not infrequently, suffers within twelve hours from an attack of mill fever, with sharp but transient febrile symptoms, but operatives soon become inured to such attacks. An operative stripping cotton-carding machines, after years of work, varying from five to twenty, according to idiosyncrasy and the amount of dust, finds his chest becoming affected; at first the only symptom is difficulty in breathing on Monday morning, or after any interval away from the dust, and after the first day is over he may remain unaffected for the rest of the week. As time passes this difficulty becomes worse and extends later and later into the week, developing into a typical form of asthma as long as he is in the dusty atmosphere. At the same time he loses flesh and becomes thin in face and body. As the case develops the action of the diaphragm becomes less and less effective, until the only action of this great respiratory muscle is to fix the lower ribs. At the same time the superior intercostal muscles and the extraordinary muscles of respiration are more and more called into play to carry on the ordinary act of breathing. The sternum becomes more prominent and the chest barrel-shaped. Meanwhile the extra tax thrown on the lungs leads to some degree of emphysema. There is little or no sputum produced, and what little there is, is expectorated with difficulty. When this stage is reached, *i.e.*, after about twenty years' work, the individual is usually compelled to seek other employment, and so, although the employment is not arduous, few old men are found at work. I found only 139 aged 45 years and over in every 1,000, as compared with 231 metal-grinders and 434 lace-workers. The distinctive symptoms experienced are attacks of spasmodic asthma precipitated by exposure to dust. The trouble is, however, rapidly disappearing owing to improved methods of dust prevention, which are even enabling affected men to resume their employment. These operatives do not suffer markedly from phthisis, though there is some slight excess late in life; but they experience a high mortality from pneumonia, and an excessive mortality from bronchitis.

Other cotton-operatives, weavers, occasionally suffer from an acute form of spasmodic cough with asthmatic symptoms, but this affection I believe (*90*) to be due to inhalation of a special mildew which from time to time appears on cotton thread; this disease, however, since it is probably a form of aspergillosis, does fall within the definition previously laid down of a pneumonoconiosis, and only calls here for passing mention.

Greenhow had previously noted (*38c* & *39b*) that carding-room operatives, especially those who strip the cards and the card-grinders, were very apt to become asthmatical about middle life in proportion to the amount of dust generated which varied with the grades of cotton used; but at that time the dust, owing to hand-stripping, was not generated so freely as when I made my inquiry, and Greenhow gives no clinical description of the disease. He did, however, describe (*38d*) the same condition among flax-hecklers at Pateley Bridge, where he

found at one factory 23 out of 27 hecklers habitually asthmatical; he speaks of the older men as short-breathed, with "rounded shoulders, emaciated frames, prominent eyes, and laborious wheezing respiration," and says that to find men aged 60 years at work must be deemed exceptional; and the following passage may be compared with the description given above of cotton-strippers' asthma (which was written without any knowledge of Greenhow's previous work):—

> The effect on a healthy stranger of entering these rooms is most unpleasant: the dust floating in the atmosphere irritates the nasal and bronchial passages, producing sneezing and a sense of oppression in the chest, which do not cease till some time after the visitor has left the apartment. Although the effects of temporarily inhaling the atmosphere of the dusty departments of a flax mill are so obvious, the operatives, after a time, become inured to it, and are able to tolerate it for some time without sensible injury. It was stated that the operatives are more affected by the dust at the beginning of the week, and that they always suffer more on resuming their employment after an interval of cessation. . . . As life advances, the power of resisting the pernicious influence of their occupation diminishes, and more or less of permanent dyspnœa, and other results of bronchial irritation supervene. The ordinary signs of chronic bronchitis, and sometimes of emphysema, may be detected by auscultation.

The observations published in 1873 by Dr. Purdon, of Belfast (*61*), and the experiences of 14 observers, collected in 1894 by Osborn (*50*), corroborate this description; for instance, Dr. Lunan, Blairgowrie, stated: "I cannot say I have been able to trace the origin of phthisis to flax dust. I feel quite sure that bronchial catarrh, accompanied in many cases by asthma, is frequently caused by breathing the dusty atmosphere of flax mills"; and Dr. D'Evelyn, Ballymena, said "the hacklers all die young, and all suffer from chronic disease of the lungs, caused by the flax dust. . . . The first thing a hackler does each morning is to drink a glass of raw whiskey, to clear out his bronchial tubes, otherwise he is unable to breathe. After a day off, the men have often told me their breathing is worse." Notwithstanding the then accepted axiom that all forms of dust must predispose to phthisis, five out of the fourteen observers definitely state that flax dust does not have this effect; similarly Givre, speaking of carders of silk, says (*27a*) "some authorities even deny that these dusts create a predisposition to phthisis; and see a certain antagonism between the emphysema of carders and phthisis."

Inquiries in the flax and jute industries have convinced me that similar asthma still occurs in these industries among operatives exposed to dust generated in the initial stages of textile manufacture—at flax-heckling and at jute-opening—but the symptoms, at least under modern methods of dust removal, are seldom pronounced.

When once this asthmatical condition, which is especially associated with exposure to vegetable husk, has been recognised, its occurrence in a modified form can be traced among those exposed to dusts of similar origin, such as those arising from wood bark and some of the harder woods—for instance, Powell and Hartley describe (*75*) the case of a man exposed at his work to the dust of rosewood; this man was subject to severe attacks of dyspnœa, which had the paroxysmal character peculiar to asthma; his symptoms ceased after absence from work, but returned when he resumed his employment which he finally had to give up. True, dust asthma is always associated with an excessive incidence of bronchitis. Thus 32, or 16.4 per cent., of the 195 deaths already referred to among cotton-strippers were due to this cause, as compared with 8.3 per cent. for Occupied and Retired Males.

The distinctive features of this form of asthma are (i.) occurrence of asthmatic attacks during exposure to dust, and the cessation of attacks on leaving the dusty atmosphere; (ii.) immobility of the diaphragm; and (iii.) over-action of the superior intercostal and extraordinary muscles of respiration. Contrast these with Peacock's description of asthma among Cornish tin-miners with their high mortality from dust-phthisis—"the difficulty of breathing is extreme, and is aggravated by any slight exertion, as by ascending stairs or any slight elevation, and it is usually increased by certain states of the atmosphere and when the patient takes cold. Notwithstanding the

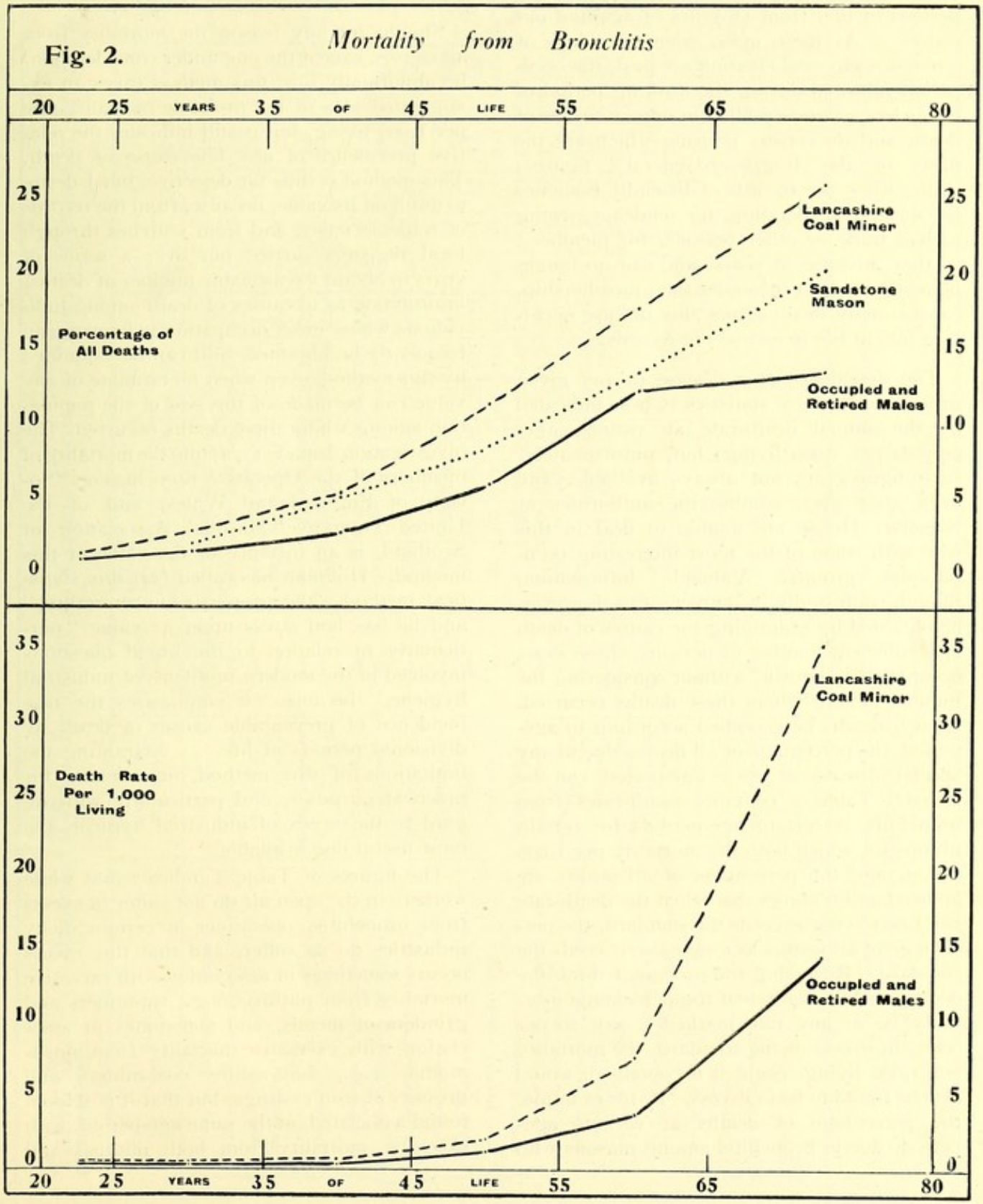
great effort which is made in breathing the movements of the chest are most imperfect, there is little change of capacity with inspiratory and expiratory acts, and the respiration is almost entirely abdominal." This type of breathing I have seen among grinders of metals in Sheffield and elsewhere, and among stone-masons, and it has been described by the Committee on the Health of Cornish Miners (*34*) and by all observers of miners' phthisis on the goldfields of the Transvaal and of Australia.

Broadly speaking, then, there are two distinct types of dyspnœa caused by dust. (1) One type, seen among operatives who suffer from dust-phthisis, in which convulsive but ineffective respiration is carried on by the diaphragm; and Watkins-Pitchford adequately explains (*44*) the condition as follows: "The phenomenon is quite independent of pleuritic adhesions, and is due to the gradual loss by the lungs of their elasticity—or, in other words, their capacity of accommodating themselves to the varying capacity of the thorax during respiratory movements. As soon as the lungs have ceased to be distensible, the chest wall becomes fixed; for as the lungs are now incapable of following the chest wall, its outward movement is necessarily opposed by the overpowering pressure of the atmosphere." (2) Another type seen among operatives who do not suffer from dust-phthisis, but who do suffer from bronchitis, in which the diaphragm is fixed and the ordinary type of asthmatic breathing is exhibited. Here the underlying pathological condition is different—the elasticity of the air vesicles is not seriously impaired, but the bronchial mucosa is chronically inflamed and ready, when dust falls on it, to start excessive respiratory impulses. Hence it follows that this form of asthma is provoked by exposure to dust and is of peripheral origin; while the other form is provoked by exertion and is a form of air hunger of central origin, indeed in advanced cases chronic cyanosis is present.

Fifty years ago asthma was common among coal-miners. "Then in every little mining village," writes the secretary of a miners' association, "there was a contingent of old miners, past work, on account of difficulty of breathing, a stage usually reached between forty and fifty years of age"; and this statement is borne out by Greenhow, who tells (*39c*) of the ravages wrought in his day among coal-miners in South Staffordshire by asthma; "some miners retain their health till an advanced period of life, but the greater number suffer, more or less, from asthmatical symptoms before attaining the age of 50, and many break down and are disabled at 40 to 50 years of age . . . a miner is usually an old man at 50; and few men . . . are found at work beyond that age." The same observer also mentioned (*39d*) the disease as prevalent in the South Wales coal-field, and while attributing it to bad ventilation of the mines, states that the miners blamed the fumes from explosives. He contrasts (*39e*) the condition with that seen among lead-miners and tin-miners, as one with emphysema more frequent, with less pneumonia, developing later in life, and, while associated with chronic bronchitis, not associated at an early period of life with slight dyspnœa. In America Drs. Wainwright and Nichols report (*88*) that, at Scranton, asthma accounted, 1894—1904, for 7 per cent. of all deaths amongst coal-miners, as compared with 1.6 per cent. amongst all occupied males; and these observers state that the condition as seen among the miners is one of chronic bronchitis and emphysema. Synchronous, however, with improved ventilation of mines the disease has disappeared in this country: improved ventilation has minimised exposure to several adverse influences, *e.g.*, fumes from explosives, soot and other products of combustion from naked lights, gaseous impurities, and dust. The opportunity of ascertaining which was the determining cause has gone, and also of obtaining a good clinical description of the condition; but the following note sent me by Dr. J. Taylor, Chester-le-Street, taken in conjunction with Greenhow's observations, suggests that coal-miners' asthma was true spasmodic asthma. Dr. Taylor writes: "One case only do I recollect. In this case the breathing was carried on by the respiratory muscles of the chest, and the diaphragm did not enter much into the mechanism of inhalation. This patient suffered at all times of the year, but a windy day always appeared

to be the starting point for an attack." If the supposition is correct that coal-miners' asthma was true asthma, possibly the miners were correct in ascribing it to fumes from explosives, which, as Oliver has pointed out (*89*), would contain nitrous fumes; and in this case the disease should not be classed as a pneumonoconiosis. The disease, however, common though it used to be, has passed almost unobserved from our midst, and conjecture as to its character and causation are idle.

Fig. 2. *Mortality from Bronchitis*



BRONCHITIS.

Prevalence.—The study of the prevalence of this disease in occupations, as indicated by mortality statistics, presents certain difficulties, because the disease is most prevalent in the general population at the later age-periods of life, from 55 years of age and onwards. At these ages, when the days of strenuous physical exertion are past, the occupation followed during the working period of life is less accurately filled in on certificates of death and on census returns, which are the basis of the Registrar-General's figures; while, when the records of Friendly Societies are under consideration, the tendency, owing to bad trade or other reasons, for members, as they advance in years, and can no longer be readmitted, to surrender their membership, causes many deaths from this disease occurring late in life to escape the records.

The prevalence of a disease in any given group of mortality statistics is best indicated by the annual death-rate at various age-periods per 1,000 living; but, unfortunately, such figures are not always available, and even after great trouble the authorities at Somerset House are unable to deal in this way with some of the most interesting occupational groups. Valuable information, though confessedly incomplete, can, however, be obtained by examining the causes of death of a sufficient number of persons whose exact occupation is known, without considering the number among whom these deaths occurred. If such deaths be classified according to age-period, the percentage of all deaths due to any special disease at each age-period can be stated. Table 3 contains death-rates from bronchitis at certain age-periods for certain groups for which both the mortality per 1,000 living, and the percentage of all deaths are known; and it shows that when the death-rate per 1,000 living exceeds the standard, the percentage of all deaths as a rule also exceeds the standard. Reversing the process, I think the deduction fair that when the percentage mortality is, at any rate markedly, say 20 per cent., in excess of the standard, the mortality per 1,000 living, could it be obtained, would also be found to be in excess. If, for example, the percentage of deaths at various age-periods due to bronchitis among masons who worked on sandstone is taken and compared with that for Lancashire coal-miners, a similarity is present which suggests that the mortality per 1,000 living, could it be obtained for sandstone masons, would, like the mortality per 1,000 for Lancashire coal-miners, be found to be in excess (see fig. 2).

Should for any reason the mortality from all causes, except the one under consideration, be abnormally low, this method gives an exaggerated idea of the probable mortality-rate per 1,000 living, but it still indicates the relative prevalence of any one cause of death. This method is thus far defective, but I desire to dwell on its value, because, from the records of trade societies, and from searches through local registers carried out over a series of years to obtain a reasonable number of deaths, information as to causes of death among individuals whose exact occupation is known, can frequently be obtained, and can be examined by this method, even when no estimate of any value can be made of the size of the population among whom these deaths occurred. The investigation I made (77*d*) into the mortality of members of the Operative Stonemasons' Society of England and Wales, and of the United Operative Masons' Association of Scotland, is an instance of the value of this method. Hoffman has called (54) this statistical method "the proportionate mortality," and he has laid stress upon its value "particularly in relation to the broad questions involved in the modern problems of industrial hygiene," because "it emphasises the true incidence of preventable causes of death by divisional periods of life. . . . Granting the limitations of this method, it remains for practical purposes, and particularly with regard to the needs of industrial hygiene, the most useful one available."

The figures of Table 3 indicate that while workers in the open air do not suffer in excess from bronchitis, operatives in certain dusty industries do so suffer, and that this excess occurs sometimes in association with excessive mortality from phthisis, *e.g.*, tin-miners and grinders of metals, and sometimes in association with excessive mortality from pneumonia, *e.g.*, Lancashire coal-miners and dressers of iron castings, but that it is seldom found associated, at the same age-period, with excessive mortality from both phthisis and

Table 3. *Death-Rates from Bronchitis for Certain Classes of Males distributed in Age-Periods.*

	CLASS	EXPOSURE TO DUST	PERIOD UNDER REVIEW	TOTAL NUMBER OF DEATHS FROM BRONCHITIS	PER 1,000 LIVING AT AGE PERIOD						PERCENTAGE OF ALL DEATHS AT AGE PERIOD						PREVALENCE OF PHTHISIS	PREVALENCE OF PNEUMONIA	MEDIAN AGE AT DEATH FROM ALL CAUSES
					20–	25–	35–	45–	55–	65 AND OVER	20–	25–	35–	45–	55–	65 AND OVER			
ENGLAND AND WALES *	Occupied and Retired Males	——	1900–1902	40,223	0·03	0·08	0·22	1·06	3·62	14·07	0·6	1·2	2·5	5·7	10·2	13·2	Standard	Standard	57 to 58
	Agriculturist Agricultural Districts	Country Air	do.	1,509	0·00	0·03	0·07	0·22	0·95	8·43	0·0	0·7	1·3	2·4	5·3	10·7	Low	Low	over 67
	Fisherman	Seaside Air	do.	71	0·00	0·06	0·20	0·36	1·64	9·17	0·0	0·7	1·6	2·4	6·0	9·1	Low	Low	59
	Shipbuilding	Outdoor Industrial	do.	292	0·00	0·03	0·23	0·75	3·04	11·57	0·0	0·7	2·6	5·2	9·8	13·0	Low	Normal	58 to 59
	Tailor	Indoor Industrial	do.	697	0·07	0·06	0·21	1·18	3·88	15·22	1·6	1·0	1·9	5·9	10·4	13·6	In Excess	Low	61 to 62
	Shoemaker	do.	do.	1,182	0·05	0·11	0·32	0·99	3·65	15·12	1·0	1·6	3·1	5·3	10·7	14·1	In Excess	Low	62 to 63
	Coal Miners: Durham & Northumberland	Coal only	do.	255	0·05	0·05	0·14	0·47	3·06	18·82	1·0	1·1	2·2	3·4	9·7	12·1	Low	Low	54
	Coal Miners: Nottingham & Derbyshire	Mainly Coal	do.	170	0·00	0·06	0·17	0·42	3·94	34·62	0·0	1·7	2·8	3·8	12·6	23·2	Low	Low	54 to 55
	Coal Miners: Monmouth & South Wales	Coal, Shale etc.	do.	435	0·01	0·07	0·20	1·32	8·15	24·06	0·3	1·2	2·3	8·3	20·5	22·8	Low	In Excess	47 to 48
	Coal Miners: Lancashire	Coal, Shale etc.	do.	296	0·05	0·16	0·46	1·94	7·44	34·96	1·0	2·6	5·1	1·1	17·5	25·6	Low	In Excess	47 to 48
	Brick Plain Tile Terra Cotta Maker	Clay	do.	172	0·00	0·05	0·30	1·08	2·52	14·98	0·0	1·2	4·4	8·4	10·9	15·4	Normal	Normal	58 to 59
	Wool Worsted Manufacture	Wool débris	do.	385	0·03	0·18	0·16	0·95	3·59	19·33	0·6	3·4	1·7	5·3	8·8	13·1	slight excess	Low	61 to 62
	Cotton Manufacture	Cotton debris	do.	704	0·05	0·07	0·29	1·68	6·16	23·29	1·1	1·2	2·9	8·0	13·1	15·7	Normal	In Excess	55 to 56
	Sawyer & Wood Turner Cooper etc.	Sawdust	do.	377	0·07	0·09	0·46	1·52	4·22	17·97	1·8	1·7	4·8	7·2	11·7	15·0	In Excess	Some excess	60 to 61
	Cutler Scissors Maker	Metal, Quartz, Bone, Emery etc.	do.	126	0·00	0·00	1·00	2·87	7·21	23·07	0·0	0·0	5·7	8·9	13·0	17·1	In Excess	Some excess	56 to 57
	Potter Earthenware etc. Manufacture	Clay and Flint	do.	278	0·00	0·10	1·29	5·58	14·84	27·62	0·0	1·9	8·6	17·2	25·2	20·5	In Excess	slight excess	52
	Tin Miner	Quartz	do.	66	0·00	0·44	0·86	3·57	13·00	32·45	0·0	3·3	3·2	9·3	18·9	20·6	In Excess	Low	54 to 55
	Dressers of Iron Castings	Sand, Clay, Emery, Metal, Charcoal	1893–1912	52	—	—	—	—	—	—	0·0	0·0	4·0	10·3	23·5	23·6	Low	In Excess	49
§	Sheffield: Grinders	Metal & Quartz	1908–1912	56	—	—	—	—	—	—	?	1·6	2·2	8·1	15·4	26·0	In Excess	Low	49
§	Sheffield: Cutlers	Metal, Bone, Emery Linen, etc.	do.	63	—	—	—	—	—	—	?	2·9	3·8	5·3	12·9	16·6	In Excess	Normal	59 to 60
† ENGLAND AND WALES	Stone Getter — Sandstone Dresser	Quartz	1910–1912	291	—	—	—	—	—	—	0·0	1·7	4·9	7·3	13·1	20·0	Excess	Low	56 to 57
† ENGLAND AND WALES	Mason — Limestone	Calcium Carbonate	do.	75	—	—	—	—	—	—	0·0	0·0	2·1	3·5	9·0	11·2	not excessive	not excessive	61 to 62
† ENGLAND AND WALES	Grinder of Metals	Metal & Quartz	do.	54	—	—	—	—	—	—	0·0	1·7	1·8	10·5	12·6	23·5	In Excess	Low	48 to 49
† ENGLAND AND WALES	Slate Quarrier Worker	Slate	do.	35	—	—	—	—	—	—	0·0	0·0	1·5	7·2	5·9	5·5	In Excess	No Excess	60 to 61

* Calculated from Supplement to Sixty-Fifth Annual Report of Registrar-General for Births, Deaths and Marriages in England and Wales, Part II., 1908. Wyman & Sons. (Cd. 3619.)

§ Calculated from data given in Annual Report on the Health of Sheffield for 1913, pp. xiii-xiv

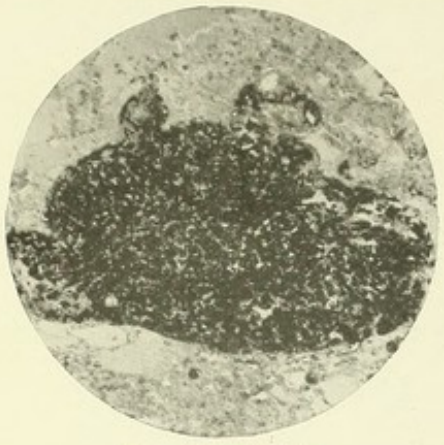
† Calculated from data courteously supplied by General Register Office.

NOTE.—All figures in excess of the standard for Occupied and Retired Males are underlined.

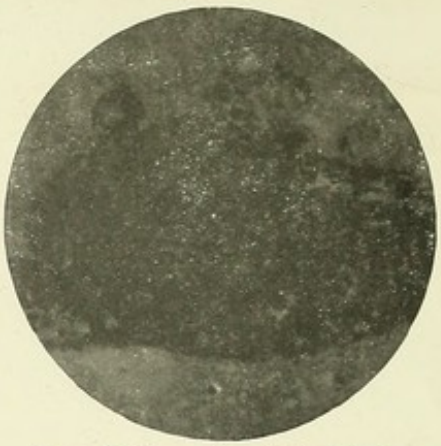
pneumonia. To establish a relation between dust inhalation and the prevalence of respiratory diseases among coal-miners, some reference to the nature of the dust to which these men are exposed is necessary. My predecessor in this lectureship, Dr. Shufflebotham, remarked (55) that "fibroid lung is rare amongst coal-miners, and anthracosis does not seem to entail disablement," but he pointed out, just as Dr. Trotter had previously done (87), that coal-miners are exposed not merely to coal dust, but also to dust of the strata in which the coal is found; and Oliver has suggested (94*a*) that "when a coal-miner's lung shows pronounced fibrosis, it is generally an indication that the individual has worked in a coal seam which contained a quantity of stone; it is the stone dust in the coal, not the coal itself, which . . . is the cause of the fibrosis" (see fig. 3). Probably the prevalence of respiratory diseases among miners depends on the amount and the character of the stone dust present. The influence of coal-dust itself which has been carefully investigated (88) in America by Dr. Wainwright and Dr. Nichols is remarkable. These workers, having first ascertained that statistical data from every available source agreed in establishing that coal-miners are singularly free from phthisis, exposed guinea-pigs for about two months to the inhalation of coal-dust, and then injected a culture of tubercle bacilli into them. Such anthracosed animals developed "extensive tuberculosis of the abdominal viscera and of the glands round the tracheal injection, but *the lungs were free,*" while control animals developed "extensive tuberculosis of the lungs and abdominal viscera." Such facts are evidence that coal-dust in some way protects against phthisis. Coal dust then is not injurious, but, because it can be easily distinguished, it is useful to demonstrate the path by which dust enters and travels through the lungs. First it reaches the alveoli, where it is taken up by phagocytes (see fig. 3*a*); these carbon pigmented phagocytes pass through the walls of the air vesicles into the lymph channels and deposit their pigment in the perivascular spaces (see fig. 3*b*). Should the lymph channels in any place be blocked by fibrous tissue, the pigment collects there, whether the blocking be localised (see fig. 3*c*), or diffuse (see fig. 3*d*). It is found particularly beneath the pleura (see fig. 3*e*); and also in the pulmonary glands (see fig. 3*f*), and in the bronchial glands (see fig. 3*g*), where also it accumulates where there is fibrous tissue (see fig. 3*h*). In other lymphoid tissue, however, for example, the mesenteric glands (see fig. 3*i*), comparatively small deposits are to found. Broadly speaking, in Durham and Northumberland coal lies in nearly horizontal seams, and is got with a minimum disturbance of other rock; while in Lancashire the coal seams, though resembling those of Durham and Northumberland in thickness, lie at a steep inclination and much intervening rock has to be "ripped." The Nottingham and Derbyshire coalfield in this respect resembles the Durham and Northumberland field, and the mortality from respiratory diseases, bronchitis and pneumonia, among the miners on the two fields is low. Exposure to dust arising from intervening rock in the Monmouth and South Wales coalfield resembles that of the Lancashire field, and the mortality from bronchitis and pneumonia among the miners on these two fields is high.

At the outset then the form of respiratory disease which follows upon dust inhalation is found to vary with the kind of dust inhaled; and bronchitis, which occurs in excess when ever there is exposure to injurious dust, appears to be *par excellence* the chief of the pneumonoconioses. The importance of bronchitis as an indication of the injurious properties of a given dust has been somewhat overlooked for two reasons (i) the excessive mortality from phthisis experienced in some dusty industries has overshadowed it, and (ii) bronchitis is a complaint which advances slowly, and, though causing incapacity, does not cause much mortality during the working period of life. Probably when invalidity statistics of occupation become available, this disease will stand out clearly as the chief cause of invalidity in dusty occupations.

Causation.—The curves, shown in fig. 2, which represent the mortality from bronchitis at various age-periods among those exposed to dust inhalation, are similar (only at a higher level) to that for the general population, suggesting that the disease in the dust groups is due to the same influences, only in intensified form, as produce the disease in the



Pigmented Nodule.

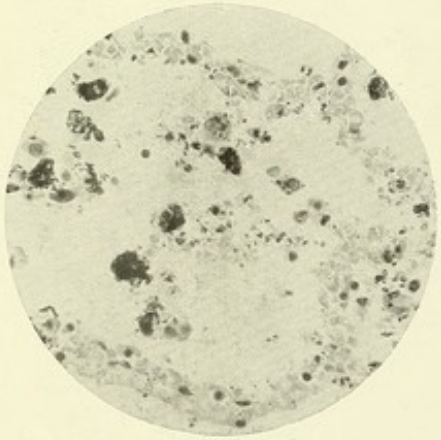


Same Field seen by polarised light.

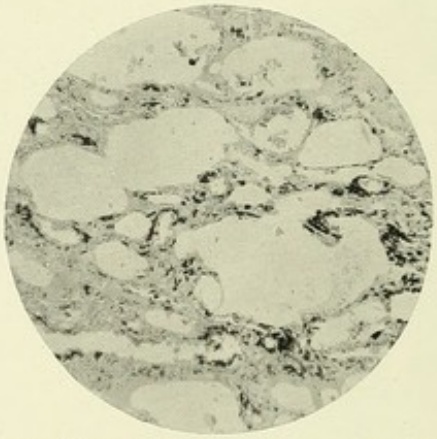
FIG. 3.—COAL MINER'S LUNG.

(North Staffordshire).

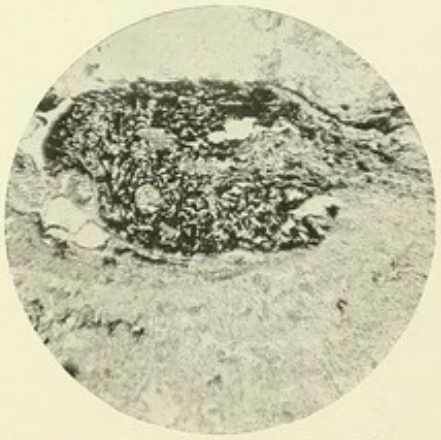
The presence of dust other than coal is indicated by the polarisation of light.



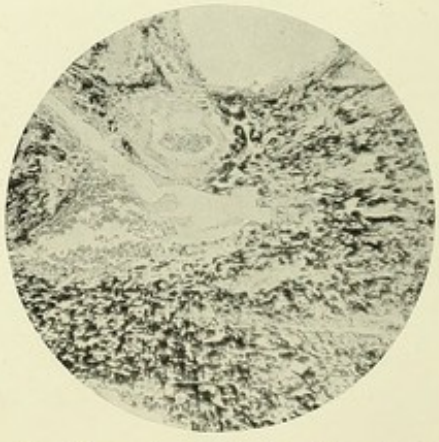
(*a*) Free Carbon particles in air vesicles with several phagocytic cells taking up the particles into their cytoplasm.



(*b*) Carbon particles in the lymphatic spaces of air cells and perivascular channels

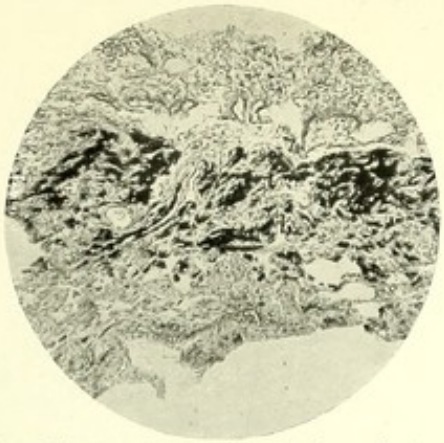


(*c*) Fibrous and pigmented nodule.

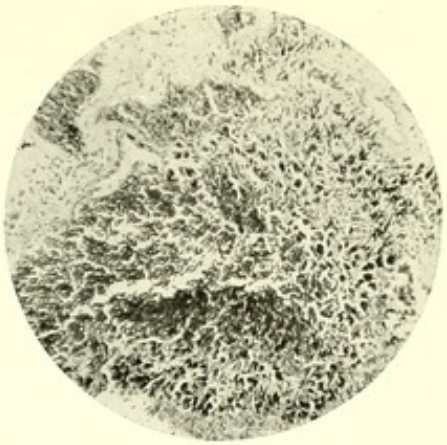


(*d*) Pigment associated with general fibrous change.

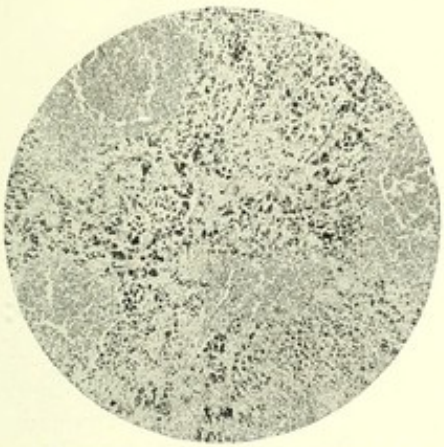
FIG. 3.—PLATE I.



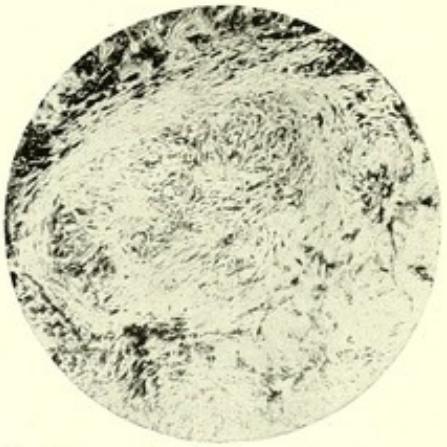
(*e*) Pigment in the lymphatics of the deeper layer of the pleura.



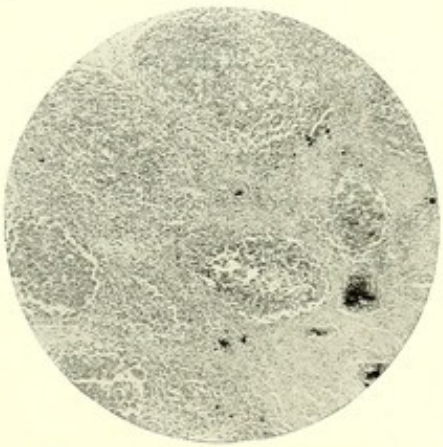
(*f*) Small lymph gland containing pigment.



(*g*) General deposition of pigment in a bronchial gland.



(*h*) Pigmented and fibrous hyperplasia of bronchial gland.



(*i*) Mesenteric gland with comparatively little pigment.

FIG 3.—PLATE II.

(*For this Series* (*a*) *to* (*i*) *I am indebted to Dr. F. Shufflebotham*

general population. This method of obtaining a clue to the causation, in any given group, of a disease which also occurs in the general community appears to have been first used by Givre (*27b*), though the abscissæ of his curves were calendar years and not age-periods. The value of this method will be more apparent when dust-phthisis is under consideration.

The causation of bronchitis in general, leaving on one side the influence of microbic invasion, may be summed up as any stimulation of the bronchial mucosa which determines such an inflammatory hyperæmia of the walls of the air passages that an excessive exudation of mucus results. Dust suspended in inspired air falls on the walls of the bronchi and bronchioles; and, to be removed, must be entangled in mucus and swept back by ciliary action until it can be expelled by coughing. To effect this purpose the secretion of mucus and the ciliary action increase with the amount of dust inhaled. Increase this process beyond physiological elasticity and there occur degeneration and destruction of the ciliated mucosa, and, synchronously, a mechanical ballooning of the alveoli, emphysema, which is produced by frequent coughing to remove the unusual amount of mucus secreted. Other influences besides dust may over-stimulate the bronchial mucosa, but the important part dust plays is shown by comparing the incidence of the disease among fishermen and agriculturists with that among classes who live in atmospheres more laden with dust.

While the influence of dust in general in the causation of bronchitis may be accepted, certain dusts do not appear to exert such an influence. Thus coal-dust, unmixed with other material, *e.g.*, Durham and Northumberland miners, is not associated with bronchitis. And, although mortality statistics are not available, careful investigations made in France (*56*) into the manufacture of plaster-of-Paris, and by Arlidge (*40c*), whose observations among alabaster workers I can personally confirm, suggest that calcium sulphate belongs to this class. Investigations as to the effect of limestone on masons made by Barwise (*57*) in Derbyshire, by Howard in the Isle of Portland, and myself (*58*) among masons in many districts, indicate that limestone dust has no marked effect in causing bronchitis, and recent mortality figures stated in Table 3 justify this conclusion. Pure clay, as contrasted with highly siliceous fireclays, seems also to have no effect. Inquiry into the manufacture of cement (*59*), and mortality figures for cement workers, which only show 23 deaths from bronchitis out of a total of 293 —*i.e.*, 7.8 per cent. as compared with 8.4 per cent. for Occupied and Retired Males—necessitates the addition of cement to the list, notwithstanding the amount of dust generated (*60*) in its manufacture and its hygroscopic nature. Nor is there any evidence that dusts of animal origin, such as bone, leather and horn, when unmixed with other things, conduce to bronchitis.

On the other hand, the data of Table 3 show that exposure to the inhalation of certain dusts is associated with a high mortality from bronchitis; the mortality among masons who dress sandstone places sandstone dust in this list; the mortality of Lancashire coal-miners condemns dust of the strata between the coal seams; the mortality among potters must be ascribed to flint dust; that of tin-miners to quartz dust; and that of cotton operatives and wood workers to dust arising from the materials they manipulate. To this list may be added from clinical evidence dusts of flax (*38d*), jute (*62*), hemp (*14*), slag-wool (*59a*), glass and emery (*53*). These dusts differ widely in origin and in chemical composition, but possess the following common properties: (i) such brittleness as leads them to break into fine dust; (ii) insolubility in the normal secretions of the air passages; and (iii) non-plastic (*i.e.*, probably non-colloidal) structure.

Clinical Symptoms.—Bronchitis induced by dust is essentially a chronic disease. Cough, and free expectoration of sputum coloured with the dust inhaled, are the primary symptoms which are directly referred by the worker to his occupation. If the period of exposure has not been unduly prolonged, absence from occupation is followed by cessation of the symtoms which, however, return when work is resumed. Dr. E. S. Reynolds, with his knowledge of the industrial conditions of Manchester, has described (*93*) at this stage the existence of a fine tube bronchitis especially prevalent among those exposed to emery dust; a condition which he considers is, in

adults, seldom seen apart from exposure to dust.

After more prolonged exposure the bronchitic condition becomes so established that only slight improvement follows upon rest; and, sooner or later, the symptoms usually associated with chronic bronchitis appear, shortness of breath and other signs of cardiac distress. These are, however, well-known, and need not be restated here.

Differential Diagnosis.—What has been said of clinical symptoms shows that the diagnosis of pure dust-bronchitis cannot be safely made from the study of an individual case, but only from previous knowledge of the exposure of the individual to dust, and of the known effect this dust has in causing bronchitis. Even then, since bronchitis occurs in the general population, the influence of dust inhalation only raises a question of probability in the causation of each case, a probability which the presence in the sputum of dust known to be associated with excessive bronchitis supports. When present in an individual in whom dust inhalation has caused pulmonary fibrosis, or the barrel-shaped chest typical of asthma, the physical signs of these conditions may indicate the connection between dust inhalation and the disease; but when it is present in individuals exposed to such dusts as emery, glass and slag-wool, unless the fine tube bronchitis described by Reynolds is present, there may be nothing to distinguish the case from any ordinary case of bronchitis. If a group of individuals is under examination, the unusual number and the early age of those affected may suggest the origin of the trouble; and at one factory where exposure to dust of emery and powdered glass was in excess, although no history of any prevalence of phthisis among the workers could be obtained, and although no definite signs of fibrosis could be detected, I found an unusual number of men, as may be seen by referring to Table 4, in the prime of life suffering from bronchitis, emphysema, and having an average chest expansion below normal.

Table 4 EXPOSURE TO EMERY AND GLASS DUST

Nature of Work	Number Examined	Number with Chests Showing Signs of Disease	
Engaged in dusty Processes	50	13	26 Per Cent.
Yard Labour, Enginemen, &c. Exposed to General Dust of the Factory.	25	2	8 Per Cent.

This diminution in chest expansion which is characteristic of dust inhalation is also a characteristic of chronic bronchitis, as also is the presence of emphysema, although this condition is less marked when pulmonary fibrosis is present.

A correct diagnosis, though often difficult to arrive at, is of importance in advising the patient as to the future; and some help may be obtained from radiography, for evidence does show that the changes induced in the bronchial mucosa may be detected as shadows which in size and distribution coincide with the ramifications of the air passages, and differ markedly from the shadows thrown in cases of dust-phthisis. These shadows resemble those seen in cases of marked bronchitis of other causation; and they are more marked as the exposure to dust has been more prolonged (see figs. 4 and 5).

Pathology.—The degeneration of the bronchial mucous membrane, and the condition of emphysema coincide with those described for ordinary bronchitis in every text book, and require no repetition here. After death dust particles will not often be detected; for the disease is of its nature slow, causing prolonged incapacity, and recent exposure to dust inhalation must, therefore, be exceptional.

PNEUMONIA.

Prevalence.—If, as seems practically certain, dust inhalation is an influence which predisposes the bronchial mucosa to attacks of bronchitis; and if, as will be discussed later, dust inhalation can also modify the parenchyma of the lungs so that it is peculiarly prone to succumb to tuberculosis, then dust inhalation can hardly be expected to leave unaffected the intermediate zone of the lungs, the bronchioles and alveoli, the site of a distinct form of pulmonary disease, pneumonia. Yet dust inhalation as an influence predisposing to pneumonia is not widely recognised—indeed, neither Dr. P. Kidd in his Lumleian lectures (*66*) nor Sir James Barr in his address on pneumonia (*63*) allude to such an influence. But there is no *a priori* reason against it, for pneumonia has certain etiological characteristics in common with other respiratory diseases; bronchitis is probably due, as a final cause, to microbic invasion of

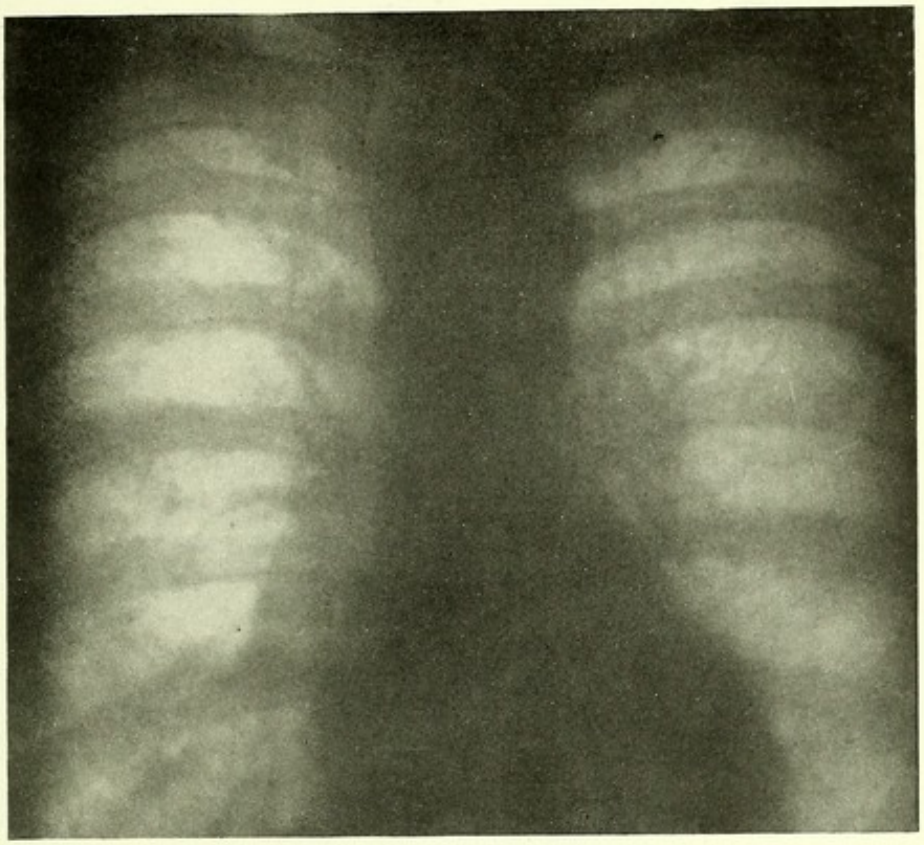


FIG. 4.

SKIAGRAM (taken by Dr. Gouldesbrough) of an apparently healthy man after 14 years' exposure to fine emery dust in the manufacture of emery paper.

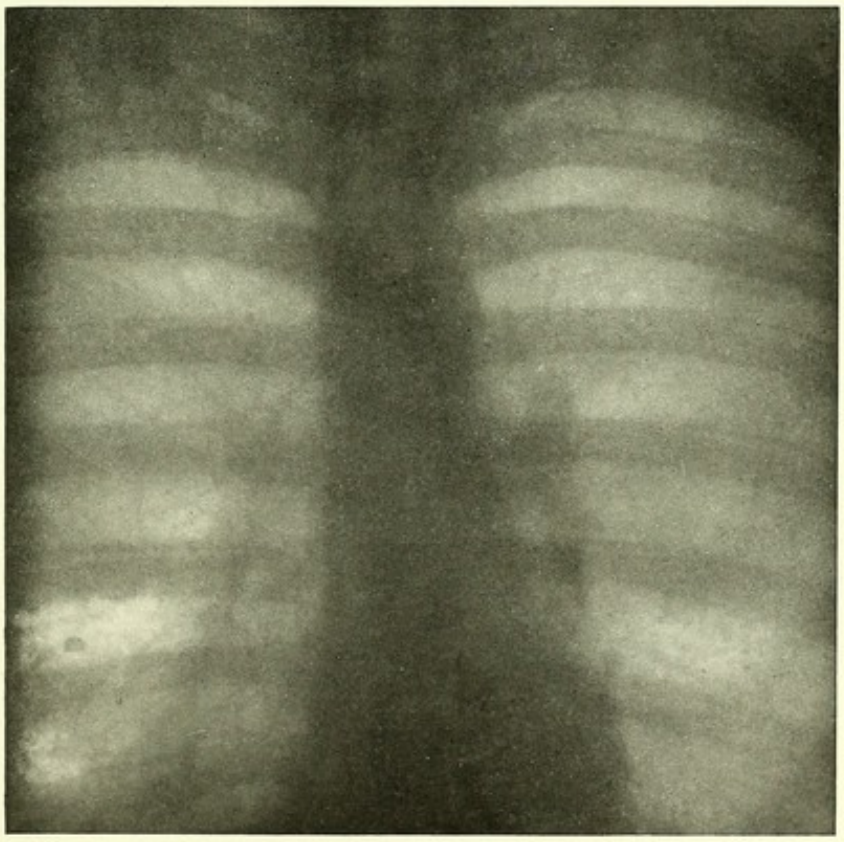


FIG. 5.

SKIAGRAM (taken by Dr. Gouldesbrough) of an apparently healthy man after 27 years' exposure to fine glass dust in the manufacture of glass paper.

NOTE.—Skiagrams of workers exposed for shorter periods to these dusts showed similar but less marked shadows.

the bronchial walls; and phthisis and pneumonia are certainly due to microbic invasion of those parts of the respiratory apparatus, which are the sites of these two diseases. A Committee appointed by the Royal College of Physicians has reported (*64*) that in the spread of phthisis among human beings inhalation of infected dust appears to be the more important means of infection, an opinion strongly supported by the recent work of Chaussé (*101*); and Dr. R. R. Armstrong holds (*65*) that "there seems reason to believe that all forms of pneumonia, not excepting lobar pneumonia, are inhalation infections." Dr. Kidd, it is true, considers (*66a*) that pneumonia is primarily a blood infection, but while doing so he leans to Calmette's view that tubercle bacilli reach the lungs viâ the blood stream through the intestine; and so still recognises a common path of infection. Further, an interesting inverse relation exists between phthisis and pneumonia; thus Kidd, during his extensive clinical and post-mortem experience, has observed (*66b*) that "lobar pneumonia complicating declared and progressive tuberculosis of the lungs is exceedingly rare," and has raised "the question whether there is not some opposition between the two diseases." Now, this opposition is suggestive, for it is found in the mortality statistics of dusty industries (see Table 5); and, as will be seen later, pathology suggests, at any rate for those exposed to dust inhalation, a reason for this antagonism. Hence the prevalence of pneumonia among operatives exposed to certain dusts, but who do not suffer from dust-phthisis, and the absence of any prevalence of pneumonia among operatives who do suffer from dust-phthisis, point to dust exerting a definite influence upon the prevalence or non-prevalence of pneumonia.

In mortality statistics lobar pneumonia and broncho-pneumonia are grouped together, and this fact compels a conjoint consideration of these two forms of respiratory disease, although the special investigations, from which evidence as to a causal relationship between pneumonia and dust inhalation, is drawn, have generally had reference to the occurrence of lobar pneumonia. In late age-periods of life broncho-pneumonia often drops the curtain on cases of chronic bronchitis, and so swells the mortality figure for pneumonia in a group with a high death-rate from bronchitis; and the effect of this statistical method of considering the two diseases together must be kept in mind when mortality rates after the age of 55 years are examined.

Various authorities have maintained that a connection exists between dust inhalation and pneumonia. Merkel held the opinion (*40d*) that people employed in a dusty atmosphere suffer in a higher proportion from croupous pneumonia than others, Hirt considered (*74*) this disease is immediately caused by dust, and West states (*47a*) that "the victims of dust inhalation . . . are exposed to greater risk than others of acute pneumonia." Pneumonia among workers exposed to basic slag dust (pseudo-pneumonie à scories) has been carefully investigated by Monnier (*68*) and by Gautret (*69*). These observers have found the pneumobacillus of Friedlander, and the pneumococcus present, either alone or together, in these cases, and also slag dust; and have described the symptoms as similar to, but more severe than those of ordinary pneumonia, and they look upon prognosis as particularly grave. Heim and Agasse-Lafont, when reviewing (*41*) these researches, admit the possibility of traumatic lesions to the respiratory organs, due to the inhalation *en masse* of caustic dust, lesions which prepare the way for the multiplication of microbes, usual inhabitants of the air passages, with the development of bronchitis and of pneumonia. Aufrecht, of Magdeburg, also states (*96*) that pneumonia is prevalent among slag workers, affecting in two years 48 per cent. of the men at one works; and he further found that the disease among these men had a case fatality of 30 per cent. In the same connection interest attaches to the inquiry carried out by Ballard for the Local Government Board into Middlesbrough pneumonia; he concluded (*70*) that "slag dust, to which the epidemic had been attributed, was not the cause of the pneumonia, but that, when from any cause pneumonia becomes epidemic, persons largely exposed to the inhalation of this dust may and do suffer more than persons not so exposed, and that the disease with them is of high fatality." This question of high case fatality has also been noted when pneumonia occurs among those exposed to other dusts; thus in the case of dressers of

Table 5. *Death-Rates from Pneumonia for Certain Classes of Males distributed in Age-Periods.*

	CLASS	EXPOSURE TO DUST	PERIOD UNDER REVIEW	TOTAL NUMBER OF DEATHS FROM PNEUMONIA	PER 1,000 LIVING AT AGE PERIOD						PERCENTAGE OF ALL DEATHS AT AGE PERIOD						PREVALENCE OF PHTHISIS	PREVALENCE OF BRONCHITIS	MEDIAN AGE AT DEATH FROM PNEUMONIA
					20–	25–	35–	45–	55–	65 AND OVER	20–	25–	35–	45–	55–	65 AND OVER			
* ENGLAND AND WALES	Occupied and Retired Males	——	1900–1902	34 614	0·39	0·59	1·17	1·81	2·65	4·35	8·7	9·4	10·8	9·7	7·5	4·1	——	——	50 to 51
	Agriculturist Agricultural Districts	Country Air	do.	1027	0·20	0·24	0·55	0·81	1·16	2·77	6·1	6·3	10·3	8·7	6·4	3·5	Low	Low	59 to 60
	Fisherman	Seaside Air	do.	57	0·11	0·52	1·06	0·73	1·64	1·98	1·6	6·2	8·6	4·7	6·0	2·0	Low	Low	46 to 47
	Shipbuilding	Outdoor Industrial	do.	292	0·35	0·60	1·16	1·38	2·97	3·70	10·7	12·3	13·2	9·6	9·6	4·1	Low	Normal	52
	Tailor	Indoor Industrial	do.	392	0·26	0·34	0·70	1·64	2·10	3·94	6·4	6·2	6·5	8·2	5·6	3·5	In Excess	Normal	54
	Shoemaker	do.	do.	617	0·33	0·41	0·78	1·49	2·07	3·76	6·3	6·2	7·5	8·1	6·1	3·5	In Excess	slight excess	56 to 57
	Coal Miners: Durham & Northumberland	Coal only	do.	222	0·15	0·25	0·48	1·36	1·89	5·13	3·1	5·4	7·1	9·9	6·0	3·3	Low	Low	51
	Coal Miners: Nottingham & Derbyshire	Mainly Coal	do.	104	0·19	0·17	0·72	0·89	2·11	6·09	5·0	5·1	12·0	7·9	6·7	4·1	Low	some excess late in life	49
	Coal Miners: Monmouth & South Wales	Coal, Shale etc.	do.	409	0·43	0·73	1·20	1·93	3·74	6·05	8·6	12·6	13·5	12·1	9·4	5·8	Low	Excess late in life	43 to 44
	Coal Miners: Lancashire	Coal, Shale etc.	do.	353	0·54	1·01	1·55	2·82	5·13	6·92	11·1	16·7	17·2	16·2	12·1	5·1	Low	In Excess	44 to 45
	Brick Plain Tile Terra Cotta Maker	Clay	do.	149	0·60	0·36	1·02	1·23	2·27	4·72	14·8	9·0	15·0	9·6	9·5	4·8	Low	Normal	48 to 49
	Wool Worsted Manufacture	Wool débris	do.	264	0·23	0·55	1·11	1·67	2·21	5·49	4·5	10·5	12·0	9·3	5·4	3·7	Normal	Normal	53
	Cotton Manufacture	Cotton débris	do.	599	0·46	0·63	1·08	2·21	4·02	6·21	10·4	11·3	10·8	10·4	8·5	4·6	slight excess	In Excess	49
	Sawyer & Wood Turner Cooper etc.	Sawdust	do.	258	0·44	0·33	1·20	2·10	2·92	5·41	11·2	7·4	11·3	9·9	8·1	4·5	Normal	In Excess	53 to 54
	Cutler Scissors Maker	Metal, Quartz, Bone, Emery etc.	do.	83	0·20	1·56	1·00	2·60	3·80	6·92	5·0	20·2	5·7	8·1	6·9	5·1	In Excess	In Excess	51 to 52
	Potter Earthenware etc. Manufacture	Clay and Flint	do.	119	0·47	0·41	1·24	2·37	3·53	7·00	12·5	7·4	8·3	7·3	6·0	5·2	In Excess	In Excess	48 to 49
	Tin Miner	Quartz	do.	19	0·69	0·22	0·86	2·23	2·60	3·93	12·5	1·4	3·2	5·8	3·9	2·5	In Excess	In Excess	48 to 49
	Dressers of Iron Castings	Sand, Clay, Emery, Metal, Charcoal	1893–1912	91	—	—	—	—	—	—	23·3	18·9	33·8	28·2	14·5	5·4	Low	In Excess	45
§	Sheffield: Grinders	Metal & Quartz	1908–1912	31	—	—	—	—	—	—	7·7	9·5	4·4	5·0	6·0	3·9	In Excess	In Excess	47
§	Sheffield: Cutlers	Metal, Bone, Emery Linen, etc.	do.	45	—	—	—	—	—	—	7·1	8·6	13·2	7·4	8·2	6·9	In Excess	In Excess	57 to 58
† ENGLAND AND WALES	Stone Getter, Dresser, Mason: Sandstone	Quartz	1910–1912	147	—	—	—	—	—	—	9·5	8·7	6·7	7·3	5·7	4·0	In Excess	In Excess	53
†	Stone Getter, Dresser, Mason: Limestone	Calcium Carbonate	do.	49	—	—	—	—	—	—	11·1	15·4	0·0	8·5	7·5	2·3	not excessive	Low	62 to 63
†	Metal Dressers and Glazers	Sand, Clay, Emery, Metal, Charcoal	do.	67	—	—	—	—	—	—	33·3	27·7	31·4	23·1	16·9	4·4	Some excess late in life	In Excess	42 to 43
†	Slate Quarrier Worker	Slate	do.	36	—	—	—	—	—	—	10·0	4·3	9·0	11·2	3·2	3·1	In Excess	Low	51 to 52

* Calculated from Supplement to Sixty-Fifth Annual Report of Registrar-General for Births, Deaths and Marriages in England and Wales, Part II., 1908. Wyman & Sons. (Cd. 2619.)

§ Calculated from data given in Annual Report on the Health of Sheffield for 1913, pp. xiii-xiv.

† Calculated from data courteously supplied by General Register Office.

NOTE.—All figures in excess of the standard for Occupied and Retired Males are underlined.

iron castings (see Table 5), their trade secretary told me that information of a member suffering from pneumonia is nearly invariably quickly followed by news of his death; while Watkins-Pitchford, when referring (*44*) to cases "suggestive of an acute broncho-pneumonia" among native gold-miners in the Transvaal, says, "in such cases the patient dies after a short illness," and he attributes the disease "to the inhalation of very large quantities of dust over a short period." On the gold-fields attention has been centred on the study of dust-phthisis, still Purdy, after an investigation in New Zealand and Tasmania, found (*71*) a high incidence of pneumonia in the mortality returns of quartz-mining districts, and he considers "the damage done to the lungs by inhalation of dust inhalation. Similarly, Dr. Haldane informs me, miners' phthisis is not known in the gold-mining district of Cripple Creek, Colorado, where, though dust is freely generated by rock-drills, the workings are not in quartz, but in a hard, siliceous rock. On other American gold-fields, where the gold is in quartz, miners' phthisis appears to exist. Pneumonia at Cripple Creek, though no information is to hand as to its prevalence, is recognised to be very fatal, but this is ascribed to the elevation of the district—10,000 feet above sea-level. In the gold-mining industry, however, the best evidence of the occurrence of pneumonia comes from Western Australia (*73*), where Dr. Cumpston, the Royal Commissioner of 1910, reports that about 1900 "many men took on machine work for the

Table 6. DEATHS FROM PNEUMONIA AND PHTHISIS AMONG GOLD MINERS IN WESTERN AUSTRALIA.

Cause of Death		1900–04		1905–09	
		Age-Period		Age-Period	
		15 to 44	45 and over	15 to 44	45 and over
Pneumonia	Number	78	49	44	38
	Percentage of All Deaths from All Causes	13·2		9·3	
	Proportion	1·59	1·00	1·16	1·00
Phthisis	Number	49	23	57	48
	Percentage of All Deaths from All Causes	7·5		11·9	
	Proportion	2·13	1·00	1·19	1·00

irritating dust makes a suitable soil for the pneumococcus"; and Summons gives (*72*) the mortality per 1,000 from pneumonia, 1905-06, among Bendigo miners as 2.74, as compared with 1.21 for adult males in Victoria, 1903-05; but, as neither observer states the age-distribution of the pneumonia cases, the usual contradistinction between the incidence of pneumonia and phthisis when due to dust, though it may be surmised, cannot be definitely asserted. This contradistinction, however, according to Bremridge's observations quoted (*92*) by Oliver, exists on the Kolar gold-field in India, where "miners' phthisis is remarkable rather for its absence than its presence," but "a large proportion of the miners die from pneumonia," induced, however, it is thought by other causes than first time (with excessive exposure to dust), and therefore the death-rate from pneumonia was high"; after finding that experimental exposure of animals to massive inhalation of dust produced a pneumonic condition of the lungs, Dr. Cumpston concluded "that when a young man first becomes exposed to the action of dust his lungs quickly develop the same condition as was seen in these animals, and therefore he is very liable to attacks of the organism of acute pneumonia." In later years fibrosis, which, as will be shown later, is inimical to the occurrence of pneumonia, developed, and synchronously phthisis increased and pneumonia diminished.

The figures of Table 6 show well the effect of initial exposure to quartz dust, and the altered mortality which follows prolonged

exposure. Another industry in which dust in generated, and in which pneumonia has been stated to be unusually prevalent, is the slate trade; in this industry, according to evidence referring to Merionethshire slate quarries given (76) by Dr. Evans and by Dr. Richard Jones in 1895, and reiterated (77) by the latter in 1912, pneumonia is unusually prevalent among rockmen, miners, and labourers who work underground in the slate quarries, while phthisis is not in excess. In France fibrosis of the lungs, attributed by Dr. Sejournet to dust inhalation, has been described (97) among slate workers at Fumay; but phthisis does not seem to affect these workers so much as bronchitis and pneumonia. More recent statistics for 1910-12, relating to slate workers as a whole in England and Wales, courteously placed at my disposal by the General Register Office, do not, however, disclose any undue prevalence of pneumonia. No explanation can at present be given of the discrepancy. Other evidence may be quoted as establishing a relation between pneumonia and dust inhalation; for example, Dr. Bensusan, after eighteen years' experience in Johannesburg, tells me that, whatever be the time of year, a sandstorm in that city is always followed by an outbreak of pneumonia, and that he is accustomed on the occurrence of such a storm to set aside beds in the hospital for the reception of the inevitable crop of cases.

The most interesting statistics, however, are those from the coal trade, which have already been referred to when bronchitis was under discussion. In certain districts, viz., the coal-fields of Durham and Northumberland, and of Derbyshire and Nottinghamshire, the death-rate from pneumonia is unusually low, while in other districts, viz., the coal-fields of Lancashire, and of Monmouthshire and South Wales, it is unusually high. Unfortunately for the present purpose, coal-miners are exposed to dust of coal as well as that of intervening strata; and since the low death-rates from phthisis experienced by coal-miners in every country (88) compel us to admit that coal-dust has a definite inhibitory effect upon the prevalence of phthisis, the presence of the usual inverse relation between the prevalence of phthisis and of pneumonia loses its significance. Interest, however, attaches to the pneumonia death-rate, the proportionate mortality for which among Lancashire miners is high for every age-period, an evil distinction only shared in Table 5 by dressers of iron-castings, among whom also the phthisis mortality, though not so low as among the miners, is not high.

The dust of dressing shops is of interest in that it contains a high proportion, 79 per cent., of free silica, and yet exposure to it has not been found (as will be shown in the next lecture to be usual when free silica is present) to be associated with a high phthisis mortality, though it is with a high mortality from bronchitis and pneumonia. Another dust containing free silica, which does not seem to predispose to phthisis, is that of clay mixed with quartzite, used in the manufacture of certain refractory bricks; but such evidence as can be obtained suggests that exposure to this dust is associated with a high mortality from bronchitis and pneumonia. Similarly dust of Altoft's shale, which is known to contain over 30 per cent. of free silica, does not seem to predispose to phthisis, but its relation to the occurrence of other respiratory diseases is not known. These three dusts are so far similar that they contain free silica associated with clay in varying proportions. In the present state of our knowledge speculation is idle as to why, as the facts seem to indicate, the presence of clay modifies the action of silica in predisposing to phthisis, while leaving in relief its power of predisposing to pneumonia and bronchitis, especially as the point is undecided whether the same sequence of events follows the inhalation of similar dust in certain branches of the pottery industry. I may, however, say that certain investigations, now being carried on by Dr. H. C. Ross, suggest that this action of clay in modifying the influence of silica dust, which appears to be similar to that exerted by coal, depends on the presence of certain organic constituents in these materials.

Conclusion.—Pneumonia appears to be predisposed to by the inhalation of those dusts which are associated with an undue prevalence of bronchitis; some of these dusts are associated with an excessive mortality from phthisis, and some are not. And when the phthisis mortality is not in excess, the pneumonia mortality is in excess throughout occupational life; but when the phthisis mortality

is in excess, this excess occurs at those later age-periods which represent prolonged exposure, while the excessive mortality from pneumonia occurs at those earlier age-periods which represent initial exposure.

Causation.—The inflammatory processes in the lungs which constitute an attack of pneumonia are caused by the multiplication of micro-organisms in the pulmonary alveoli and bronchioles; and the pneumococcus of Fraenkel is the chief though not the only micro-organism which can initiate such inflammation. This pneumococcus is a normal inhabitant of the upper air passages, and any influence which interferes with the vitality of the lower air passages may be expected to increase the probability of microbic invasion, whether that influence be alcohol impairing ciliary action (*41a*), or cold, or traumatism, either acute, due to local injury, or chronic, due to dust inhalation.

As in the case of bronchitis, the curve representing the mortality from pneumonia at various age-periods among those exposed to dust inhalation sufficiently resembles, though at a higher level, that for the general population to suggest that the influence which determines the mortality it represents acts in a similar way to the influence which determines the incidence of the disease among the general population—*i.e.*, it is an influence which increases the liability to pneumococcal invasion (see fig. 6).

Whether we agree with Kidd (*66a*) that pneumonia arises primarily as a blood infection, or with Armstrong (*65*) that all forms of pneumonia are inhalation infections, the conclusion still holds good that some predominating influence is at work which produces in an exaggerated form the same statistical type of mortality among those exposed to the inhalation of certain dusts.

The prevalence of pneumonia in certain dusty occupations, already alluded to, suggests that inhalation of dust of the following materials predisposes to the disease, viz., basic slag, some forms of slate, the strata in which coal is found, silica when mixed with clay, cotton, wood; but that the disease is not found in excess among those exposed to the inhalation of dusts composed of pure coal, clay, or of those dusts associated at certain age-periods with excessive phthisis mortality; that is to say, pneumonia is probably above the average among all persons exposed to dusts which cause bronchitis with the exception of persons exposed to dusts which also cause phthisis, and even among these persons it is above the average during the earlier years of exposure. This latter fact probably indicates that the pathological changes, to be discussed later, which pave the way for tubercular infection, are inimical to pneumonic infection; and in the fact that those changes involve to a large extent the obliteration of the alveolar spaces and bronchioles, the areas specially concerned in pneumococcal inflammation, may lie the cause for the inverse relation noted between the prevalence of pneumonia and phthisis in dusty industries. Moreover, the occurrence of a high death-rate from pneumonia at early age-periods in such occupations as the manufacture of pottery, tin-mining, and dressing sandstone, in which phthisis is prevalent in later age-periods, suggests that, if such obliteration of the special pneumonic areas did not occur, the pneumonia death-rate would continue excessive throughout life. Pneumonia, then, is predisposed to by influences which interfere with the normal function of the alveoli and smaller bronchioles, as long as these influences do not obliterate these structures. The bronchi and larger bronchioles, on the other hand, remain patent even in pronounced conditions of pulmonary fibrosis, and their damaged mucosa falls a prey to bronchitis. But the tubercle bacillus for which the seat of election is the parenchyma of the lungs, rather than the lining epithelium, finds in fibrosed tissue with its impaired vitality suitable medium for growth. Thus is provided some explanation of the diverse mortality rates from respiratory diseases experienced in dusty industries, all dusts which are insoluble in the fluids found in the lungs, and which are of sufficiently small size to be inhaled, impair the functional activity of the respiratory mucosa, and are influences predisposing to bronchitis and pneumonia, while of these dusts those, which by their presence in the parenchyma of the lungs, determine fibrous formation with obliteration of the seat of election of pneumonic activity, cease after a time to predispose

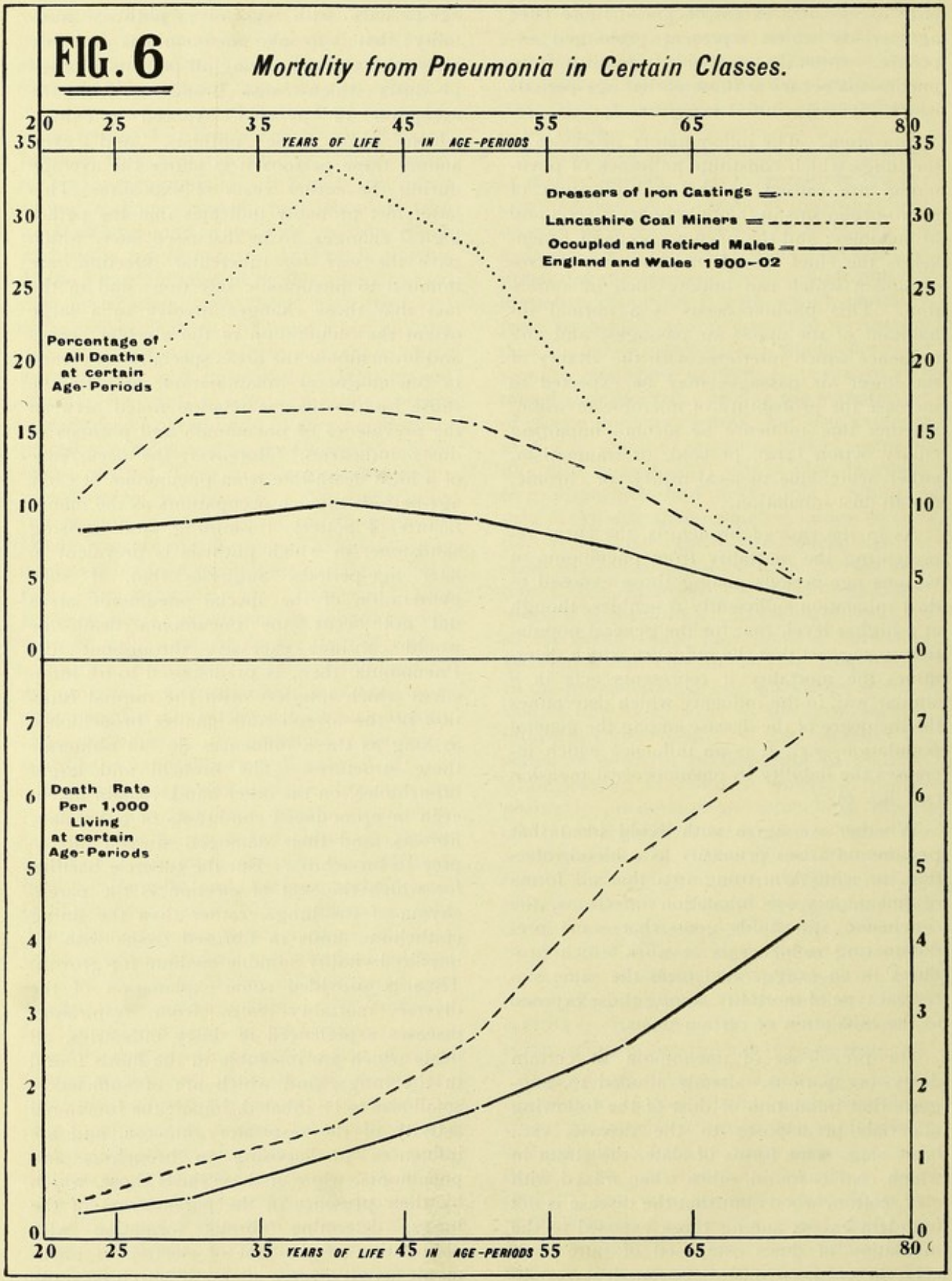


FIG. 6
Mortality from Pneumonia in Certain Classes.
YEARS OF LIFE IN AGE-PERIODS
Dressers of Iron Castings
Lancashire Coal Miners
Occupied and Retired Males England and Wales 1900-02
Percentage of All Deaths at certain Age-Periods
Death Rate Per 1,000 Living at certain Age-Periods
20 25 35 45 55 65 80

to pneumonia, but continue to predispose to bronchitis, and at the same time introduce a new factor by preparing the way for tubercular infection.

Before accepting a purely mechanical explanation of the occurrence of pneumonia in dusty industries, the possibility that other properties of dust or other adverse influences are necessary to reinforce the effect produced by the presence of inert particles of dust must be considered. Briault exposed (*78*) guinea-pigs, some to silica dust, some to nitrous fumes, some to silica dust and nitrous fumes, but only with the combined exposure obtained marked symptoms of acute pulmonary inflammation. Similarly Prof. Beattie tells me that in his extensive experiments (*77a*) on animals with many kinds of inert dust, though he obtained marked congestion of the vessels of the alveolar walls and some exudation, he did not produce definite pneumonia which could be attributed to dust inhalation. In basic slag dust Gautret considers (*69*) the lime and phosphoric acid present may reinforce the mechanical action of the dust particles; in other dusts, *e.g.*, that of iron dressers' shops, such other superadded influences as from time to time lower the general resistance of individuals, *e.g.*, illness, over-fatigue, or alcohol, may determine the onset of pneumonia, a theory held by Brouardel.

An attack of pneumonia is probably determined by either (i) an invasion of an army of microbes too large to be resisted by normal individuals, *e.g.*, epidemic pneumonias, and pneumonia among non-resistant South African natives, or (ii) such acute interference with the ciliary activity of the pulmonary epithelium that a normal amount of infection cannot be thrown back, *e.g.*, massive dust inhalation, and, according to Cesa Bianchi (*41a*), indulgence in alcohol, or (iii) chronic interference with the activity of the pulmonary epithelium, for instance by dust, so reinforced by other adverse influences, *e.g.*, illness, over-fatigue, nitrous fumes, that a normal amount of infection cannot be resisted; and these determining influences may act with varied power and in any combination.

Clinical Symptoms.—The acute nature of this disease prevents those affected from continuing at work; for this reason I have not been able to study the disease at first hand. Others, however, have noted, particularly Ballard, Monnier, and Gautret, in their respective investigations into basic slag pneumonia, that the incidence of the disease is not so much increased as is the case fatality; and, as already mentioned, a high case fatality prevails among dressers of iron castings. Gautret says that in cases of basic slag pneumonia the symptoms are more profound, accompanied at times by severe or even fatal hæmoptysis, the affected area is more diffuse and more often bilateral than in ordinary cases; and that convalescence is slow and accompanied with mucopurulent sputum suggestive of phthisis, but that tubercle bacilli are not found, while final recovery negatives this suggestion. Cases of pneumonia, however, in the origin of which dust has played no part may exhibit such characteristics; and apart from this increased case-severity I have been unable to find any definite indication that pneumonia as it occurs among those exposed to dust differs clinically in any way from pneumonia in general, nor indeed, if the causative influence suggested above is accepted, is such difference to be anticipated.

Differential Diagnosis.—Just as in the case of bronchitis, the connection between the disease and dust inhalation must depend upon an intimate knowledge of the occupation followed, of the exposure to dust, and of the mortality prevalent in that occupation. A correct diagnosis is of importance because, with the known tendency to a fatal termination, every case from its onset, however apparently slight, must be considered serious and treated accordingly. Detection in the sputum of particles of dust known to be associated with pneumonia may assist in diagnosing the occupational origin of the attack.

Pathology.—Theoretically excessive exposure to dust might be expected by mass influence to be followed by excessive secretion from and proliferation of the alveolar cells, thus causing an acute form of broncho-pneumonia; and such cases have been described (*44*) by Watkins-Pitchford in which "the lungs are found post-mortem, to be deeply congested, œdematous, and finely mottled with islands of pigmentation, but exhibit none other of the characteristic appearances

Table 7. *Death-Rates from Phthisis for Certain Classes of Males distributed in Age-Periods.*

	CLASS	EXPOSURE TO DUST	PERIOD UNDER REVIEW	TOTAL NUMBER OF DEATHS FROM PHTHISIS	PER 1,000 LIVING AT AGE PERIOD						PERCENTAGE OF ALL DEATHS AT AGE PERIOD						PREVALENCE OF BRONCHITIS	PREVALENCE OF PNEUMONIA	MEDIAN AGE AT DEATH FROM PHTHISIS
					20–	25–	35–	45–	55–	65 AND OVER	20–	25–	35–	45–	55–	65 AND OVER			
ENGLAND AND WALES *	Occupied and Retired Males	——	1900–1902	61,598	1·60	2·14	2·89	3·18	2·59	1·51	35·4	34·0	26·6	17·0	7·3	1·4	——	——	38 to 39
	Agriculturist Agricultural Districts	Country Air	do.	1,254	1·09	1·10	1·02	1·16	0·88	0·81	33·2	29·5	19·1	12·6	4·9	1·0	Low	Low	40
	Fisherman	Seaside Air	do.	83	0·12	1·40	1·60	1·00	1·64	0·18	13·0	16·6	12·8	6·5	6·0	0·2	Low	Low	36 to 37
	Shipbuilding	Outdoor Industrial	do.	368	1·23	1·53	2·04	2·11	1·59	0·70	37·5	31·3	23·2	14·6	5·2	0·8	Normal	Normal	39
	Tailor	Indoor Industrial	do.	1,133	2·11	2·64	4·12	4·22	3·43	1·79	50·4	45·6	38·3	21·1	9·1	1·6	Normal	Low	38 to 39
	Shoemaker	do.	do.	1,806	2·95	3·27	4·41	4·40	3·21	2·15	56·8	49·4	44·5	24·0	9·4	2·0	slight excess	Low	39
	Coal Miners — Durham & Northumberland	Coal only	do.	403	1·65	1·10	1·01	1·32	1·62	1·71	33·8	24·1	14·9	9·6	5·1	1·1	Low	Low	33 to 34
	Coal Miners — Nottingham & Derbyshire	Mainly Coal	do.	149	1·13	0·64	0·77	1·19	1·47	2·24	29·8	19·3	13·0	10·6	4·7	1·5	Some excess late in life	Low	35 to 36
	Coal Miners — Monmouth & South Wales	Coal, Shale etc.	do.	409	0·90	0·95	1·20	1·43	2·34	1·44	18·0	16·4	13·2	9·0	5·9	1·4	Excess late in life	In Excess	35 to 36
	Coal Miners — Lancashire	Coal, Shale etc.	do.	255	0·89	0·81	1·25	1·76	2·46	2·19	18·4	13·4	13·9	10·4	5·8	1·6	In Excess	In Excess	39
	Brick Plain Tile Terra Cotta Maker	Clay	do.	148	1·01	0·93	0·99	1·52	0·89	0·83	26·9	23·4	14·6	11·5	3·9	0·9	Normal	Normal	35 to 36
	Wool Worsted Manufacture	Wool débris	do.	447	2·38	1·81	2·26	2·89	2·35	1·88	46·7	34·5	24·5	16·0	5·7	1·3	Normal	Low	39
	Cotton Manufacture	Cotton debris	do.	1,067	1·70	2·05	2·98	3·75	2·86	2·24	38·7	36·5	30·0	17·7	6·1	1·5	In Excess	Some excess	37 to 38
	Sawyer & Wood Turner Cooper etc.	Sawdust	do.	439	1·36	2·17	2·83	4·10	2·56	2·39	34·6	41·5	29·4	19·4	7·1	2·0	In Excess	Some excess	42
	Cutler Scissors Maker	Metal Quartz, Bone, Emery etc.	do.	263	2·02	3·78	9·01	11·10	9·01	1·92	50·0	48·8	51·3	34·5	16·3	1·4	In Excess	slight excess	44
	Potter Earthenware etc. Manufacture	Clay and Flint	do.	294	1·40	2·03	3·82	7·26	4·85	1·84	37·5	37·0	25·6	22·4	8·2	1·4	In Excess	slight excess	42 to 43
	Tin Miner	Quartz	do.	154	1·73	7·00	11·71	16·06	16·24	14·75	31·3	52·5	43·2	41·9	23·6	9·4	In Excess	Low	44
	Dressers of Iron Castings	Sand, Clay, Emery, Metal, Charcoal	1893–1912	69	–	–	–	–	–	–	35·4	34·0	21·3	12·8	7·1	4·2	In Excess	In Excess	37 to 38
§	Sheffield — Grinders	Metal & Quartz	1908–1912	312	–	–	–	–	–	–	46·3	69·8	71·7	68·1	39·3	13·1	In Excess	Low	44 to 45
§	Sheffield — Cutlers	Metal, Bone, Emery Linen, etc.	do.	102	–	–	–	–	–	–	50·0	45·7	47·2	26·3	13·6	5·8	In Excess	Normal	45 to 46
† ENGLAND AND WALES	Ganister Workers	Quartzite	1891–1911	103	–	–	–	–	–	–	—	75·0	84·4	75·0	60·0	25·0	Not known	Not known	39 to 40
†	Stone Getter, Dresser, Mason — Sandstone	Quartz	1910–1912	576	–	–	–	–	–	–	28·6	37·2	44·9	35·3	21·6	2·9	Excess	Low	48 to 49
†	Stone Getter, Dresser, Mason — Limestone	Calcium Carbonate	do.	108	–	–	–	–	–	–	27·8	23·1	35·0	19·7	9·3	1·9		Excess only at 20- & 25-	45
†	Stone Getter, Dresser, Mason — Granite	Quartz (¼), Felspar & Mica	1901–1912	100	–	–	–	–	–	–	45·0	35·3	30·8	33·7	21·1	5·0	Not certain	Low	46 to 47
†	Metal Dressers & Glazers	Sand, Clay, Emery, Metal, Charcoal	1910–1912	55	–	–	–	–	–	–	33·3	21·3	21·6	18·5	15·5	4·4	Excess	Excess	43 to 44
†	Grinder of Metals	Metal & Quartz	do.	243	–	–	–	–	–	–	70·0	65·0	65·5	52·6	31·1	7·1	Excess	Low	44
†	Slate Quarrier, Worker	Slate	do.	125	–	–	–	–	–	–	50·0	36·2	28·4	19·4	20·8	7·2	Low	No Excess	53 to 54

* Calculated from Supplement to Sixty-Fifth Annual Report of Registrar-General for Births, Deaths and Marriages in England and Wales, Part II., 1903. Wyman & Sons. (Cd. 2619.)

§ Calculated from data given in Annual Report on the Health of Sheffield for 1913, pp. xiii-xiv

† Calculated from data courteously supplied by General Register Office.

NOTE.—All figures in excess of the standard for Occupied and Retired Males are underlined.

Abel, Aide, Ltd., Statistical Chart Engravers, Holborn, W.C.

of fibrosis," and he ascribes these cases to the inhalation of very large quantities of dust over a short period. Such cases, however, are rare.

Probably when exposure has been prolonged signs of chronic bronchitis will always be found associated with those of pneumonia, but, except in so far as dust particles may be detected in the tissues after death, as, for instance, in Watkins-Pitchford cases, in which "microscopic examination with polarised light shows the connective tissue to be laden with siliceous particles and the alveoli to be distended with serous exudate and catarrhal cells," and in those of Gautret in which basic slag dust was found, no pathological condition has been described which differentiates pneumonia as it occurs in dusty industries from pneumonia as it occurs in the general population.

DUST-PHTHISIS OR PULMONARY SILICOSIS.

Prevalence.—The recognition of an intimate connection between dust inhalation and phthisis, a connection which has been emphasised from the earliest times by writers on diseases of occupation when discussing certain dusty trades, was dealt with in the first lecture. Popular recognition is also to be found in the common use of the term "rot" applied to the disease when it occurs among potters, masons, grinders, and knappers.

The data of Table 7, however, show that phthisis does not occur in excess in all industries in which dust is generated, but only in some (this point will be discussed later when causation is considered); and, that when phthisis is in excess the mortality experienced differs statistically from that of ordinary pulmonary tuberculosis (i) in being always associated with a high mortality from bronchitis, but seldom, if ever, with a high mortality from pneumonia (points which have already been discussed in the previous lecture); and (ii) in exhibiting a latent period before it is fully manifested (varying with the intensity of exposure from two years for Transvaal goldminers to ten years for sandstone masons). The writings of Alison and of Peacock, already quoted, contain references to the comparatively late age-incidence of dust-phthisis, and many later observers have dwelt upon this characteristic which I have ventured to speak of as a "latent period." Statistically the existence of such a latent period is well seen in curves representing the phthisis mortality of certain classes at various age-periods. The curve for the shoemaker so closely resembles that for Occupied and Retired Males as to suggest the inference that the disease is due to the same predominating influence in both classes acting with varying degrees of intensity. Givre, who used this method of determining the influence underlying an undue prevalence of phthisis, concluded (*27*), because the curves representing the annual tubercular mortality for silk workers and for the general population of the town of Lyons were parallel, rising and falling together, and never crossing, that tuberculosis among these workers, although more frequent than in the town, was due to the same influences as affected the general population, only accentuated by the conditions of employment—" la même cause efficiente agit sensiblement de même." Since phthisis has been stated (*64*) by a Committee appointed by this College to be infectious and acquired, the curve representing the incidence of the disease among the general community, represented by Occupied and Retired Males, may be taken to represent the effect of infection upon the community. Hence the deduction may be drawn that the predominating influence for the shoemaker is increased exposure to infection. In the case of the tinminer, however, the curve is very different; and the latency it shows suggests some influence requiring time for full development. Probably this latency represents a period during which a change favourable to the occurrence of tuberculosis is slowly taking place in the lungs; and the condition of pulmonary fibrosis which is known to be present is direct evidence of such a change. The curve for publican, inn servant, gives a similar indication; and probably the latency in this case represents a period during which the tissues of the body as a whole are being adversely modified by the influence of alcohol.

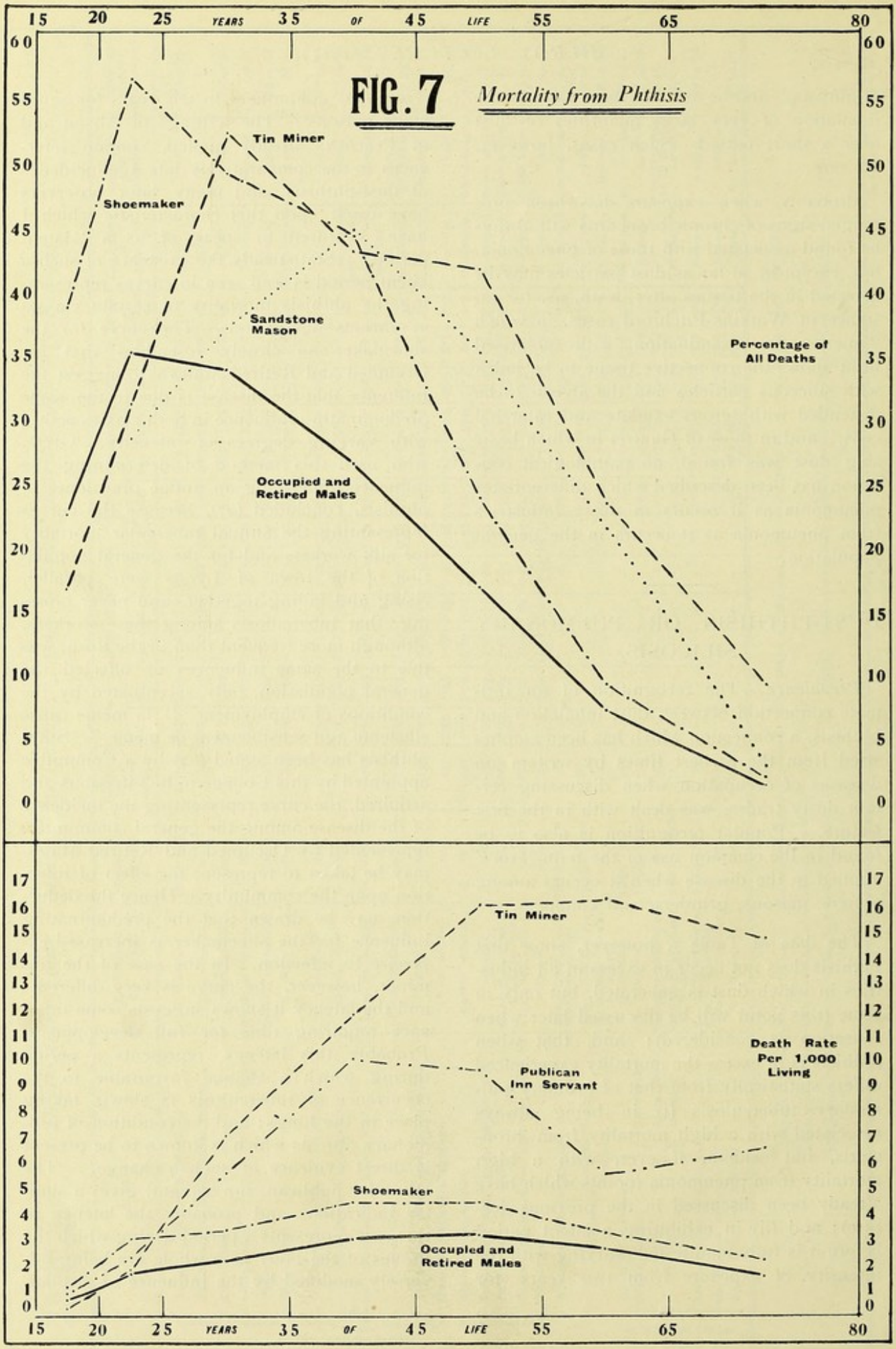


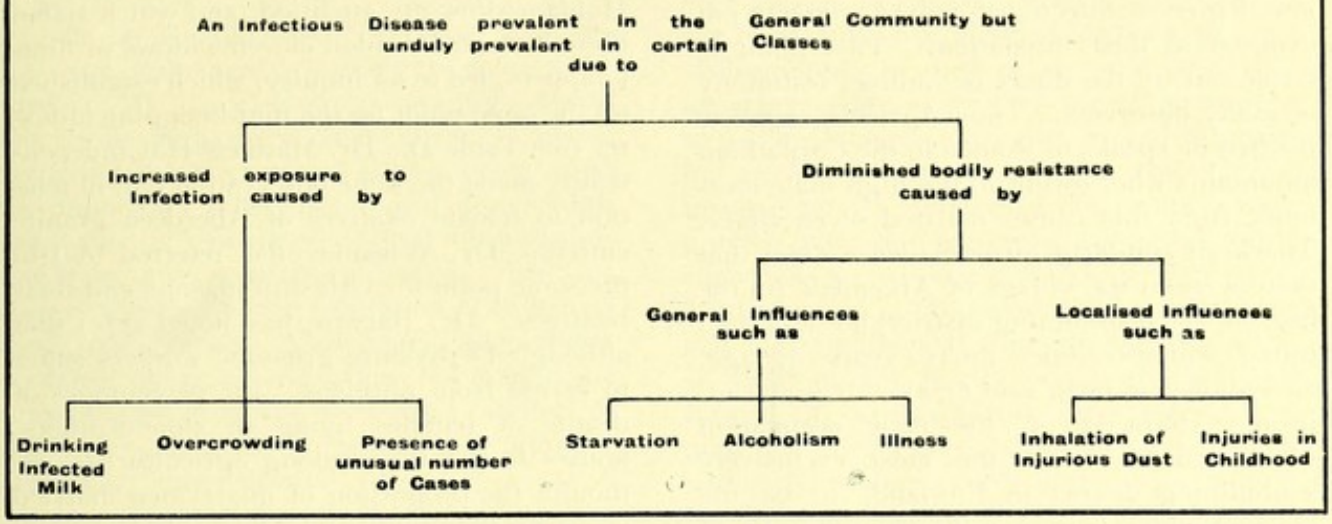
FIG. 7 *Mortality from Phthisis*

Statistics may also give further assistance in pointing out what influence determines an excess of phthisis present in any given class. These influences may be grouped thus:—

case of increased exposure to infection, on the other hand, no reason exists for anticipating excessive mortality from any other cause, nor do we find it.

Table 8.

Tuberculosis



Now, a localised influence, such as a dust which predisposes to phthisis, chiefly affects one organ, the lungs, and persons exposed to such a dust may be expected to suffer a'so from other lung diseases, but not necessarily from any other cause (see Table 9). A general

These statistical characteristics of phthisis mortality, varying as the disease depends on one or another preponderating influence, suggest a method, important for industrial hygiene of determining which particular influence preponderates in a given industry.

Table 9 COMPARATIVE MORTALITY OF CERTAIN CLASSES AGED 15 YEARS AND UPWARDS 1900-2

Cause of Death	General Infection (Occupier & Retired males)	Increased Infection (Shoemaker)	Alcoholism Publican Inn Servant	Dust (Tin Miner)
Influenza	100	67	171	104
Alcoholism and Liver Diseases	100	77	670	28
Cancer	100	103	110	101
Phthisis	100	145	173	436
Other Lung Diseases	100	84	148	419
Nervous Diseases	100	101	178	84
Circulatory Diseases	100	100	144	105
Bright's Disease	100	86	243	143
Accident	100	38	88	92
Suicide	100	100	216	32

influence, such as alcohol, affects all the tissues of the body, and alcoholic subjects may be expected and are found to suffer in excess from other forms of disease. In the

Thus, recent mortality data indicate that slate workers, who as a group are exposed to the inhalation of slate dust, suffer in excess from phthisis at every age-period, but the character

of this mortality which falls immediately, showing no latency, and absence of an excessive mortality from other respiratory diseases, suggest that the excess of phthisis is due to infection arising from undue prevalence of phthisis in the locality, rather than to inhalation of dust.

Dust-phthisis is also peculiar in showing a low degree of infectivity among contacts not exposed to dust inhalation. This point is borne out by the direct or indirect testimony of many observers. Thus Agricola suggests it when he speaks of women in the Carpathian mountains who, owing to the high male mortality from this cause, married seven times. Thackrah similarly gives evidence (*23a*) that there were in the village of Arkendale (in the heart of the lead-mining district) not less than thirty widows under thirty years of age. Greenhow, in 1858, said (*37a*), "It is the injurious character of the male occupation which causes Alston, the most exclusively lead-mining district in England, to be the place in which there is a larger proportion of widows than in any other place in the kingdom," and he gives data from which the following have been taken :—

DEATH-RATES PER 1,000 FROM PULMONARY AFFECTIONS

District	Under 5 years		20 years and over.	
	MALE	FEMALE	MALE	FEMALE
Alston (LEADMINING)	5·61	3·24	14·40	7·79
Redruth (TINMINING)	12·31	10·62	9·42	4·79
Liverpool	30·92	23·64	10·44	7·59
Manchester	20·09	16·95	10·60	9·06
New Forest	4·61	7·65	6·25	6·61

Peacock, in 1864, pointed out (*32*) that among tin-miners the disease "usually occurs in persons who do not present any hereditary predisposition to phthisis—their parents and other relatives having often attained advanced age and being quite healthy." In recent years, as the result of his investigations, 1902-1904, into the nature and cause of excessive mortality among tin-miners, Dr. Haldane observed (*77b*) that the wives and children of these men never seem to be affected, although occupying the same room as the affected men who never go to hospital but sit at home and expectorate sputum loaded with tubercle bacilli. And in 1909 Steel-Maitland and Squire drew attention (*100*) to the number of widows of tin-miners in Cornwall, and of ganister workers at Wortley, even though, due to men outnumbering women in the latter district, the women often marry again. A paragraph in the daily press, to which Dr. Haldane drew my attention, and which stated there were in Brandon eleven widows of flint-knappers, led to an inquiry, which established (*8*) the same point for the flint-knapping industry (see Table 2). Dr. Matthew Hay independently made the same observation (*79*) in relation to female relatives of Aberdeen granite cutters. Dr. Wheatley has referred (*80*) to the same point for Grinshill masons and their relatives. Dr. Barwise has noted (*77c*) that although Derbyshire gritstone workers suffer in excess from phthisis, "the percentages of deaths of females living in stone-workers' houses is less than among agriculturists, although the proportion of quarrymen married is greater than that of the agricultural group." My own investigations (*77d*) into the mortality of stone-masons, their wives and widows, disclosed the same absence of any excessive phthisis mortality among the latter. And quite recently Dr. Turner has told me that his inquiries in South Africa into the occurrence of phthisis in the native kraals have failed to establish that natives returning home with dust-phthisis from the gold-mines disseminate the disease among the tribes. No explanation of this curious low infectivity of dust-phthisis has been offered; but it is the more remarkable because where there is exposure to inhalation of a dust which does not predispose to phthisis, the phthisis mortality among females (not exposed to dust) may be higher than among males (exposed to dust), a point which has been dwelt upon for coal-mining districts by Goldman (*86*) and by Trotter (*87*).

Conclusion.—Exposure to inhalation of a dust which predisposes to phthisis must, then, be immediately suspected, if the mortality statistics for any group establish or suggest the characteristics detailed above, viz.: (i) latency of onset of the disease, (ii) the presence of an excessive death-rate from other respiratory diseases of which bronchitis is the chief, (iii) the absence of excessive mortality

from non-respiratory diseases (unless of course some other baneful influence is at work), and (iv) the absence of excessive phthisis mortality among contacts not exposed to dust inhalation. Conversely, the absence of these characteristics should raise the question whether the dust is one which predisposes to phthisis, however dusty the occupation of those concerned may be. The Royal Commission on Metalliferous Mines and Quarries (*1a*) has summarised the question in these words: "If in any given class a high death-rate from pulmonary tuberculosis is found occurring at a later period of life than is usual for pulmonary tuberculosis, and if this high death-rate is associated with a high death-rate from other respiratory diseases, then this class is exposed to the inhalation of injurious dust; and, further, pulmonary tuberculosis occurring in such a class does not exhibit the same incidence on the wives and families of those affected, as is characteristic of ordinary pulmonary tuberculosis."

Causation.—Study of the prevalence of phthisis in dusty occupations has established that there is no definite relation between the amount of dust present and the prevalence of the disease.

Table 10 *Phthisis Mortality*
IN CERTAIN DUSTY INDUSTRIES AT AGES 20 TO 64

CLASS	Mortality per 1,000 Living	Percentage of All Deaths
Coal Miner	1·17	14·2
Cement Maker	—	18·0
Mason, Limestone (Plymouth & Bristol)	—	18·7
Occupied and Retired Males	2·44	19·8
Potter	3·34	21·4
Cutler (Sheffield)	5·90	26·7
Granite Cutter	6·00	30·4
Tin Miner	9·42	38·2
Grinder (Sheffield)	15·20	57·8
Mason, Sandstone (Manchester)	—	60·1
Flint Knapper	—	77·8
Ganister Worker	—	77·9

Coal-miners are as much exposed to dust as tin-miners, cement makers are more exposed to dust than are granite cutters, masons who dress limestone are equally exposed to dust with masons who dress sandstone, while in the processes of grinding and glazing metal articles in which the amount of dust present has been estimated* by G. Elmhurst Duckering H.M. Inspector of Factories, the point has been definitely established: in this industry metal articles are first ground by operatives, known as grinders, on wheels of hard gritstone (sandstone) from Derbyshire and Yorkshire, which usually revolve in troughs containing water, and are then glazed and polished on emery wheels and linen bobs by men known as cutlers. Duckering found at the breathing level of grinders from 36 to 50 milligrammes of dust in 10 cubic metres of air where edge tools were being ground on wet stones—an amount much exceeded during the occasional process of dry racing, *i.e.*, turning up the surface of the stones with a pointed rod, but less than he found where edge tools were being glazed by cutlers (1,114 milligrammes in the process of hollowing and 647 milligrammes in that of flat glazing in 10 cubic metres of air). Glazing wheels are formed of various substances—emery, alundum and corundum—oxides of aluminium,—and carborundum—carbide of silicon (usually bonded with clay and sand), all substances which rank next to diamond in hardness, and are harder than silica,—yet grinders suffer far more from phthisis than do cutlers. Further consideration, however, has shown that whenever dust-phthisis is present, one special form of dust is also present, viz.: dust of crystalline silica. Silica (oxide of silicon, SiO_2), as quartz, quartzite, flint and chert, forms a large portion of the earth's surface, and in one or other form it is present in every dust, the inhalation of which has so far been found to predispose to dust-phthisis—the precious metals, gold and tin, and in many districts lead also, are found embedded in or associated with quartz and quartzite; sandstone dressed for building purposes is composed of quartz particles; ganister, used for the manufacture of refractory silica bricks, and buhrstone, used for the manufacture of millstones, are extremely hard quartzite rocks; granite is composed of about one-third part of quartz; flint, which is pure silica, is used by the potter, and also gives rise to the dust so injurious to flint-knappers; while grindstones are composed of gritstone, and analyses of dust found in the air of places where metal articles are ground on wet stones show that it is composed of from 50 to 100 per cent. of free silica, and that the amount of dust present

* The method of determination employed by Duckering is described in detail in the Annual Report of the Chief Inspector of Factories for 1910, pp. 301-306.

and the percentage of free silica in it increased with the hardness of the stone used and the weight of the article ground.

On the other hand, silica does not occur, or only in negligible amounts, in coal, cement, clay, emery, plaster-of-Paris, and limestone. The element silicon is not in itself the important factor, for it is present as a silicate in cement and clay, yet workers exposed to dusts of these materials do not suffer from dust-phthisis.

The statement made above that there is no definite relation between the amount of dust present and the prevalence of dust-phthisis is not true if silica dust only is considered, for there is evidence to show that the prevalence of the disease *ceteris paribus* varies directly with the amount of silica dust present. Granite cutters, for example, suffer in proportion to their exposure to dust.

This exposure, due to the use of pneumatic tools, is greater than that of stone-masons, but the phthisis mortality of granite cutters is less than that of sandstone-masons. Granite, however, is composed of only one-third part of quartz, as compared with 80 per cent. or over in most varieties of sandstone. Dundee stone-masons, again, though exposed to dust as much as other masons, suffer less from phthisis, but the stone they dress—Leoch stone, which belongs to the old red sandstone series—is an abnormal sandstone containing only a small amount of quartz.

A study of phthisis mortality rates in dusty occupations has, then, established the fact that when phthisis occurs in excess it is always found associated with exposure to dust containing crystalline silica. Detailed discussion here of these mortality-rates would extend this lecture too much, but reference may be made to the stone-masons' industry. Here is an industry throughout which similar tools are used, and similar methods followed, where work is carried on in similar premises usually in the open air, and wages, varying somewhat in different districts, are rather higher

Table 11 — ABERDEEN GRANITE CUTTERS

Causes of Deaths	Class of Employment	Percentage of All Deaths	Median Age at Death
Tuberculosis	Building Section (open to the Air)	25 ·0	54
	Monumental Section (closed-in)	38 ·0	46 – 47
Respiratory Diseases other than Tuberculosis	Building Section (open to the Air)	13 ·8	60
	Monumental Section (closed-in)	16 ·3	48 – 49
All other Diseases	Building Section (open to the Air)	61 ·2	56 – 57
	Monumental Section (closed-in)	45 ·7	43 – 44

Compiled from lists of deaths published annually in the "United Operative Masons' and Granite Cutters' Journal," published in Aberdeen).

Table 12 — *Percentage Mortality from Phthisis*

AMONG MASONS WHO DIED IN CERTAIN LARGE TOWNS

CLASS	DISTRICT	PERIOD UNDER REVIEW	PERCENTAGE MORTALITY FROM PHTHISIS	Stone used
Stone Masons	Bristol	1871 – 1911	12 ·0	Limestone, e.g. Bath and Portland.
	Plymouth		12 ·5	
	Dundee		18 ·6	Leoch Sandstone in which Quartz is subordinate in amount.
	Cardiff		21 ·8	Four parts Limestone to one part Sandstone.
	Falmouth		24 ·9	Granite, i.e. one-third Quartz.
	Birmingham		37 ·6	About equal parts Limestone and Sandstone.
	Bradford		44 ·2	Sandstones only of various kinds.
	Edinburgh		46 ·1	
	Liverpool		48 ·1	
	Newcastle-upon-Tyne		53 ·0	
	Manchester		56 ·7	
All Males aged 20 Years and over	England and Wales	1871 – 1900	14 ·6	
	Scotland	1901 – 1908	12 ·7	

NOTE.—Further extensive statistical details will be found in Minutes of Royal Commission on Metalliferous Mines and Quarries, Vol. III., Appendices P, Q, and R, pp. 222-38, 1914. Wyman & Sons, Ltd. (Cd. 7478.)

in the sandstone districts of the North and the Midlands than in the limestone districts of the South-west—a trade wherein, all things considered, the conditions of life as lived by the men both at home and at work are practically the same throughout the country. Yet the mortality from respiratory diseases among masons in various districts differs considerably, and this difference coincides with differences in the chemical composition of the stones worked. Thus masons in districts where limestones are worked do not suffer in excess from phthisis, and are at least as healthy as the community in which they live, while masons in districts where sandstones are worked suffer in excess from pulmonary tuberculosis, and have a shorter prospect of life. This evidence, from which every other influence except the different chemical composition of the stones worked can be eliminated, is sufficiently conclusive for me to adduce some figures supporting it, based on the assumption that a mason who died in a district where limestone is chiefly worked, worked chiefly on limestone, and that a mason who died where sandstone is chiefly worked, worked chiefly on sandstone. This assumption can only give approximately accurate results, but fortunately for the purposes of this investigation, the migratory habits of masons have not been sufficient to obscure the issue; and the differences found are sufficient to justify the conclusions that if these influences could be eliminated the limestone mason would take a favourable position in tables of occupation mortality, while his fellow working on sandstone would be found in an even worse position than that the statistics now presented allocate to him. The positive and negative evidence in favour of this peculiar influence of silica dust is so strong as to compel recognition; and to suggest, even without clinical and pathological evidence, that inhalation of silica dust produces a condition which renders the individual more prone to succumb to pulmonary tuberculosis. The existence of a statistical latent period before this condition is fully developed points to some preliminary change requiring time for its establishment, and indicates that the predisposition to the disease is not simply a mechanical interference with the function of the respiratory mucosa, such as has been postulated to account for the prevalence of bronchitis and possibly of pneumonia in certain dusty industries. Pathology indicates what these changes are, and that the respiratory apparatus is the part mainly affected; and justifies us in considering the predisposing condition one of pulmonary silicosis, and the type of pulmonary tuberculosis which supervenes as tubercular silicosis.

Clinical Symptoms.—Certain definite clinical characteristics distinguish dust-phthisis from ordinary pulmonary tuberculosis. Holland perceived this (*91a*) in 1843 and divided the cases of phthisis he saw into two classes—"in the one, we placed the prominent and clear-sounding chest on percussion, in which the cough was often extremely troublesome and the expectoration copious, the breathing very hurried, almost without exertion; and yet the appetite was good, and the digestive functions regular; nor was the body much emaciated. In the other, the contracted or flat chest, and which on percussion emitted a somewhat dull sound, though not invariably, in which the expectoration was also often copious, but less frequently accompanied with severe fits of coughing, and the cough had perhaps existed only from six to eighteen months; when in combination with the other train of symptoms, it had probably been present as many years: emaciation, occasional profuse perspirations, increasing debility, and the slightly accelerated and soft pulse, were likewise generally observed." Peacock also recognised the distinction, and even went so far, after investigating the health of Cornish miners for the Royal Commissioners of 1862, as to claim (*32*) for miners' disease, in which the causative influence of dust was not then recognised, a pathological entity separate from ordinary phthisis. Both Holland's classification and Peacock's claim must be allowed, if the changes produced by long-continued inhalation of silica dust are distinguished from those due to subsequent infection by tubercle bacilli. Peacock and Holland, however, made their observations before Koch had demonstrated that ordinary phthisis was due to the growth in the lungs of tubercle bacilli. When in 1904 the Departmental Committee appointed to inquire into the health of Cornish tin-miners pointed

out (*34b*) that tubercle bacilli could be demonstrated, either during life or after death in such a large proportion of cases of miners' disease as to justify the conclusion that a fatal issue was practically always due to tuberculosis, attention was so concentrated on the part played by the tubercle bacillus that the disease came to be almost regarded as a subgroup of pulmonary tuberculosis. Oliver, however, has maintained (*94a*) that the condition which precedes and predisposes to tubercular infection must be carefully distinguished from the subsequent tuberculosis, and evidence from the Transvaal (*81*) and from Western Australia (*48*) indicates that this condition may in itself, without the intervention of tuberculosis, lead to a fatal issue; in such cases Watkins-Pitchford says (*44*) "death usually results from cardiac failure due to the increasing dilatation of the heart." I have specimens from the lungs of (i) a Transvaal goldminer who died in this way, and (ii) a Staffordshire potter whose end was similar.

Inhalation of silica dust takes several years, varying with the intensity of the exposure, in producing any obvious effect, but gradually the affected person notices that on exertion his breathing capacity has become limited, and that a cough, slight at first, has become persistent, though seldom accompanied by much expectoration; otherwise he may look robust and feel well. This limitation of the breathing capacity which has been noted by nearly every authority from Hippocrates to the South African Miners' Phthisis Commission of 1912 has been described as pathognomonic of the disease, and may at first be the only objective sign present. In several inquiries in which some thousands of operatives have been examined, I have collected physical data which demonstrate this limitation, and the results are stated in Table 13; but, as Dr. Cumpston points out (*48a*), "the actual amount of expansion in inches is not of so much importance as the *manner* in which the chest-wall behaves when an attempt at expansion is made." This diminution in chest expansion must, however, not be relied on as an indication of exposure to silica dust, as it is present, though not so markedly, among workers exposed to other dusts.

Should the condition progress until the age of 45 to 50 years, a definite clinical picture is presented. Such a man is usually somewhat below the average height for his class, for exposure to dust inhalation during the growing period of life appears to inhibit the normal growth; he looks well, but if he had just ascended stairs or has hurried, he is out of breath for a few minutes; on being questioned, though unable to count to twenty without taking a breath, he speaks of himself as fit, and may even boast of being a credit to his trade; but he owns to frequent colds, especially in winter, and to a troublesome cough; he expectorates but little sputum, and what there is is rejected with difficulty and is coloured by the dust produced in his work; in some industries, particularly in tin-mining and gold-mining, he is stated to be subject to distinctive attacks of dyspnœa, but this

Table 13 AVERAGE CHEST EXPANSION IN INCHES OF MALES IN CERTAIN CLASSES AT VARIOUS AGE PERIODS

Age Period	Leisured Class	Factory Operatives not exposed to Dust	Aberdeen Granite Trade			Sheffield Cutlery Trade			Strippers of Cotton Carding Machines
			Polishers, &c. very slightly exposed to Dust	Cutters in open sheds	Cutters in closed sheds	Cutlers	Light Grinders	Heavy Grinders	
14–	2·31	—	2·58	2·14	2·40	2·43	2·54	2·67	3·33
17–	2·45	—	2·57	2·64	2·60	2·27	2·37	2·49	
20–	2·79	2·84	2·58	2·43	2·44	2·24	2·45	2·19	2·75
25–	2·90	2·33	2·21	2·51	2·11	1·96	2·40	2·19	2·45
30–	2·87	2·13	2·14	2·19	1·93	2·09	2·14	2·04	
35–	2·64	2·20	2·04	2·13	1·80	1·92	2·07	1·89	1·86
40–	2·35	2·15	2·07	2·16	1·67	1·73	1·75	1·52	
45–	2·39	2·24	1·98	1·92	1·84	1·56	1·58	1·55	1·69
50–	2·24	1·64	2·04	1·73	1·44	1·49	1·58	1·51	
55–	2·12	—	1·80	—	1·56	1·50	1·53	1·45	1·72
65 AND OVER	1·87	—	—	—	—	1·13	—	—	

symptom is not prevalent among metal grinders or granite cutters. When he is stripped he is found to be well nourished, but respiration is seen to be carried on nearly entirely by diaphragmatic action, and, even though urged, he seems incapable of inducing his intercostal muscles to lift his ribs.

Closer examination reveals an absence of vibrissæ guarding the entrance to the nostrils, while the anterior quarter-inch of the nasal mucosa is smooth, dry, and pale-coloured; behind this the membrane, which is probably covered with a crust of dust, is red and inflamed. Dust may also be seen lying on the pillars of the fauces, and on the back of the pharynx, which is insensitive to the touch of a spatula used to depress the tongue. On percussion, although the note elicited may be similar on both sides, careful observation may detect isolated patches of dulness, especially beneath the scapula or in the axillary line; but the level of the heart dulness is found to commence at the fourth rib cartilage, though the dulness at the apex does not obviously differ from the normal; and the level of the liver dulness is found in the fifth intercostal space or at the fifth rib; in cases, however, where cough has been a marked symptom in the progress of the disease, this exposure of the heart and liver by retraction of the lung margins may be masked by the development of emphysema, and as the result of two co-existing pathological conditions the dulness may be found at the normal levels; but emphysema is seldom if ever present in an accentuated form. When the exposure to dust is intense, as in the case of South African gold-miners, the formation of fibrous tissue is so massive that the retraction of the lungs here described is not seen, nor has emphysema time to develop. All observers are not agreed on the results of auscultation. Peacock speaks (*32*) of "general feebleness of the respiratory sounds, and extreme weakness of the sounds and action of the heart"; possibly he wrote after examining men in an advanced stage; still Watkins-Pitchford says (*44*) "auscultation reveals areas of diminution or abolition of the vesicular murmur." Personally I have heard the heart sounds clearly and easily with no accentuation of the sound of the closing pulmonary valve compared with the closing of the aortic valve, and the breath sounds, with a tendency to be puerile, more clearly than usual at the bases, both in the axillæ and at the back; but my observations were made on factory operatives taken from work. They agree, however, with those of Holland, who states (*91*) that the respiratory murmur "is generally bronchial in its character, conveying the impression that respiration is principally carried on through enlarged bronchial ramifications"; with Greenhow's statement (*38e*) that in "typical cases of needle-pointers' disease . . . the natural murmur of respiration was more or less changed into a coarse, sonorous, and sometimes almost tubular sound"; and with the report of the Committee of the Transvaal Medical Society on Miners' Phthisis, which, after pointing out (*34c*) that "percussion and auscultation often give very indefinite results, owing, no doubt, to the diffuse nature of the disease," goes on to speak of "harsh, interrupted or broncho-vesicular breathing." Writers describing the condition of gold-miners speak of the frequency of pleurisy, but I have detected this in only a few cases, and possibly the more intense exposure of the miners to dust, as compared with the slower exposure, extending over many years, of men employed in factories, accounts for the difference; certainly these latter men do not complain of pleuritic pain to the extent reported among the miners. On measuring the chest the difference between the circumference at the nipple line in deep inspiration does not exceed that in deep expiration by more than one inch; and the value of this measurement, as an indication of breathing capacity may be checked by using a spirometer, when the maximum amount of air expired was found by Dr. Barnes to be about 2,500 c.c. Associated with diminished air capacity, the blood pressure in the radial artery is found to be raised; observations made on men coming directly from work, and taken while the men were standing up, gave readings which most clinicians would consider unusually high; but how far rest modifies this pressure I cannot say. I found, under the conditions named, that, when the chest expansion was below one inch, the blood pressure was seldom below, and usually considerably above, 170 mm. of mercury. Summons gives (*49a*) the results of 20 blood examinations from fibroid cases, and

of 10 from tubercular fibroid cases, of which the averages are as follows:—

CASES	Haemoglobin	Erythrocytes per cub. m. m.	Leucocytes per cub. m. m.
Fibroid	98 Per Cent.	5,090,000	11,500
Tubercular Fibroid	71 Per Cent.	4,150,000	17600

He considers the high count of red corpuscles in fibroid cases "must be looked upon as an effort to compensate for the interference with the respiratory processes in the lungs."

When the condition advances further, the working capacity becomes seriously impaired, but I have been astonished to note how long a man exhibiting all the above symptoms in an accentuated degree will continue to work at such arduous processes as the grinding of metals or the dressing of sandstone; as Holland says (*91b*) "they complain only when disease interferes with the ability to pursue the occupation." Finally, however, the overtaxed heart dilates and the picture changes, the cough becomes more bronchitic, œdema of the legs and ascites supervene, and the end from heart failure is in sight, after which chronic bronchitis on the death certificate gives but little indication of a death from occupational disease.

Few cases, however, progress to such a termination; occasionally apparently strong and healthy men, and these cases are specially reported among gold-miners, die suddenly; while in the majority of cases at some stage the progress of the silicosis is interrupted by tubercular infection, the onset of which is usually quickly indicated, for the patient begins to lose weight rapidly, and his cough becomes more constant and troublesome. When this infection occurs after prolonged exposure to silica dust the progress of the tuberculosis differs from that of ordinary tuberculosis—the sputum in which tubercle bacilli may be either plentiful or difficult to find, is not so copious, hæmoptysis is rare, night-sweats and febrile symptoms are absent. The *spes phthisica,* however, is present, and I have seen men with but a few weeks to live cheerfully and hopefully struggling at their work. Fine crepitations may be first detected in such cases in the middle or lower parts of the lungs rather than at the apex; I have heard them at the level of the fourth rib in front, in the axilla, and at the angle of the scapula behind when no signs could be detected of apical tuberculosis; and this has also been noted (*34d*) among Cornish tinminers. Cavities, though they occur, can seldom be detected clinically, owing to the dense character of the altered intervening pulmonary tissue.

Some authorities claim that tuberculosis when implanted on silicosis progresses more slowly than ordinary pulmonary tuberculosis, but I am not aware of any data to justify this claim. On the other hand, Barwise reports (*77c*) that stone-masons "go on with their work suffering with chronic bronchitis and then die after a short illness. The history is rarely one of chronic consumption." And Wheatley, speaking of stone-masons, sums up (*80*) thus: "The progress of the disease may be extremely chronic, lasting in some cases for upwards of twenty years, or, particularly in those cases where tuberculosis supervenes, it may be very rapid." While Watkins-Pitchford says (*44*) "a South African miner who shows definite incapacity from the disease has, in present circumstances, an expectation of life of probably not more than two years," an estimate supported by Porter's figures (*82*) that of 203 deaths from phthisis which occurred amongst miners employed underground 38.9 per cent. occurred in the first year of illness, and that in five years 78.8 per cent. were dead; while among 67 deaths from phthisis among housewives only 65 per cent. succumbed before the end of the fifth year. The rapid onset of the tubercular change has also been noted (*34d*) among tinminers, among whom "the comparative suddenness with which the disease often begins . . . is somewhat surprising. A man who appears to be in the best of health may rapidly fall a victim." Personally I consider that the silicosis which precedes and predisposes to tuberculosis is slow in developing, but that tuberculosis implanted on silicosis is rapid in its course and unusually fatal in its termination.

Differential Diagnosis.—Knowledge of the exact occupation followed and of the exposure which such occupation entails to the inhalation of silica dust is of great value in making a correct diagnosis. Silica dust is generated in the following among other industries:—Gold-mining, tin-mining and lead-mining;

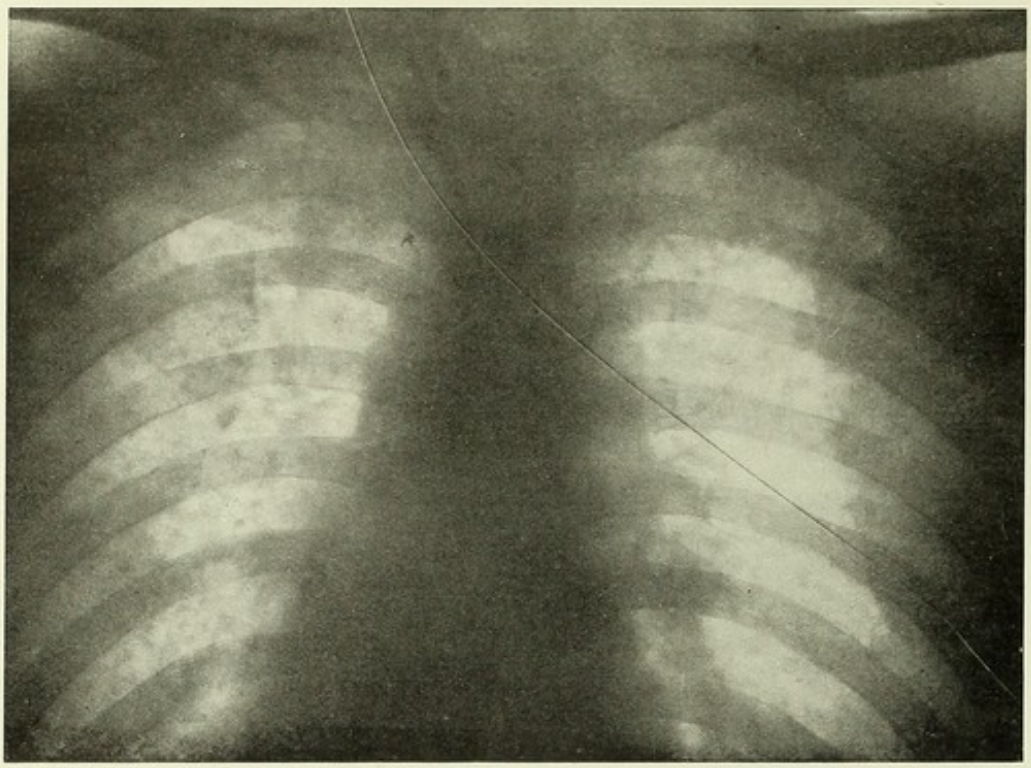


FIG. 8.

SHEFFIELD GRINDER after over 20 years' work; he was in good health at the time, and is known to have remained in good health for two years afterwards.

(*Taken by Dr. R. Hallam, Sheffield.*)

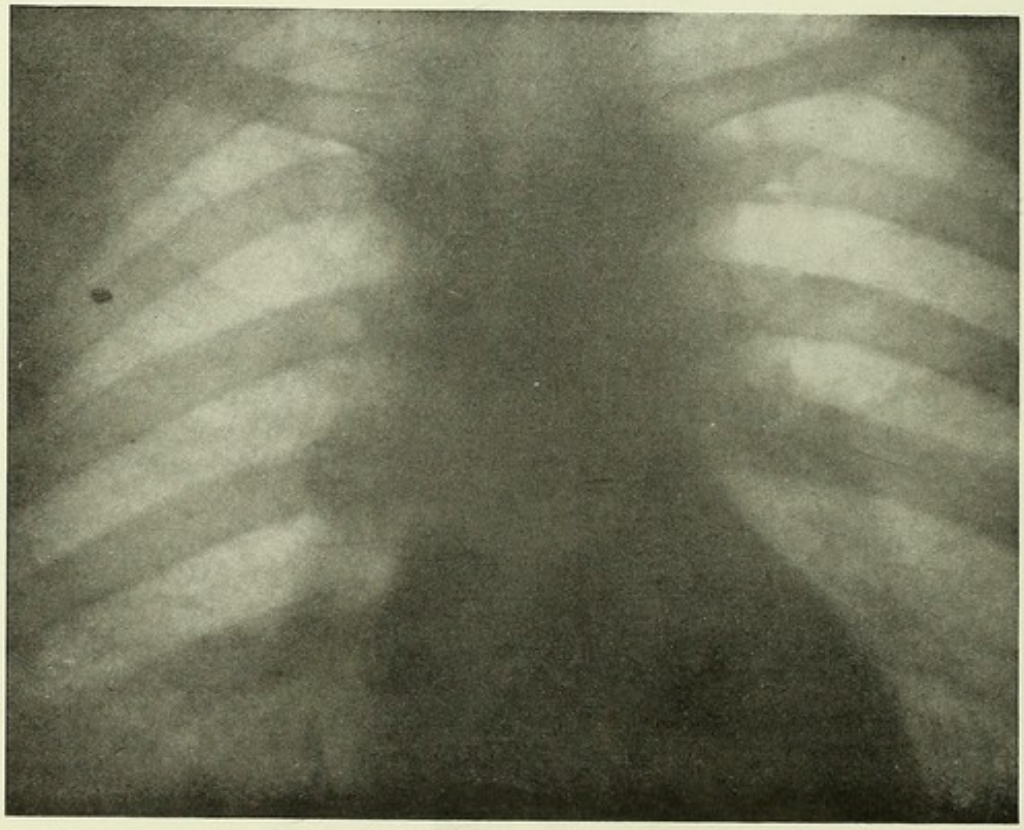


FIG. 9.

TRANSVAAL GOLD MINER.

Early Silicosis.

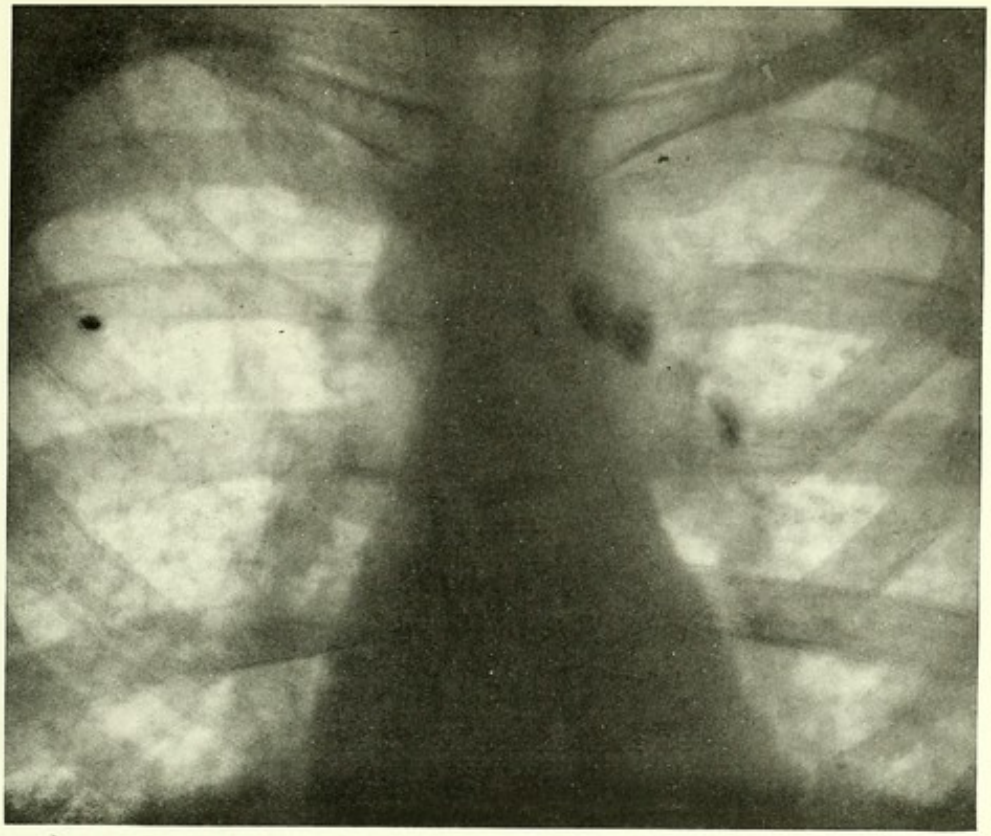


FIG. 10.

TRANSVAAL GOLD MINER.

Medium Silicosis.

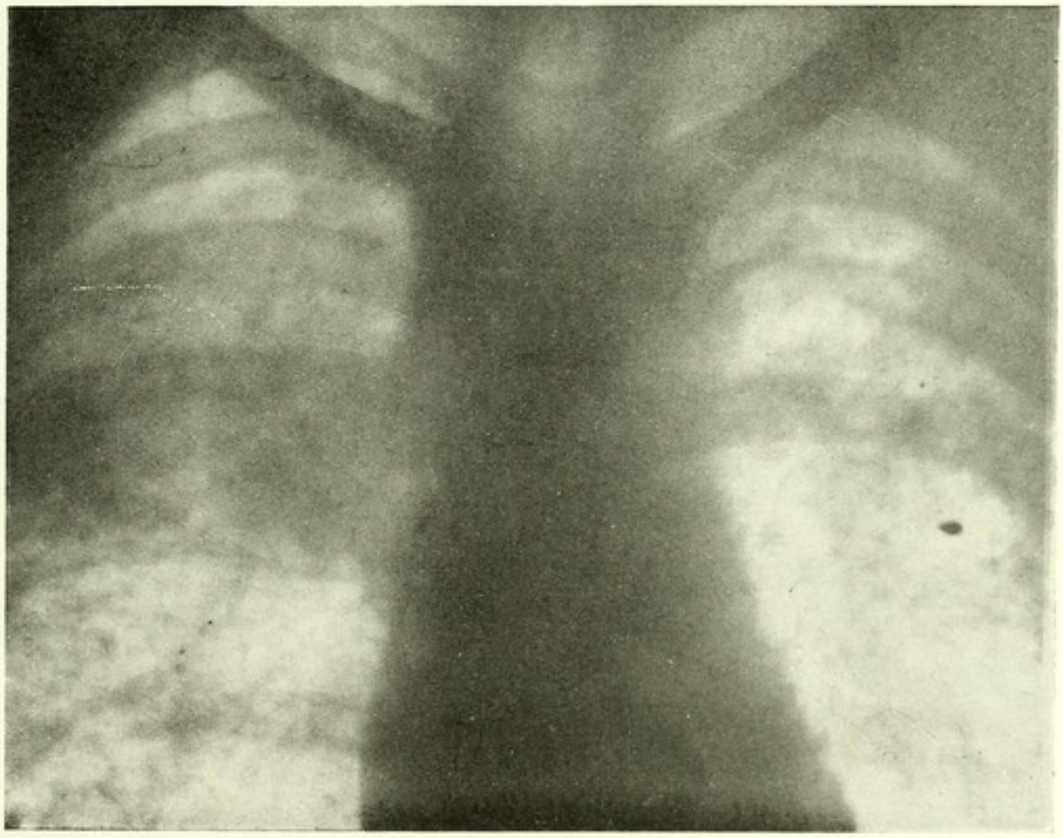


FIG. 11.

TRANSVAAL GOLD MINER.

Medium to late Silicosis with Tuberculosis.

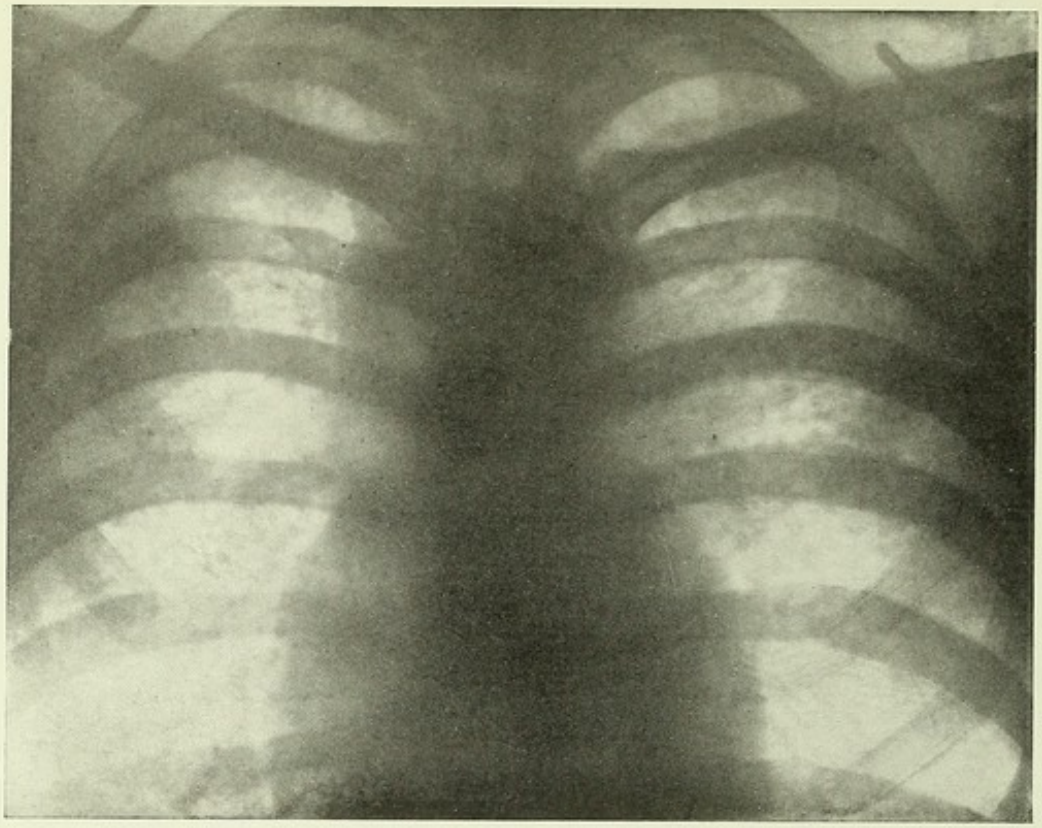


FIG. 12.
TRANSVAAL GOLD MINER.
Advanced Silicosis.

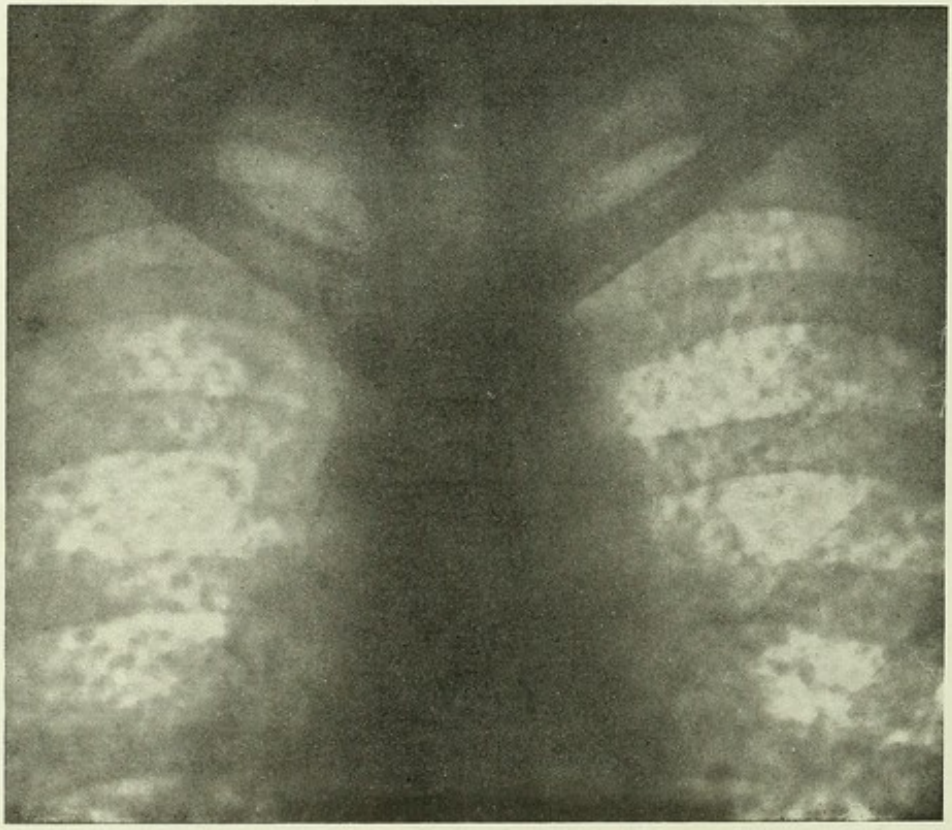


FIG. 13.
TRANSVAAL GOLD MINER.
Advanced Silicosis, Coarse Type.

(Figs. 9–13 were obtained from Dr. Andrew Watt, Simmer and Jack Hospital, Transvaal.)

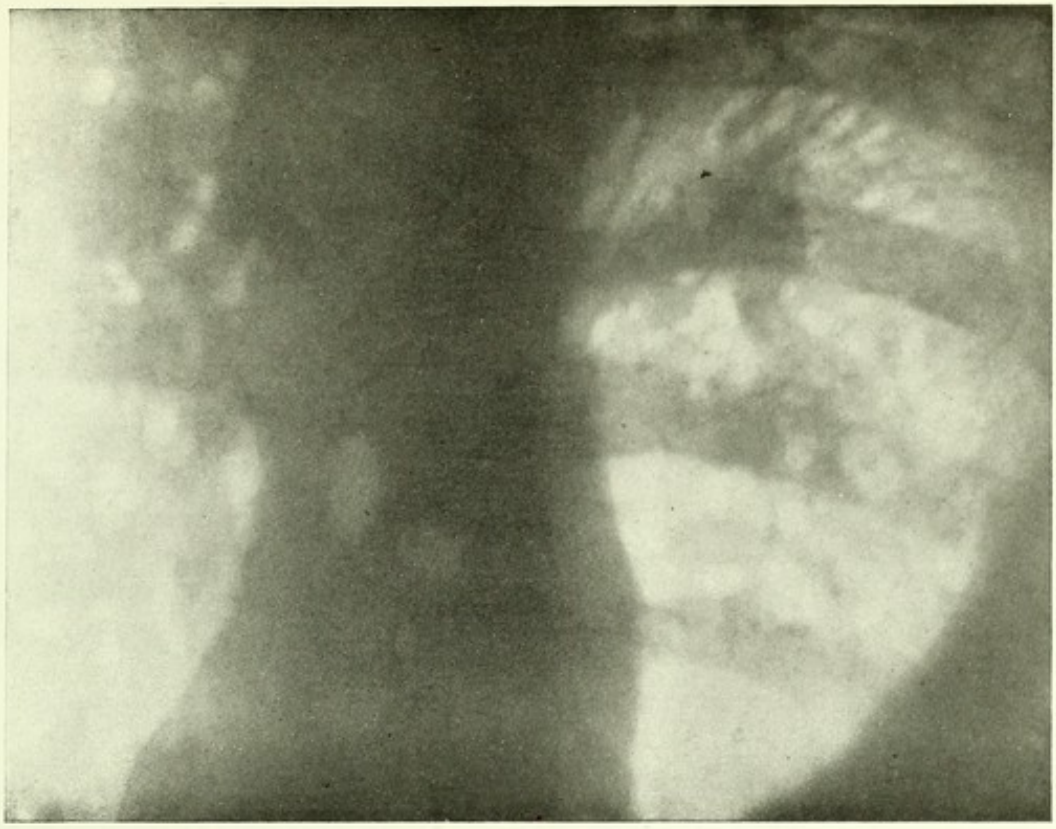


FIG. 14.

Healthy Derbyshire Mason, after working 20 years on gritstone (hard sandstone).

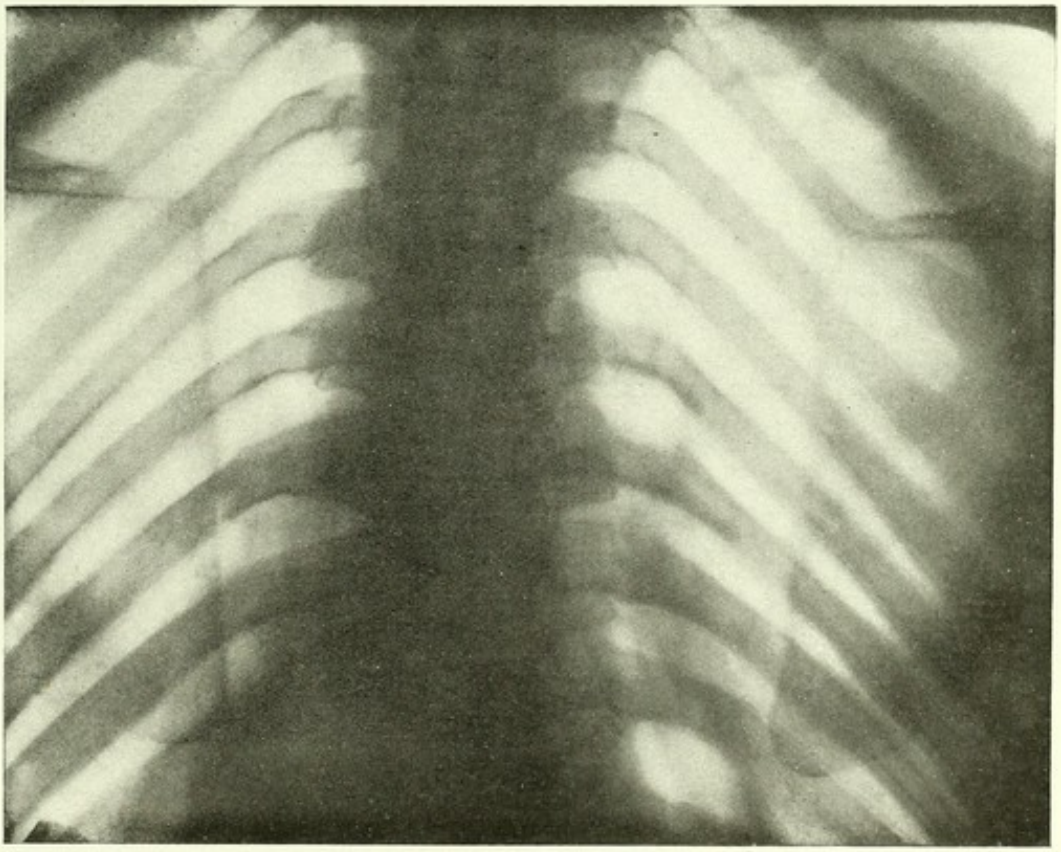


FIG. 15.

Healthy Derbyshire Mason, after working 17 years on limestone.

(Figs. 14 and 15 were taken by Dr. Barwise.)

quarrying and dressing sandstone and granite; flint-knapping; making honing-stones for scythes; building millstones which are used and require dressing in such diverse industries as the milling of flour, rice, cocoa, cement (occasionally), and white lead; the manufacture of grindstones which are used and require racing, trueing and surfacing in the grinding of metal articles, mother-of-pearl, bone, horn and other materials; sand-blasting to clean castings and to etch glass; crushing flints and quartz to make silica flour used in the manufacture of pottery, certain abrasive soaps, sand-papers, chicken food, disinfecting powders, and silica paints; the mining and quarrying of ganister and silica stone; and the manufacture of silica-bricks. As I have already indicated, but few cases of uncomplicated silicosis come under observation in this country unless sought for. Cases in which tubercular infection is fairly recent may come under notice, and these cases present great difficulty, particularly if tubercle bacilli cannot be found in the sputum,—the heart and liver dulness, owing to some degree of emphysema, may be found normal, the supervention of tuberculosis may have modified the high blood pressure, and also have led to an increased air capacity.† Such a case, then, may only give a history of loss of weight and cough, and may present no definite physical signs. Summons, however, has pointed out (*49b*) that assistance can be obtained from radiography; for the fibrosis, pathognomonic of silicosis, throws distinctive shadows. These shadows are similar to, but more marked than, those seen in radiographs of disseminated pulmonary tuberculosis; but their presence in cases in which no clinical symptoms of tuberculosis can be detected negatives the suggestion that they represent tubercular foci. Radiographs, taken by Dr. R. Haliam in 1911, of two Sheffield grinders then at work and in apparently good health, and who were found two years later to be still at work and still free from any indisposition, show such shadows, which appear in all radiographs of men exposed for prolonged periods to silica dust. Sir Douglas Powell has described them (*98*) in a miner returned from South Africa, in whom, though fibrosis was readily detected, no sign of tuberculosis could be found. Dr. A. Watt, of the Simmer and Jack Hospital, Transvaal, has obtained from gold-miners a series of radiographs in which the shadows are more marked as the degree of fibrosis is more pronounced. And Dr. Barwise has observed them in an apparently healthy Derbyshire sandstone-mason. These shadows have not been found in radiographs of men employed in dusty industries when the dust does not contain free silica; the shadows seen in those exposed to emery and glass dust (*v.* previous lecture) are quite different; while Dr. Barwise failed to see any shadows at all in the lungs of a Derbyshire limestone-mason. In a person exposed to silica dust the presence of such shadows, in the absence of any obvious signs of tuberculosis to account for them, may be of great value in diagnosing silicosis, and in such a person any marked loss of weight should raise the suspicion of tubercular infection, and call for careful investigation and guarded prognosis. When the tubercular condition is so advanced as to be easily recognised, the fact that it is implanted upon silicosis becomes of less importance, except in so far as this knowledge must increase the gravity of the prognosis, and influence any advice given as to suitable employment.

Pathology.—The condition of the lungs found after death, although it has been carefully described by many pathologists, requires consideration in the light of recent work, particularly that of Mr. Shattock (*99*) and of Watkins-Pitchford (*44*). On opening the chest in a marked case of silicosis the lungs do not fall away from the chest wall; they may even bulge forward as though they had previously too little room—such cases occur particularly in the massive fibrosis of South African gold-miners, but in other occupations with less intense exposure to dust, and among older men, contraction of the fibrous tissue lessens the bulk of the lungs and an atrophic cirrhosis results. Pleuritic adhesions are always present, and the pleuræ are markedly thickened. When removed from the chest, usually a matter of difficulty, the lungs may be so firm and dense as to stand on their own base like a plaster cast. When cut into the

† Of this I am not quite certain, but I have noted tha in definite cases of tubercular silicosis, the air capacity is usually greater than might have been anticipated taking into consideration the length of exposure to dust inhalation. For the determination of this point observations are required on the air capacity of the same individual before and after the onset of tuberculosis.

knife grates on densely resisting tissue, a point first remarked by Diembroek, and I have seen in Sheffield specimens which required a saw to cut them. This stony hardness is not found when the fibrosis is massive and of rapid formation, and in such cases Watkins-Pitchford says (44), "The most I have observed is that the edge of the microtome razor sometimes suffers during the subsequent preparation of sections." Although microscopic examination usually reveals cavities greater in number and in size than were detected during life, cavity formation is not so marked a feature of silicosis as of ordinary phthisis; nor does the presence of cavities necessarily indicate the presence of tubercular infection, for necrosis with cavity formation is sometimes caused by interference with the circulation due to fibrous contraction; such cavities may be distinguished from tubercular cavities by their more ragged outline and by the absence of any lining wall of inflammatory tissue. Emphysema of the free margins of the lungs, though not unusual, is seldom marked. In a section the naked eye detects many nodules, rather larger than tubercles, which are found to be especially dense and hard; in the initial stage these nodules in distribution and size closely resemble the black spots seen in coal-miners' lungs, and they stand out as islands surrounded by normal tissue; they indicate deposition of dust particles associated with pigment, probably soot particles, in blocked lymph channels. Such deposition appears first round the bronchioles, and is centred on the smaller blood vessels, and Watkins-Pitchford† calls this the first stage. Next these islands extend and coalesce until they form the background against which the normal tissue appears as islands; this is the second stage. Now a new thing appears—grey, firm, dense tissue—at first in islands or nodules corresponding in position with the black islands of the initial stage; such islands or "silicotic nodules" may have a diameter of 10mm., and when fully developed they occupy the position of a lobule of the lung. These islands, in their turn, spread and coalesce, until on section the lung tissue resembles a piece of grey granite. No sign of an alveolus is to be seen in this grey tissue which "has not the slightest resemblance in texture, consistence, or colour to the lung tissue in which it has developed" (44); and in a section seen under the microscope the obliteration of alveoli over large areas makes it difficult to understand how such tissue can have served any useful function. In a case of massive silicosis this tissue can hardly be distinguished from a fibroma (see fig. 29a), and suggests the presence of some influence stimulating rapid

Table 14. AMOUNT OF SILICA AND ALUMINA IN 100 PARTS OF CERTAIN DRIED LUNGS

MATERIAL	* A Tailor aged 23	† Male Zulu aged 30	White † GoldMiner (Transvaal)	Slate Miner ††	
				Interior portion	Exterior portion
Total Ash containing	6·68	4·96	9·30	8·79	10·39
Silica	0·90	0·73	4·47	3·97	4·63
Alumina	—	0·27 §	0·90	2·38 §	2·74 §

§ The amount of Alumina present suggests that in these lungs the Silica was present as Aluminium Silicate.

Fuller details of inorganic analyses of lungs are to be found in:—

(1) *Deutsch. Archiv. für klini-che medecin. Kussmant. Bd. 2. 1867 pp. 89-115.
(2) Transaction of Pathological Society of London, Vol. xx. 1869 H. H. Greenhow.
(3) Annual Report of Chief Inspector of Factories for 1900, pp. 491-4. (Cd. 668.)
(4) Miners' Phthisis at Bendigo, W. Summons, 1907. Stillwell & Co., Melbourne.
(5) Bulletin Mens de la Societ. d'étude scientifique de la tuberculose, Albert Robin, Feb. 1907, Paris.
(6) Oppenheimer, Handbuch der Biochemie. Pincussohn.
(7) †The Ash of Silicotic lungs, J. MaCrae. The South African Institute for Medical Research, 1913.
(8) ‡Minutes of Evidence of Royal Commission on Metalliferous Mines and Quarries, Vol. iii, pp. 231-2, 1914. (Cd. 7478.)

† Dr. Watkins-Pitchford's original work is here largely followed, because the intense exposure in the Transvaal mines has given him unique opportunities, of which he has taken every advantage of working out the pathological changes in their most acute form, while former observers have had to rely on the more chronic and atrophic forms of fibrosis.

Fig. 16 (*a*)
Microphotograph from Lung of a Staffordshire Potter.

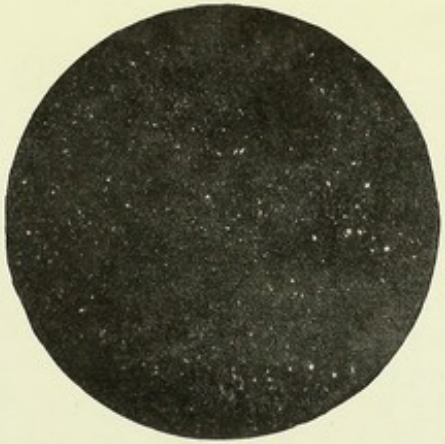


Fig 16 (*b*)
Same Field seen by polarised light.

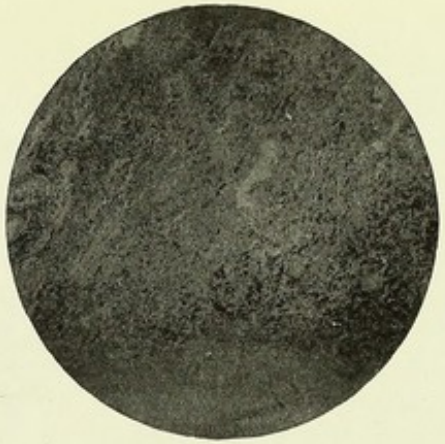


Fig. 17 (*a*)
Microphotograph from Lung of Ganister Worker.

Fig. 17 (*b*)
Same Field seen by polarised light.

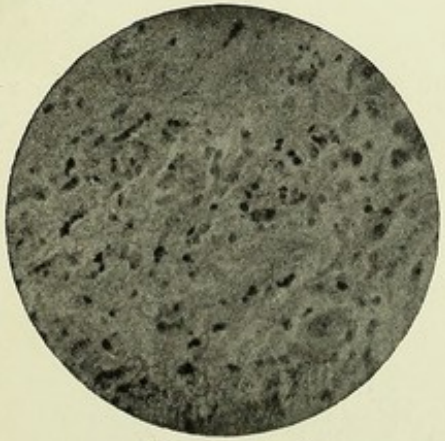


Fig. 18 (*a*)
A part of Fig. 17 under higher magnification.

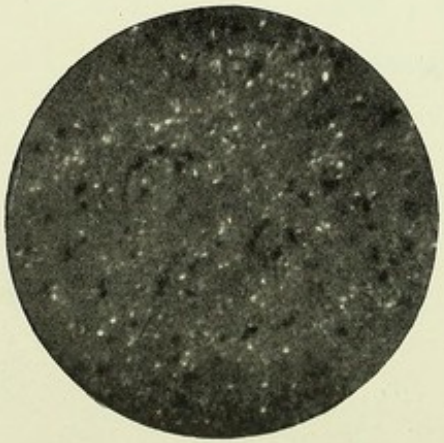
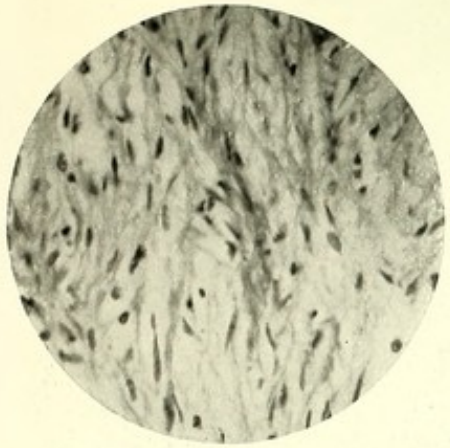


Fig 18 (*b*)
Same Field seen by polarised light.

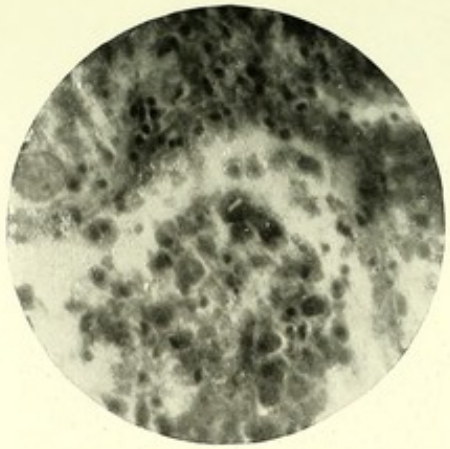
Fig. 19 (*a*)
Microphotograph from Lung of Transvaal Gold Miner.

Fig. 19 (*b*)
Same Field seen by polarised light.

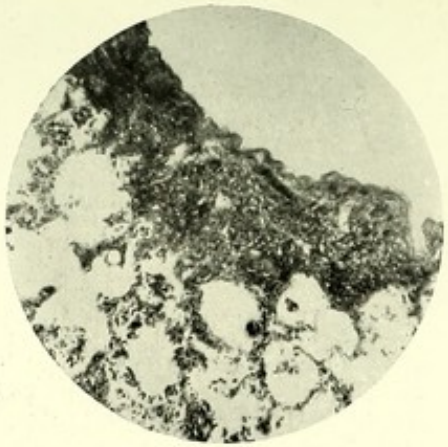
(*Figs. 16 to 19 taken by Mr. Mead, St. Thomas's Hospital.*)



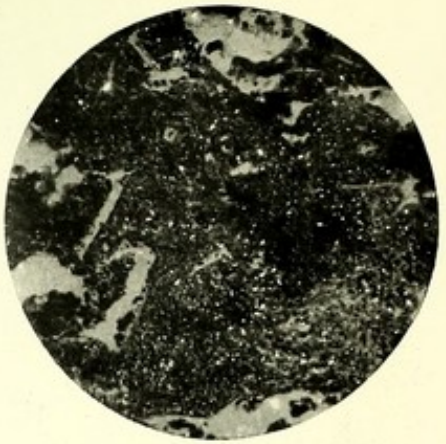
(*a*) The new tissue of a so-called "Silicotic Nodule"; mainly composed of long branching cells, suggestive of a fibro-sarcomatous growth.



(*b*) † Alveolus with cellular contents; one of the cells in the cavity of the alveolus bears an elongated particle of silica.



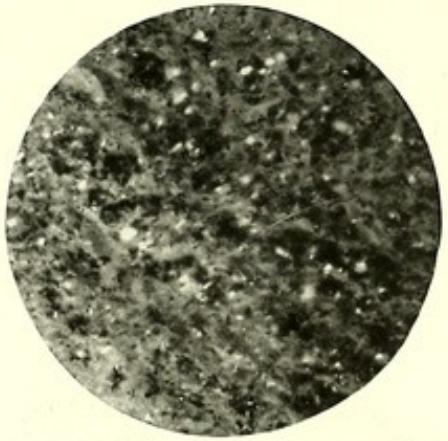
(*c*) † Wall of bronchiole in early, acute silicosis. The new tissue which has developed in the fibro-elastic coat of the bronchiole bears numerous particles of silica and is encroaching upon the adjacent alveoli.



(*d*) † Lung tissue in middle stage of silicosis. The new tissue which has produced an extensive interstitial fibrosis is laden with siliceous particles.

(*e*) One of the central regions of a light grey area. Incarceration of extraneous pigment in a filigree of new connective tissue.

(*f*) † Same specimen as (*e*); showing the close association of silica particles with extraneous pigment.



(*g*) † Lung tissue in the late stage; there is a loss of all morphological detail; the silica particles, with blurred outlines, lie in a necrotic matrix which will shortly undergo liquefaction; nuclei and nuclear fragments are scattered throughout the field.

(*h*) † Bronchial lymph node. The node has undergone first a fibrosis, then a necrosis. Silica particles, with blurred outlines, are sparsely scattered throughout the field.

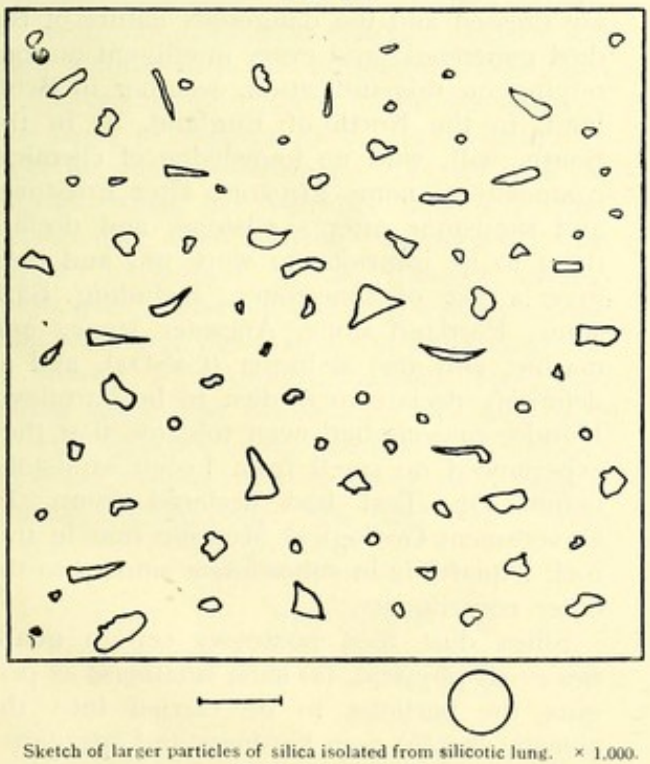
† Taken by double exposure, first by ordinary illumination, and then by polarised light.

FIG. 20.—Microphotographs from Transvaal Gold Miners.

(Courteously provided by Dr. Watkins-Pitchford.)

overgrowth of connective tissue cells. Even in the atrophic cases he examined Greenhow detected (*83*) this fibro-nucleated tissue, and ascribed it to a new growth of connective tissue. Shattock, however, has so recently carefully traversed (*99*) this subject, describing accurately the microscopic appearances of lungs exposed to various dusts, that further detail may be omitted here. The gritty nature and chemical analysis of such tissue demonstrate that these divergencies from normal are produced by silica dust permeating the lung tissue; and this may be demonstrated by examining a section with polarised light, a method of examination which was, I believe, first used in the careful clinical and experimental inquiry carried out in France into the effect of inhaling plaster-of-Paris dust (*56*), and which is now regularly employed in South Africa. As early as 1865 Greenhow used polarised light (*83a*) to identify silica in dust particles recovered from a metal-grinder's lungs, but there is no record that he detected the particles *in situ*. By this method silica particles can be detected within phagocytic leucocytes, and there can be little doubt that such leucocytes remove these particles from the alveoli, and perhaps through the walls of the finer bronchioles, into the lymph channels and so to the endothelial and connective tissue cells. The particles so detected (*46*) vary in size, 70 per cent. are less than 1μ in diameter, and 30 per cent. vary from 2μ to 10μ in maximum diameter; in shape they are usually roughly spherical, but elongated acicular forms occur and are found in the lungs in greater proportion than in dust, a point which may indicate either some selection by the phagocytes, or some filtering action as the dust passes through the air-passages into their final ramifications. Fibrosis of the lungs results from other pathological conditions than silicosis, but this effect with polarised light will only be obtained when certain dust particles are present. Whatever difficulties of diagnosis, then, may present themselves during life, after death a definite pathological picture is present, except for a few borderland cases, leaving no doubt as to the part played by the inhalation of silica dust in contributing to the fatal issue. Further pathological work, however, is required upon lungs exposed during life to other dusts, such as the dust of glass, emery, carborundum, corundum, cement, slate, clay, cotton, hemp, and jute, to ascertain what changes dusts other than silica originate. Certain experiments have been carried out (*77a*) by Professor Beattie by exposing animals to atmospheres containing various kinds of dust. When summarising this work the Royal Commission on Metalliferous Mines and Quarries reported that "after varying periods of exposure to dusts suspected to be dangerous in view of the phthisis mortality statistics among those who work in them, the animals, guinea-pigs, were found to develop a fibrous condition of the lungs similar to that found in the lungs of operatives who inhale these dusts, but exposure to dusts thought to be innocent because no excess of phthisis is found in those who are exposed to their inhalation, either did not cause this condition to develop or only to a moderate degree. Professor Beattie found a few exceptions which call for further investigation" (*1b*). This line of research is being followed up by Professor Beattie at Liverpool and by Dr. Haldane at Oxford. There is, however, no evidence, clinical or statistical, to suggest

Fig. 21.



Sketch of larger particles of silica isolated from silicotic lung. × 1,000.
The line represents 10μ.
The circle represents a red blood corpuscle.
(Reproduced from "The Ash of Silicotic Lungs." John McCrae).

that workers exposed to such "innocent" dusts suffer in excess from tuberculosis, and until the presence or absence of fibrosis in the lungs of such workers is demonstrated, speculation is idle as to whether the predisposition of individuals with silicosis to tuberculosis depends on the fibrous changes in their lungs, or on the presence of silica in such fibrous tissue. The statistical latent period, however, previously alluded to, a period during which the fibrous change is developing, suggests that impaired vitality consequent upon fibrosis is the determining factor. Still the question remains unanswered: How does silica alone among dusts stimulate the formation of fibrous tissue?

CHARACTERISTICS OF SILICA.

Compounds of the element silicon are necessary to the vegetable kingdom, but in the animal kingdom they occur in mere traces. In nature silicon is found in the form of (i) silica or oxide of silicon, SiO_2, which exists in crystalline and amorphous forms; the best examples of the crystalline form are rock-crystal, quartz, quartzite, chalcedony, flint, sandstone, and quartzose sand, which, taken together, form a large proportion of the earth's crust, and this is the form of silica of which the dust is found to be specially injurious; the best examples of the amorphous form are opal and diatomaceous earth (and possibly silica in vegetation), but opportunities of studying the effects of dust arising from these forms are difficult to obtain and no data are available; and (ii) silicates of which clay (aluminium silicate) is an example; no high mortality from phthisis has been found among those exposed to dusts of silicates, indeed makers of ordinary clay bricks have an unusually low mortality from phthisis.

Of these compounds we are at present only concerned with crystalline silica. Chemically, it is an extremely stable and insoluble substance, possessing, however, acid properties; and physically it possesses the property on fracture of breaking up into very fine particles, a number of which are sharp-pointed and of acicular form; this form is found more frequently in the lungs than in dust in which it is greatly outnumbered by other shapes. Silica, considered as a mineral, contains liquid and gas inclusions (cavities); and also possesses another curious property which may be stated in the words (*84*) of Sir Ray Lankester: "Another 'smell' which is extremely mysterious is that produced by two quartz-pebbles, or even of two rock crystals, or two pebbles of flint or of corundum, when rubbed one against another. A flash of light is seen, and this is accompanied by a very distinct smell, like that given out by burning cotton wool. It is demonstrated—by careful chemical cleaning before the experiment—that this is not due to the presence of any organic matter on or in the stones or crystals used. It seems to be an exception to the rule that 'odour' (as distinct from pungent vapours or gases) is only produced by substances formed by plants or animals. . . . In any case it seems, according to our present knowledge, that the smell given out by the rubbing of pieces of silica (quartz, flint, etc.) is due to particles of silica (oxide of silicon) volatilised by heat of friction, which are capable of acting specifically on the olfactory sense organ." Interest attaches to this characteristic smell, for operatives, especially masons, have long recognised a connection between the smell of what they term "sulphur" given off when certain stones are dressed and the dangerous nature of the dust generated; and every intelligent mason, relying on this indication, whether in Scotland, in the North of England, or in the South, will, with no knowledge of chemical composition, name gritstone after gritstone, and sandstone after sandstone, and declare them to be injurious to work on, and then give a list of limestones, including Bath stone, Portland stone, Ancaster stone, and marble, and also alabaster ($CaSO_4$), and as definitely declare their dust to be harmless; Dundee masons had even told me that they experienced no smell from Leoch sandstone before Dr. Teal had declared from the Government Geological Museum that in this rock "quartz is in subordinate amount to the other constituents."

Silica dust then possesses certain qualities:—(i) physical, (*a*) such smallness as permits the particles to be carried into the alveoli, and (*b*) such hardness and angularity as suggest that the particles can act as centres of irritation; and (2) chemical, (*a*) acidity

which, owing to the presence of the element silicon, may render the particles capable of entering into and modifying the colloidal structure of protoplasm, and (*b*) smell, possibly due to a vapour, as yet undetermined, given off when silica is fractured. Only further investigation can determine which it is that leads the pulmonary connective tissue to proliferate, and whether the undoubted predisposition to pulmonary tuberculosis caused by inhaling silica dust is due to this proliferation.

CONCLUSION.

I have attempted to justify the claim that dust inhalation plays an important part in determining the occurrence of respiratory diseases—some dusts, such as coal, it is true, not only appear to have no power of producing pneumonoconioses, but even may possess some inhibitory influence on phthisis; other dusts, such as limestone and plaster-of-Paris, are negative in their action; but most dusts have an injurious influence, and of all dusts that of silica is the most injurious. I have also been tempted to wonder whether recognition of the especially dangerous qualities of silica dust is a new thing, and whether the great Rabelais, who, you will remember, was a physician as well as an author, and who made his heroes kill their victims with full anatomical detail, had not some inkling of these differences between the dusts of various rocks, and whether in 1533 he was presenting an allegory in Pantagruel when he describes (*85*) how that personage, after slaying Loupgarou, the captain of certain giants, "smote among those giants who were armed with freestone and beat them down as a mason does knobs of stone. . . . Pantagruel struck down one whose name was Maulchitterling, who was armed cap-a-pie with gritstone . . . otherwise the greater part of them were lightly armed, that is, with tufa, and others with slates."

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