The industrial diseases of South Africa : being the annual lecture of the Cape of Good Hope (Western) Branch of the British Medical Association / by W. Watkins-Pitchford.

THE INDUSTRIAL DISEASES of South Africa,

BEING THE

ANNUAL LECTURE

OF

The Cape of Good Hope (Western) Branch of the BRITISH MEDICAL ASSOCIATION,

BY

W. WATKINS-PITCHFORD, M.D., F.R.C.S., Etc.

Delivered at Cape Town
February 9th, 1914.

[*Reprinted from The Medical Journal of South Africa*]

JOHANNESBURG:
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The Industrial Diseases of South Africa.

By Dr. WILFRED WATKINS-PITCHFORD, South African Institute for Medical Research, Johannesburg.

Gentlemen,—By inviting me to deliver the Annual Lecture of the Cape of Good Hope (Western) Branch of the British Medical Association your Council has conferred a distinction upon me which I greatly appreciate. Despite the fact that Dr. Simpson Wells has given me a free hand in the matter, the selection of a subject on which to address so critical an audience has been a matter of some difficulty. Your last Annual Lecture was entrusted to Sir Almroth Wright, who discoursed to you upon "The Principles of Vaccine Therapy." I have been tempted to follow the lead thus given and relate to you our experiences with the vaccine therapy and vaccine prophylaxis of pneumonia on the Rand. The recent publication by the Institute of Dr. Maynard's memoir will, however, make many of you familiar with this topic, and I must therefore go further afield.

The gold-mining industry of the Witwatersrand, upon the successful prosecution of which the prosperity of our country, at the present time, so largely depends, is hampered by other diseases than pneumonia, and foremost amongst these other diseases is pneumonokoniosis. As this complaint is also believed to affect the ostrich-feather industry I have been led, by a natural process of thought, to adopt as the subject of my discourse to-night "The Industrial Diseases of South Africa." Such a title has the advantage of sounding impressive, whilst the subject has the advantage of being of very limited dimensions. The facts available are, however, not quite so limited as those which formed the basis of the celebrated treatise on "The Snakes of Ireland."

An industry may be defined as a particular class of productive work, and an industrial disease as a disease which causes disablement of the worker, and which arises out of, and in the course of, employment in an industry.

With these definitions before us we have now to ascertain what are the more important industries of South Africa and what disabling diseases arise from their prosecution. We have, at the outset, to ask ourselves by what title any one industry is to be considered more important than another. Brief reflection shows us that those industries which a nation carries on merely in order to supply its own wants—that is to say, its home industries—must have a strictly limited development; it is obvious, moreover, that a claim by any one of such industries to be of greater importance will be at once challenged by the others. No productive occupation can preponderate over others in a country unless the products of the industry exceed local requirements and are exported to other countries—in other words, unless it has risen to the grade of a commercial industry.

Civilisation is now so advanced and widespread that it is doubtful whether, in the future, any nation will be permitted to enrich itself by mere conquest, yet the fundamental law remains unrevoked that without either conquest or commerce a nation must degenerate and die. Those industries which enable a nation to participate in the international traffic in goods are therefore of vastly greater importance to it than its home industries.

Guided by such principles, we conclude that the important industries of South Africa are those which provide the community, either directly or indirectly, with the materials for its export trade.

For the ten months ending October 31st, 1913, our leading exports, arranged in the order of their value, were gold, diamonds, wool, ostrich feathers, hides and skins, coal, and goat hair. The approximate relative value of these exports—taking the value of the total exports at 100—were: gold 57, diamonds 19, wool 7, ostrich feathers 4, hides and skins 3, coal 2, and goat hair 1·5.

When we come to apply our definition of an industrial disease—as a disabling disease which arises

out of and in the course of employment in an industry—to practical purposes we find that it requires some further refinements. Lobar pneumonia is the scourge of the native labourers—more especially those from the East Coast and Tropical regions—when brought to the Rand for work in the gold mines; tuberculosis is also much more prevalent among our mine "boys" than among many of the native communities from which they are recruited. Are we justified, on this account, in considering lobar pneumonia and tuberculosis as industrial diseases on the Rand? The fact that these boys would probably not have developed these diseases if they had not obtained employment on the goldfields certainly entitles us to consider the diseases as having arisen "out of and in the course of their employment." Neither of these diseases is, however, *specific* to the gold-mining industry; others besides mine boys develop tuberculosis, and the liability to pneumonia of all new arrivals on the Rand, whether they be workers or not, appears to be temporarily increased. Amongst native labourers from tropical districts the disease is most rife immediately upon their arrival and before they have proceeded to the mines. The last-named fact, by itself, refutes the prevalent assumption that the disease is due to some evil in the circumstances of their occupation as underground labourers.

The difficulty of more exact definition has been very largely removed for us by a British Departmental Committee which was appointed in 1906 to report what industrial diseases should be added to the schedule of the Workmen's Compensation Act. This Committee laid it down as an axiom that any disease to be added to the schedule must be *so specific to the particular employment that its causation by the employment can be established in individual cases.* It cited the example of the bronchitis to which flax-workers are specially liable, and pointed out that, as there was nothing to differentiate this form of bronchitis from ordinary bronchitis, the disease could not be scheduled as an industrial disease. It also declined to include ordinary tuberculous phthisis—on the prevalence of which occupation has such a marked influence—

among the industrial diseases, remarking that this disease is of frequent occurrence throughout the general population and is not specific to any particular employments.

A further discrimination is made by the English Act in respect to the degree of disability which justifies a claim for compensation. The disease must be such as to entail death, or to bring about suspension or absence under certificate, or prevention from earning full wages at the particular work, for a period of at least one week.

Accepting these limitations to our conception of a strictly industrial disease we may now revert to the important industries of our country, and may proceed to note against each of them any disabling diseases which arise out of and in the course of the employment, and which are so specific to the industry that its responsibilty for the disease can be established in every case.

The gold-mining industry, in the past at all events, has been responsible for the annual disablement by silicosis of a large number of workers; it has also given rise to occasional cases of cyanide dermatitis, mercurialism, and lead-poisoning. Diamond-mining appears to be entirely above reproach in the matter of occupational diseases. Anthrax is sporadically associated, from time to time, with the industries connected with the production and marketing of wool, hides, skins, and goat-hair. The ostrich-feather trade is reputed to be responsible for many cases of chronic bronchitis, and the mining and handling of coal is productive of the condition of pulmonary anthracosis.

The agricultural and stock-rearing industries, upon the development of which the future of South Africa depends so greatly, have not as yet reached the commercial, or exporting, stage and we cannot, therefore, claim that they are important industries. They are, however, ancillary to the principal exporting industries, and their occupational diseases therefore call for brief mention before we go any further. Of the three occupational diseases associated with farming occupations which call for remark in this connection we note that two, anthrax and glanders, are directly communicated from

animals, and that one, streptotrichosis or actinomycosis, is due to the inoculation of the spores of more than one variety of a fungus which is saprophytic on grain. We shall speak of anthrax hereafter, as it is a disease associated with an exporting industry. With respect to glanders and the streptotrichoses, it may be safely asserted that they are of rare occurrence. I have had occasion to diagnose only one case of each of such diseases, affecting the human subject, during twelve years in South Africa.

Although our home industries and trades are, in principle, of secondary importance to our exporting industries, and although they are, unfortunately, but poorly developed, their special diseases are the care of the profession and will require to be recorded and provided for from time to time. Instances of this class of occupational disease are, as yet, quite rare; it is significant, however, that the Medical Officer of Health for Johannesburg makes special reference in his last annual report to the subject of plumbism amongst linotype operators.

When we compare the list of our industrial diseases with that of the diseases scheduled under the Workmen's Compensation Act of Great Britain we are at once struck by the meagreness of the former. We must, indeed, confess a certain amount of affectation in compiling any such list, for in South Africa, as we all know, we have but one industrial disease of any importance. It is useful, however, for us to appreciate the principles which underlie a subject of so great importance in other parts of the Empire, and it is well for us to be prepared in our opinions concerning diseases which may at any time become the subjects of popular attention.

The scarcity of occupational diseases in South Africa is due, partly to the paucity of unhealthy manufacturing industries, and partly to the relative sparseness of the population. The specific population of England and Wales (1911) is about 618 (in other words, each square mile of country carries 618 human beings), the manufacturing industries are more developed than in any other part of the world, and agriculture is decadent. In the Union of South Africa (1911) there are only about 12 people to each

square mile (only two of these 12 being white persons), and the agricultural industry is in the ascendant. Such facts as these justify a stringent legislation in thickly-populated, manufacturing countries on behalf of their industrial classes; they also make it apparent, however, that the same degree of stringency is not justified in the industrial legislation of a sparsely-populated country which is capable of great agricultural development.

The relation between density of population, the vogue of manufacture, and the prevalence of occupational disease is undoubtedly very intimate. In this connection it is of interest to note that with a specific, white, population of 466 (as compared with the 2 of South Africa), the Kingdom of Holland (1909) has, of late years, developed many important manufacturing industries, and is now provided with several restrictive legislative enactments for the protection of the health of the industrial worker.

The gold-producing industry of South Africa is responsible for no serious occupational disease other than silicosis. One hears, however, of occasional cases of brief disablement from cyanide dermatitis, mercurialism, and lead-poisoning. The dermatitis takes the form of an acute, eczematous affection of the hands and forearms, due to their too frequent or prolonged immersion in cyanide solution. This may happen to those workers in the extractor house whose duty it is to remove the zinc shavings from the boxes through which the cyanide solution flows. The accident may be prevented by the wearing of rubber gloves. The mercury contained in the gold amalgam, as it leaves the amalgamation house, is separated by distillation in iron retorts fitted with condensers for the recovery of the mercury. Any defects in the apparatus, or carelessness in manipulation, may expose the workers in the smelting house to mercurial vapours; cases of mercurialism arising from such causes are, however, very rare. Of equal rarity is lead-poisoning, which, when it does occur, is attributable to the fact that litharge is used as a flux in the reverberating furnaces by which the precious metal is separated from the cyanide-gold precipitate.

The cutting up of carcases, and the handling of the wool, hair, or skins of animals dead of anthrax may infect the workman, but it is unnecessary for me to remind you of the pathology of this disease, as it affects the man, in either its external or internal forms. Sheep, goats, cattle, and horses are all very liable to anthrax, and outbreaks of the disease among stock occur periodically in all parts of South Africa. The disease is so rapidly fatal that death is frequently attributed to the effects of poison, especially when a large part of a flock or herd is fatally affected almost simultaneously. In such circumstances the owner's curiosity as to the cause of death is likely to be sternly repressed and the financial disaster mitigated by the sale of the wool, hair, and hides of the dead animals. Needless to say, such action, when the real nature of the disease is known, is strictly prohibited by the Veterinary Regulations. When once such contraband merchandise has passed into the possession of the dealer it becomes a potential source of disease to all who handle it.

In my own experience in South Africa, although I have seen and diagnosed many cases of malignant pustule in man, I have not heard of any one terminating fatally. In all cases the treatment has been, I believe, free excision and the application of germicides to the raw surface. All instances of the internal form of the disease with which I have had to do have, however, proved fatal. In this form the illness is very acute, with high fever, great prostration, and early and unexpected death. The nature of the illness is very often not discovered, or even suspected, until a post-mortem bacterioscopic examination has been made.

Rumours have been heard of late of the prevalence of chronic bronchitis and an undue mortality from phthisis amongst the feather-sorters of certain towns of the Eastern Province. This is a matter about which it is very difficult, at present, to get statistical information. Dr. J. Galloway, the Health Officer for Port Elizabeth, informs me that a chronic bronchitis attributable to their occupation is certainly to be found amongst the feather-sorters of his district.

These people are mostly Eurafricans, and their complaint almost always passes by insensible gradations into pulmonary tuberculosis. Dr. W. Gilbert, the Resident Medical Officer of the Port Elizabeth Hospital, has also observed many cases of feather-sorters' phthisis, but thinks that the early stages of the disease are those of a broncho-pneumonia rather than a bronchitis, and that such broncho-pneumonia appears to be primarily non-tuberculous. Dr. G. Porter Mathew considers that the occupation is also responsible for a troublesome form of rhinitis. Popular opinion seems to be that the mischief is done more by the dust which is shaken out of the feathers than by the liberated particles of "dons" or fluff; this, however, is a speculation and not an ascertained fact. That the air of the feather-market during the sales abounds in particles of floating down is quite obvious to the casual observer.

As it is an acknowledged fact that the habitual inhalation of particles of cotton, flax, and wool (in the spinning and weaving industries of Great Britain) gives rise to chronic bronchitis in the worker, it is probable that these rumours and opinions concerning the presence of feather-sorters' phthisis in South Africa are well founded. Whether or not the complaint can be justly deemed a strictly industrial disease future investigation can alone decide.

Coal-miners and bunkerers—more especially when the softer varieties of coal are dealt with—are liable to very extensive infiltration of the lung tissues with coal dust. This condition is known as pulmonary anthracosis. Although it is undoubtedly an abnormal one yet it is probably not correct to consider it a disease. It is not productive of extensive perilymphatic induration, nor of generalised fibrosis of the lungs, and this peculiarity is probably the reason why it does not predispose to a pulmonary tuberculous infection. The coal-miners of England and Wales share with agricultural labourers the privilege of being recorded as the least liable of all industrial classes to suffer from phthisis. Dr. F. J. Allen, who has been Medical Officer to several of the leading Transvaal collieries for ten years, informs me that although an excessive degree of

anthracosis is quite common it does not impair the health or capacities of the worker in any way. With a knowledge of such facts the claim of anthracosis to be considered one of our industrial diseases falls to the ground. I have referred to the condition because much misunderstanding exists with respect to its significance, and also because observations which disprove our suppositions are of as much value to us as those which verify them.

Diseases due to the deposit in the lung tissues of particles of foreign matter which have been inhaled were classed by Zenker under the general title of "pneumonokoniosis" (πνευμων — the lungs, κονισ—dust). The necessity for differentiating such diseases according to the nature of the foreign particles has given rise to a series of impressive titles. Thus from flinty or siliceous particles we have chalicosis, or silicosis; from clay dust—aluminosis; from particles of steel, iron, or iron oxide—siderosis; from tobacco dust—tabacosis; from fragments of cotton or linen fibre—byssinosis, and so on. If it be necessary to differentiate that variety of pneumonokoniosis due to particles of feather and down we must ourselves resort to the puerile pastime of word-making. Perhaps we have been unwittingly forestalled in the effort, but I suggest "ptilosis" (πτιλον — down) as an appropriate designation for the condition.

The pulmonary changes which characterise all the pneumonokonioses are essentially the same, though varying widely in degree, and range from chronic bronchitis to complete destruction of the lung by fibrosis. It appears to be a general rule that dust composed of particles of organic matter originates more especially a chronic bronchitis, whilst hard mineral dust gives rise more particularly to interstitial pneumonia or fibrosis; in the lung of the corn-miller we find much chronic bronchitis, bronchiectasis, and emphysema, whilst the lung of the banket gold-miner is essentially a fibrosed organ. The different varieties of mineral dust, again, differ very widely in their capacity to produce pulmonary fibrosis, and it may be stated, in general terms, that minute, hard, and angular fragments are more noxious in this respect than larger, softer, and more

amorphous particles. Coal-dust produces, as already stated, relatively little fibrosis, whilst the particles given off in the grinding of steel and glass, or in the drilling and cutting of flint, are responsible for the development of the condition in its highest degree.

The severity and duration of pneumonokoniosis, and the degree to which a tuberculous infection of the lung is facilitated, depend not only upon the chemical and physical characters of the inhaled particles but also upon the general health and environment of the patient; the resistance to the disease of those who are well-fed and well-housed and who regularly indulge in open-air sports is very marked when compared with those who are less fortunate in their environment and inclinations.

All forms of dust are not necessarily injurious to the lungs; coal and chalk amongst minerals, and starch amongst organic substances, may be inhaled in the form of dust with relative impunity. With respect to starch-dust, Kauffmann (J. of Hyg., 1909, p. 220) has produced evidence to show that the inhaled granules become converted into soluble starch and sugar by the action of amylolytic enzymes. This harmlessness of starch-dust stands in marked contrast to the hurtfulness of flour.

The primary cause of the development of fibrous tissue in the lungs in certain classes of pneumonokoniosis is the local irritation due to the presence of particles of foreign matter in the lung tissues. This, however, is not the only cause, for the chronic bronchitis, chronic broncho-pneumonia, and chronic congestion which accompany, in varying degrees, the different forms of the disease are, by themselves, capable of producing an interstitial fibrosis. A secondary fibrosis of the lung is, indeed, quite commonly found to occur independently of the inhalation of foreign particles; it is found either as accompaniment or sequel of passive congestion of the lungs, and of various infective processes such as lobar pneumonia, catarrhal pneumonia, and tuberculous and syphilitic disease of the organ. It is probable that extensive pulmonary fibrosis, with whatever disease it may be associated, results in part from obstruction of the lymph circulation. In

some cases of elephantiasis—a disease which is characterised by extreme hyperlasia of the cutaneous and subcutaneous connective tissue—an obstruction and dilatation of the lymphatic channels can be demonstrated. In the pneumonokonioses, however, the lymphatic channels are not dilated, for their obstruction is not due to the plugging of their channels by foreign bodies—the channels are obliterated by the hyperplasia and induration of the normally loose and delicate tissues through which they pass. This peri-lymphatic induration is a local process due to the irritation of the connective tissue by foreign particles in its interstices; having once formed, it tends to obliterate the lymph channels, and thus a far more extensive hyperplasia due to lymphatic obstruction may be originated.

We may now turn to a more detailed consideration of pulmonary silicosis, a disease which both from its prevalence and its economic importance at the present time may be justly termed "the industrial disease of South Africa."

Prevalence.

The Transvaal Miners' Phthisis Commission of 1903 was of the opinion that probably 21 per cent. of all the underground miners were actually or probably affected with silicosis. A similar Commission in 1912, after examining 3,136 underground miners, found that 990, or approximately 32 per cent., were suffering from miners' phthisis in one or other of its stages, and that about two-thirds of the cases were in the stage of early or moderate fibrosis. Owing, however, to the very different manner in which the investigations of these two Commissions were carried out their respective estimates cannot be justly compared. It may nevertheless be remarked that although the general conditions of underground hygiene were better in 1912 than in 1903 yet the number of rock-drills employed had more than trebled in the interval. In December, 1905, 1,800 of these machines were at work, whilst in December, 1912, there were 5,600.

For the year 1912 the average monthly number of underground White miners on the Witwatersrand was 11,607, and in this year 161 White miners died

in the Transvaal of miners' phthisis or silicosis. It is quite certain that all the deaths of Europeans during 1912, from silicosis contracted on the Rand, did not occur in the Transvaal, for a considerable number of Europeans return to their mother country when they find themselves invalided; even if we ignore this source of error we find that the death-rate from this disease amongst the White underground workers was 13·8 per thousand for the year.

For the same year the average monthly strength of the Native underground mining population was 149,782. The total Native mine population was 192,522, and from this 153 deaths were registered during the year as being due to silicosis or miners' phthisis. With respect to the Natives this mortality figure is, however, even more misleading than in the case of the Europeans. When the health of any mine "boy" begins to fail he is paid off, and, if fit to travel, is sent back to his own home. In the great majority of instances this means that the Native leaves the Transvaal. The number of Natives thus repatriated during 1912 on account of ill-health from all causes was 9,251, being at the rate of 48 per thousand per annum.

For the first ten months of last year there was a monthly average of 11,840 White miners engaged in underground work on the Rand, and if the proportion of 32 per cent. for those affected with miners' phthisis still held good we may conclude that some 3,700 of these were affected with silicosis.

The Miners' Phthisis Sanatorium, erected in the neighbourhood of Johannesburg for the gratuitous reception and treatment of sixty sufferers from silicosis, was opened in November, 1911. During the year 1912, 131 patients were admitted, 46 died, and 67 were discharged. For the year 1913 the figures were—154 admitted, 46 died, and 103 discharged.

Such facts as these lead us to the conclusion that, despite the devotion of mine managers, the labours of Commissions and Committees, and the endorsement by the Government of numerous restrictive regulations, silicosis still remains a trammel and a menace to the gold-producing industry of South Africa. This fact is a paradox, for, as we shall shortly see, the disease is now a preventible one.

The explanation of such a state of affairs is probably to be found in the irrepressibility of a certain human weakness—the unreasoning desire to draw big wages even at the risk of health and life, which are worth more than gold.

Etiology and Prevention.

The prevailing rock of the Witwatersrand is quartzite—a very hard rock which is essentially a siliceous sandstone which has become hardened and solidified by metamorphosis. Into this quartzite the shafts of the gold mines are sunk, and in it the tunnels are driven. Many of the shafts are several thousand feet deep, and the drives or tunnels of a single mine may aggregate many miles in length. The quartzite is broken here and there by "dykes" of igneous rock, usually diabase, and it is underlain, at some depth, by strata of hard shale or schist. In certain of the mines of the most easterly part of the Rand dolomite, or magnesian limestone, overlies the Main Reef Series, and through it the shafts have to be sunk. Interbedded in the quartzite is the Main Reef Series, which consists of two or more shallow beds of conglomerate, locally known as "banket"; these beds are parallel with and near to one another, and are inclined at an agle of from 10° to 90°. Their upper edges reach the surface, and they extend downwards to an unknown distance into the earth. This conglomerate consists of quartz pebbles, and grains and pellets of pyrites, all of which are cemented into one solid mass by silica. It is in the material which cements the pebbles together, and in the pyrites, that the gold, usually in a finely divided state, is found.

Deposits similar in character to the Witwatersrand banket occur in other parts of the Transvaal, in the Orange Free State, in Zululand, and on the Gold Coast. In Rhodesia the gold is, as a rule, found in veins and masses of quartz.

The whole scope of the gold-producing industry of the Rand is to mine the conglomerate, bring it to the surface, crush it to powder, and extract its gold by the amalgamation and cyanide processes. The silicosis which is associated with the industry is

caused by the inhaling of the dust which arises when the conglomerate, as well as the various rocks which have been mentioned, are drilled, blasted, shovelled and crushed.

All foreign particles which can be inhaled and which can find their way into the pulmonary tissues must of necessity be sufficiently light or minute to float in the air, for a short time at least, and they must be sufficiently small to enter between, or be phagocyted by, the cells which line the air passages and pulmonary alveoli. For both physical and biological reasons, therefore, the size of the particles which produce silicosis, or any other pneumonokoniosis, is limited.

Although silicosis is essentially associated with the presence of excessive numbers of siliceous particles in the lung tissues, yet, as far as the silicosis of the Witwatersrand is concerned, these siliceous particles are always accompanied by minute and amorphous fragments of argillaceous, ferruginous, and carbonaceous nature. We have already remarked that the severity and duration of a pneumonokoniotic process is influenced by the physical and chemical characters of the inhaled particles. Minute splinters of hard and totally insoluble quartz or glass are probably the most potent of all forms of dust in exciting pulmonary fibrosis. Next come sharp particles of steel, and then, in descending order of noxiousness, clay, iron ore or iron rust, and chalk. Owing to their softness and capacity for being gradually dissolved in the tissues, particles of chalk give rise to practically no fibrosis. For some reason, which is not yet ascertained, particles of coal, charcoal, or soot, although apparently quite insoluble in the body, give rise to little or no pulmonary induration.

In sections of a silicotic lung those siliceous particles which are above 1μ in diameter can usually be readily distinguished from the fragments of non-siliceous mineral by the use of polarised light. This agent also shows us that the particles of quartz are characteristically present in the form of clean-cut, eliptical, acicular, chisel- or strap-shaped splinters which are sometimes slightly curved; the non-siliceous mineral fragments, on the other hand, are

usually amorphous. Crystalline quartz has, in the mass, a glass-like, conchoidal fracture, and the not uncommonly found curve in the particles in the lung is doubtless a vestige of this peculiarity. Splinters which are entirely similar, both in size and shape, can be produced by submitting quartz to great crushing force, and they are found in abundance in the finer portions of the dust which may be generated by drilling, blasting, or crushing the native rock. The conclusion, which has already been mentioned, that these operations in gold-mining are those which are specially liable to originate deleterious dust, seems, therefore, to be fully justified.

In order to break the quartzite and remove the gold-bearing quartz conglomerate, cylindrical holes, varying from 4 to 8 feet in depth, are drilled into the rock. Dynamite cartridges, furnished with a detonator and a fuse, are then thrust to the bottom of these holes, and the explosion of these charges shatters the rock. The drilling of the holes to receive the explosive charge is a slow and tedious process and constitutes the main part of the work underground. Nearly all of this work was formerly done by hand, but machine-drills (or "rock-drills," as they are technically called) are now gradually superseding the "hammer-boy," as the hand-driller is termed.

The hand-drilled hole is about 1 inch in diameter and from 4 to 5 feet in depth, and is made by gradually driving a chisel-ended steel bar, or "jumper," into the rock. This is accomplished by striking many thousands of deliberate and heavy blows upon the end of the "jumper" with a hammer, whilst with the other hand the bar is held in position and given a slight rotation between each blow. A good "hammer-boy" will bore a hole into the rock to a depth of from 4 to 5 feet in as many hours. In spite of statistical returns some mining authorities hold that the "hammer-boy's" avocation, even when pursued without precautions, is not particularly conducive to miners' phthisis. This opinion certainly receives no support from the fact that stone-masons, who follow their calling in the open air and employ only hand tools for their work, are

specially liable to develop the disease. "Stonemasons' phthisis" was indeed definitely recognised as a disease before miners' phthisis.

The machine-drills, or rock-drills, are of the percussive and not of the rotary type, and are actuated by compressed air. In the commoner variety, termed "reciprocating," the "jumper" is fixed to the piston rod of the machine which drives it against the face of the rock with great force and rapidity, rotating it between each blow. In another type of rock-drill, termed "hammer-drill," the "jumper" is pressed mechanically into the hole and periodically rotated whilst the piston delivers its blows upon its outer end. A machine-drilled hole varies in diameter from about 2½ inches at its orifice to about 1 inch at its blind extremity, and is from 6 to 8 feet deep. During an eight-hour shift a rock-drill is expected to have bored through from 22 to 50 or more feet of rock—in other words to have drilled from four to eight or more holes. Much time, however, is necessarily consumed in changing the "jumpers" and in shifting and adjusting the machine to fresh places.

During all drilling operations, whether conducted by hand or machine, the Mining Regulations require that no dust shall be produced, in other words that the pulverised particles of rock shall not escape into the air. This end is secured, as far as the rock-drills are concerned, by keeping the interior and the surroundings of the hole wet with water, a procedure which converts the dangerous dust, as soon as it is formed, into mud. The penalty, upon conviction, for neglect of such precautions, or for breach of any other of the Mining Regulations, may be a fine not exceeding £150, or, in default, twelve months' imprisonment. Into all holes which are inclined downwards water is poured from time to time from a small can; into those which are horizontal or inclined upwards a jet of water under pressure is directed by hand every now and then. The efficient irrigation of these upwardly inclined holes (or "top" holes, as they are termed) is sometimes a troublesome matter, and it is in the drilling of such holes that the accompaniment of an automatic irrigating appliance is specially desirable. Certain

varieties of rock-drills, such as the Leyner, provide for the automatic washing out of the cuttings from the hole by a stream of water, thus keeping the hole wet. In these machines the drill is hollow, the central channel serving to conduct a constant supply of water into the depths of the hole.

By whatever means and in whatever direction the hole be drilled, if water be employed intelligently and in sufficient quantities, all the dust created either collects around the hole or streams down the face of the rock in the form of mud. Should such mud became quite dry its disturbance would, of course, liberate the siliceous dust into the air; such an event is prevented, however, by keeping all parts of the mine where work is being carried on continually damp.

From ignorance of the necessity for associating its working with the systematic use of water, or because of wilful neglect of the precaution, the common type of mechanically-operated rock-drill has acquired an evil reputation which is not confined to the Rand. An increase in the industrial mortality on the Cornish tin mines has been attributed to the introduction of the rock-drill, and the same statement has been repeatedly made with respect to its intensive use in the gold mines of the Transvaal. Our Miners' Phthisis Commission of 1912 found that the occupation of machine-drilling, of all occupations in the mine, was the most important in producing miners' phthisis. It is certain that if a rock-drill be operated in a dry, or inefficiently wetted, hole it becomes a formidable weapon for the destruction of human life; but it is equally certain that the rock-drill is no more harmful than any other machine tool if a simultaneous and efficient irrigation of its work be maintained.

We have now seen that the operation of drilling the rock to receive the charge of explosive can be conducted without giving rise to dust. The Miners' Phthisis Commission of 1912 nevertheless disclosed the fact, as already stated, that machine-drilling was the most dangerous of the various mining occupations; further, it gave the third place in the list of silicosis-producing operations to hand-drilling,

the second and fourth places being allotted to "tramming" and "timbering" respectively.

With respect to the particular liability of machine and hand drillers, we must bear in mind the fact that they are specially liable to inhale the dust which has been created, not by their drills, but by the blasting of the holes which were drilled by the preceding shift; their special liability is, therefore, to be charged, not entirely to the particular character of their work, but also, and perhaps in a greater degree, to the attendant conditions.

Although the rock may be drilled without the creation of dust yet the production of dust in large quantities by the operation of blasting is inevitable. In the immediate neighbourhood of the charge the rock is crushed to powder by the explosion, and this powder may be projected to a great distance from the working face. The finer particles of this dust remain suspended in the air of the working for several hours. The amount of dust thrown out by the blast may be reduced somewhat by previously wetting the floor, roof, and walls of the drive or stope, and by directing large and powerful water-sprays against the face in which the explosion is to occur—the sprays, or "water-blasts" as they are termed, being set in action just before the charges are fired. Such procedures are rendered compulsory by the Regulations. The amount of dust which floats in the air after the explosion may also be reduced by the use of automatic sprays and atomisers which have been fixed in position previously. It has also been recently suggested, I believe, that the precipitation of the suspended particles may be hastened by filling the working with steam. The passage of the dust-laden air to other parts of the mine may be hindered by the use of "water-screens," or walls of falling spray, by the closing of doors, and by diverting the current of ventilation. Even when all such devices and methods are employed a fine, siliceous dust floats in the air of the workings for some time after the blast, and tends to become diffused into the air of adjacent workings if the direction of the air currents permit. This suspended dust is so fine that it passes through practically every kind of respirator hitherto devised, and the only way

in which the miners may be prevented from inhaling it is to forbid their return to the particular section of the mine until it has all settled. For this end, and where ventilation of the working is being maintained, an arbitrary period of eight hours is probably sufficient; the Mining Regulations, however, only restrict the period of compulsory absence to half an hour, but add "and then only if the air is free from dust, smoke and fumes perceptible by sight, smell or other senses." Recent tests have, indeed, shown that by the assistance of efficient water-blasts the dust thrown into the air by the explosions may be reduced as low as 10 mg. per cubic metre within half an hour of firing the shots.

Many holes are usually drilled in the working face and blasted simultaneously. The face of a drive, raise, or winze is generally covered by a sort of rough pattern of holes, and of these those in the central part—which is technically known as the "cut"—are first blasted, and subsequently those in the peripheral parts—which technically constitute the "round." The reason why the central part, or "cut," needs to be blown out first is in order to provide a hollow into which the surrounding parts of the rock, or "round," may be broken by the second series of explosions. Miners are usually paid according to the number of feet which they have driven, sunk, or raised in development faces, and for this reason they are under the temptation, having blasted the "cut," to return and blast the "round" before the dust of the first explosion has subsided. This practice has, in the past, been a prolific source of pulmonary silicosis.

The workers who shovel the broken rock into the trucks are known as "lashers"—a word which, as I am informed, is probably a Kaffir corruption of a Dutch word derived from "laaien," to load—and those who push these trucks along the tram-tracks to the shaft are the "trammers." The occupations of "lashing" and "tramming" are the second most dangerous in the mine owing to the fine dust which may arise from the dry surfaces and crevices of the broken rock when this is shovelled, trammed, and tipped into the ore-bins and skips. The thorough wetting of the rock, both before and during its

shovelling, reduces the danger to the minimum, and, if quite efficiently performed, entirely removes it.

When the gold-bearing rock is hauled to the surface it is tipped on to "grizzly bars" in order to separate the smaller from the larger pieces. Those lumps of rock which will not pass between the bars are transferred to crushing machines, which reduce them to a diameter of about 1½ inches. The operation of crushing, unless carried out with certain precautions, is liable to generate dangerous dust, and many cases of silicosis have, in the past, originated amongst workers in the crusher house who have never been employed underground. By the provision of automatic sprays and of suitably-placed, hooded, exhaust-fans the danger of this particular occupation may, however, be eliminated.

The stamping of the ore to powder in the stamp mills and its grinding in the tube mills, as well as all subsequent processes to which it is subjected, are accompanied by the use of such an excess of water that no danger from dust exists.

It is well to remember that the excavation of rock underground is carried on for either one of two immediate purposes, the first—termed "development"—consists in the sinking of shafts and the driving of tunnels, raises, and winzes to give access to the deposits of auriferous conglomerate; the second—termed "stoping"—comprises all the operations necessary to excavate the ore which has been reached by previous "development." It is generally recognised that pulmonary silicosis is more often contracted and more rapidly progressive amongst miners engaged in "developing" than amongst those who do "stoping" work only. The reason for this is that as the operations of development are not immediately remunerative they are pushed forward with all possible speed; this haste is liable to be accompanied by neglect of precautions, and has more especially encouraged, in the past, an untimely return to the working face after blasting. In addition to this, there is the fact that rock-drills are almost exclusively used in development, whilst a large part of stoping work is done by hand.

The fact has already been mentioned that whatever be the cause of an interstitial pneumonia the

condition is liable to give rise to abnormal accumulations of extraneous carbonaceous matter in the lungs. For this reason it seems probable that particles of mineral dust will also be more readily incarcerated in the lungs of miners who are already suffering from chronic pulmonary disorders. This suggestion supports a somewhat discredited hypothesis that those who have pulmonary tuberculosis, should they have the misfortune to work underground, develop silicosis more rapidly than others. It enables us also to understand how the chronic bronchitis which arises from frequently breathing air containing nitric fumes—such as is often found in ill-ventilated workings after blasting—may aiso be accessory to a more rapid development of miners' phthisis.

The cause of silicosis on the Rand is, then, the inhalation of the fine dust which may arise during operations underground and in the crusher house. The liberation of this dust into the air can always be prevented, though sometimes at inconvenience to the worker; and when it cannot be prevented, as in blasting, the air in which the dust is temporarily suspended need not be inhaled. We see, therefore, that pulmonary silicosis is a disease which should be classed as "preventible," and that it is preventible by means which are practicable and at a cost which is not prohibitive.

A standing committee, termed "The Miners' Phthisis Prevention Committee," has been engaged for some time in the systematic investigation of methods and appliances for protecting workers in the gold mines from the inhaling of dust. Its investigations have already resulted in the framing of several new regulations, and these have now received official sanction. There is still much research to be carried out in perfecting these methods and appliances and in defining the limits of their most useful application. Even more important than these refinements, however, is the need for the systematic education of all grades of underground workers in the rationale of dust-prevention and the necessity for their honourable co-operation to secure the desired end. Not only must a sense of responsibility for ones own health be awakened, but a spirit of

sane socialism is also to be encouraged. The wilful neglect or careless application of dust-preventing precautions, and the voluntary return to work in a dust-laden atmosphere after blasting, are not only offences against ones fellows, but are also suicidal follies. The miner dreads being "gassed" by the fumes of burnt dynamite in an unventilated working, and he fears this because the fatal effects are often dramatically rapid. He too often forgets that more of his comrades are killed by siliceous dust than by "gassing," and he fails to appreciate the fact that disablement and gradual death from miners' phthisis is by far the more appalling end to his career.

It is no mere euphemism to say that a scrupulous obedience to *the spirit* of the present Mining Regulations will result in the total abolition of pulmonary silicosis from the gold fields of the Rand.

Symptoms and Physical Signs.

Miners' phthisis is generally believed to be a slowly-developing, chronic disease which usually extends over a period of several years. A few cases amongst native labourers have, however, come to my notice which appeared to be instances of "acute pulmonary silicosis." In such cases the patient dies after a short illness, the symptoms and signs of which are suggestive of an acute broncho-pneumonia. The lungs are found, post-mortem, to be deeply congested, oedematous, and finely mottled with islands of pigmentation, but exhibit none other of the characteristic appearance of fibrosis. 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. Such cases are undoubtedly rare and are probably due to the inhalation of very large quantities of dust over a short period.

In the usual form of the disease the symptoms are very gradually developed, so gradually, indeed, that for the first few months they do not excite suspicion. In this early stage the most prominent

symptom is that of a dry cough, occurring more particularly upon coming to the surface after work or on waking after sleep. To this succeed an increased liability to "bronchial colds," slight dyspnoea on exertion, transient pleuritic pains, and a definite impairment of the capacity to work. Despite the fact that the general health appears to be good the continuance of the symptoms just mentioned was considered by the Commission of 1912 to indicate that the disease had now been definitely established. At this and in the subsequent stages the existence of areas of impaired resonance to percussion may be detected, and auscultation reveals areas of diminution or abolition of the vesicular murmur; tubular breathing, bronchophony, and crepitations are not, however, characteristically present. In the advanced stage dyspnoea becomes very marked, the cough is troublesome, and there is chronic cyanosis; expectoration, however, remains absent, or scanty, unless a tuberculous complication has supervened or non-tuberculous cavitation of the affected lungs has occurred. A characteristic symptom of the advanced stage is the so-called "asthmatic attack" during which the patient makes convulsive but ineffectual efforts to draw air into his chest. Death usually results from cardiac failure due to the increasing dilatation of the heart.

A physical sign which is almost pathognomonic of the disease is impaired mobility of the chest wall, with increasing tendency for it to become fixed in the position of expiration. This is gradually developed from the earliest stage of the disease, until, towards the end, the thorax appears to have become incapable of expansion; the most violent respiratory effort cannot now affect the shape of the chest. 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 its 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.

Much doubt exists in the minds of some as to the value of radiography in the identification of pulmonary silicosis in its various stages. A considerable amount of experimental investigation doubtless remains to be done in this direction, and it may be safely asserted that great experience in the examination of silicotic patients is necessary before an opinion of any value can be formed. Dr. A. H. Watt, of the Simmer and Jack Proprietary Mines, Ltd., whose practical experience of the subject has extended over several years, finds that no results of value can be obtained unless the thorax be quite motionless; in other words, unless the exposure be a very short one. He considers that the following are the cardinal radiological signs of the early and the advanced stages of the disease respectively :—

Early Stage.—The excursions of the ribs and diaphram are generally restricted. The shadows of the bronchial branches are more extensive, thicker, and denser than the normal. An exaggeration of the normal, fine reticulation can be seen between the thicker bronchial shadows, and small round shadows are seen, which may be due to end-view of thickened vassels and bronchi. The condition is generalised and bilateral, but may be more pronounced in one lung or one part of a lung.

Later Stage.—The shadow of the whole lung appears densely mottled. In many moderately advanced and late cases a transverse, linear shadow is present on the right side; this is probably due to thickening of the interlobar pleura between the upper and middle lobes. Tuberculous complication is often indicated by the presence of one or more irregular dark patches.

In the report of the 1912 Commission it is remarked that whilst a positive diagnosis in early cases is very difficult, the characteristic complex of signs and symptoms which arises later presents no difficulty to the medical examiner experienced in the disease. With such statements all would agree, but the opinion of the Commission that the post-mortem signs of early silicosis (in its usual form) are frequently equivocal is one from which I should differ. My reasons for so doing I shall state presently.

The question of how long a man must be engaged in underground work before he may be expected to develop silicosis has been answered by the observation that a case of the disease is hardly ever recognised until after two years' employment. With regard to the duration of life in those affected the Commission of 1912 was of the opinion that this depended very largely upon the occurrence of tuberculosis, the stage of the silicosis in which this complication appeared, and the severity of the complication. 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.

Gross Morbid Anatomy.

Upon opening the thorax of a person who has died with advanced silicosis the first thing to attract attention is the fact that the lungs do not collapse. This phenomenon is mainly due to the fact that the lungs have lost their elasticity but partly also because the visceral and parietal pleuræ are intimately adherent over areas of varying extent. The regions where the pleural sac has been thus obliterated correspond to those parts of the lung in which the disease is most advanced. What remains of the pleural sac does not, as a rule, contain any excess of fluid. The adhesions between the pleural surfaces are tough, and if the organs are to be removed in an unmutilated condition very patient manipulation is required. So unyielding are these adhesions that it often happens that portions of the parietal pleura are found attached to the lung after removal, and when the left lung is affected the adherent pleura will often carry some of the pericardium with it.

The lungs themselves are unusually tough, excepting in those parts where, as a consequence of necrosis, softening and cavitation have occurred. When the disease is well-advanced the organ is abnormally heavy, scaling nearly three times the accepted mean weight. The surface of the fibrosed lung, in those parts where the pleuræ have been adherent, appears dull, opaque, and rough. It is partially coated with irregular sheets of tough membranes or

with strips and tags of connective tissue, and is often marked by multiple leashes of injected capillaries, and mottled by petechiæ and the remains of interstitial hæmorrhages. In those regions where the pleuræ have not adhered the surface of the organ is, save for occasional patches of injected capillaries, of a pearly grey colour, smooth, and of almost cartilaginous consistence. The dark blotches and delicate pigmented reticulation which mark the surface of the normal lung are entirely obscured if the pleura be much thickened; when, as is often the case, a portion of the organ appears but slightly affected with the disease the pleura appears practically normal, excepting that the subpleural deposit of pigment is particularly evident and is seen to be of exaggerated dimensions. The pleural thickening and adhesion which are characteristic of the later stages of the process are found to be just as advanced, and sometimes even more so, between the lobes of the lung as on its surface; a common result of this is that the sulci between the lobes are filled in and obliterated, and their original position may consequently be determined only with the greatest difficulty.

A typically fibrosed lung is unusually solid and inelastic to the feel, and when removed from the body behaves like a frozen organ in that it tends to retain its shape when laid upon a flat surface. It is neither collapsible by pressure nor distensible by inflation from the bronchus.

When the organ is divided through its whole length and the cut surface inspected it will be found, as a rule, that two or more of the characteristic stages of fibrosis, and sometimes all of them, can be demonstrated in different regions of the lung. In one region of the cut surface the appearances of advanced silicosis will be present, whilst another will very often appear to be almost unaffected, excepting that it will show evidences of increased deposit of pigment, and congestion with œdema. Beyond the fact that the whole or most of one lobe is usually in the same condition the distribution of the various stages of the disease throughout the lung is not constant. The region showing the most advanced stage may be at the

base, in the middle, or at the apex of the organ; the anterior margin, and the anterior and lateral parts of the margin of the base are, however, usually less affected than all other regions.

In consideration of the cause of the disease it might be anticipated that both lungs would be affected to an approximately equal degree; this, however, is not an invariable rule, and one sometimes finds one lung showing extensive and advanced silicotic changes whilst the other is but slightly affected.

From text-book descriptions one is led to anticipate that when the silicotic lung is cut into the knife will be felt to grate against the deposits of mineral matter. I have not experienced this in gold-miner's silicosis, the most that I have observed is that the edge of the microtone razor sometimes suffers during the subsequent preparation of sections.

As judged by the unaided eye there appear to be four well-defined stages in pulmonary silicosis. The first two of these stages can be identified by the arrangement of the extraneous pigment, and the last two by the arrangement of the visible deposits of unpigmented connective tissue—the arrangement in both cases being that which is disclosed upon the cut surface of the organ.

In the first stage we find an œdematous and congested lung tissue of bright red colour—or more correctly, one which becomes bright red a few minutes after the section has been made. The connective tissue partitions which normally separate the groups of lobules are unusually conspicuous. Throughout the congested lung tissue are distributed numerous islands of black pigmentation of varying size and extremely irregular outline and in the centre of many of which a divided blood vessel or bronchiole is present. This appearance of scattered deposits of pigment is merely an exaggeration of that usually found in the lungs of town-dwellers and others who inhale smoke; it differs from it, however, in the closer aggregation of the pigmented islands, in their darker colour and firm consistence, and in the fact that many of them tend to bulge from the general level of the cut surface. The cut surface appears, therefore, to be roughly mammillated, and shows a

red background discreetly mottled with black. The tissue is crepitant and floats in water. The appearances of this, the earliest stage of pulmonary silicosis, are sufficiently striking and characteristic to render the identification of the condition an easy matter.

In the second stage the projecting islands of pigmentation have increased in size, and by coalescence of some of their adjacent margins have formed a dark and very coarse reticulum which encloses small islands of deeply-congested lung tissue in its meshes. In some instances, however, the joining up of the islands to form a reticulum is not well marked; in these circumstances the pigmented islands, though remaining discrete, become so numerous or so large that they convert the ground work of the tissue from red to a dark grey. The cut surface is now coarsely granular and shows a black, or dark grey, background discretely and finely mottled with red. The tissue is still slightly crepitant and floats in water.

In the third stage the little islands of lung tissue have become almost entirely obliterated by the encroachment of the pigment-bearing connective tissue of the reticulum. The pigmentation of the more or less uniformly pigmented background is often, however, of a less intense character than that of the first and second stages. Clusters of small, discrete, circular, oval, and polygonal areas of light grey colour are now usually developed in this pigmented background—an appearance which may be very easily mistaken for tuberculous deposit. These clusters of light grey areas increase in number and size, so that the cut surface of the lobe may be eventually found to be uniformly studded with them.

These discrete, light grey areas of new formation are very characteristic of the third stage of silicosis and constitute the so-called "silicotic nodules," a term which, as we shall see, is not always appropriate. Dr. L. G. Irvine considers that one of the most striking characters of these "nodules," when they are few in number and widely separated from one another, is the firmness of their consistence: this character enables their presence to be recognised by palpation even when they are buried deeply in

the substance of the lobe. The frequency with which one sees reference to these "silicotic nodules," and their importance in elucidating the pathology of the disease justifies some special consideration of their nature.

By careful examination it can be seen that each of these grey areas corresponds in position to one of the original islands of black pigmentation, and also to a node in the pigmented reticulum of the second stage. Their size varies greatly, from a mere speck up to a diameter of 10 mm. or more, and when fully developed each area comprises the limits of a lobule of the lung. Larger areas are evidently formed by coalescence of fully developed smaller areas. Close scrutiny shows that although the general appearance is that of a light grey area, this area is, nevertheless, surrounded and coarsely partitioned by a framework of translucent, light grey, connective tissue. This framework is arranged in a very simple basket-work pattern (reminding one of a cane carpet-beater) and encloses deposits of pigmented material in its few meshes. At one or more places within the boundary of the area, and either peripheral or central, a minute, white ring or disc can often be detected: these mark the sites of obliterated vessels or bronchioles. One or more of the bars of the framework which partitions the area is often much whiter than others, and thus gives the appearance of a tag of cotton thread embedded in the grey area. The term "silicotic nodules" can be correctly applied to these areas only in their early stage, and when but few of them have developed in the affected organ; when they are numerous and closely aggregated cautious dissection will show that each area is merely the cross section of a short and crooked branch, and this branch will be found to spring from another short and crooked branch in its immediate neighbourhood. The appearance of many discrete "nodules" in the cut surface of a silicotic lung is therefore due to the cutting across of the numerous branches of a ramifying fibrosis—a fibrosis which accompanies and surrounds the vessels and bronchi of the lung in their ultimate distributions. Brief consideration of this fact shows that we must not expect a collection of these so-called

"silicotic nodules" in the lung to project discrete shadows on the X-ray plate—it would be as reasonable to suppose that the pores of a radiographed sponge would show themselves as discrete discs of light.

The cut surface of the lung in the third stage presents, then, a pigmented background which is studded over to a varying extent with discrete, light grey areas. The tissue will float or sink in water according to the degree to which grey areas have been developed in it.

The fourth stage is typically indicated by an increase and enlargement of the grey, fibrotic areas to such an extent that the pigmented matrix in which they have developed is entirely obliterated. As the grey areas increase in size they come into contact with one another and distort their shapes by mutual pressure. They then fuse more or less intimately with one another, and thus give rise to a dense, close-grained and airless tissue. This tissue has not the slightest resemblance in texture, consistence, or colour to the lung tissue in which it has developed. Minute inspection of the cut surface soon after the fusion of the grey areas shows the new tissue to be arranged in a delicately vermicular pattern of striæ, volutes, circles, and triangles, scattered amongst which are small pigmentary deposits. This delicate pattern is soon obliterated and the appearance which is most typical of the cut surface in this, the fourth, stage becomes strikingly similar to grey granite—a grey background which is stippled with black and traces of brown. The tissue always sinks in water.

The fourth stage is sometimes reached without the previous appearance of the "silicotic nodules," or conspicuous grey areas, of the latter part of the third stage. This event is exceptional, and when it occurs the uniformly pigmented tissue of the first part of the third stage becomes infiltrated by a reticulum of grey connective tissue spreading inwards from the interlobular partitions and from the vessels and bronchioles. The cut surface of such a specimen is almost homogeneous and resembles black, coarse-grained shale.

The four stages in the silicotic process, the appearances of which have now been very briefly described, may be conveniently referred to as those of discrete, and reticulated pigmentation, and discrete, and diffuse fibrosis respectively. Examples of each of these stages, and of transitions between them, are usually found in the same lung, sometimes even in the same lobe. It is often apparent, however, that either the second or third stages have been omitted; thus a few, well developed, discrete, grey areas are sometimes found in lung tissue which is only mottled, and not reticulated, with pigment, and we have already remarked that the fourth stage may ensue directly upon the second. These variations by omission do not appear to me to be of importance, and the four typical appearances which I have described seem to represent the usual steps in the evolution of the disease-process.

Were those who develop pulmonary silicosis to inhale silica-laden air free from pigmented mineral matter and the smoke from candles, acetylene lamps, and tobacco-pipes we should be largely deprived of this simple method of identifying the first two stages in the development of the disease. Carbonaceous and coloured mineral fragments, owing to their visibility, afford a useful indication of the position of other foreign particles which are ordinarily invisible; and the changes of pulmonary silicosis are due especially to the presence of translucent and colourless particles of quartz.

When pigmented particles are not greatly in excess of the siliceous fragments in the inhaled air the appearances in the lungs are modified, although the different stages in the morbid process are essentially the same. In such circumstances the stages are identified by the distribution of grey connective tissue accompanied by but little pigmented matter. The tissue, as seen on the cut surface, appears to have been laid down, at first in discrete islands, then as a reticulum with large nodes, and finally in the form of a diffuse fibrosis.

Before leaving the question of the association of pigmentary deposits with the fibrosis of silicosis let me recall a very noteworthy fact.

Any fibrotic, interstitial pneumonia, *whatever be its cause*, is liable to be associated with abnormally large deposits of pigmented matter in and around the new connective tissue. This is well seen in some cases of chronic, tuberculous phthisis, and is doubtless due to the extensive obliteration of the connective tissue spaces and lymph channels by which inhaled particles of foreign matter normally traverse the lung. The deeply pigmented lungs of many of those who die of silicosis should not therefore lead us to conclude that they have necessarily inhaled abnormal quantities of carbonaceous material, but rather that such material, having gained access to the lungs as usual, has become incarcerated there owing to obstruction of the normal passages of exit.

When once the fourth stage has been reached in any region of the lung another and a highly characteristic change becomes possible, or perhaps inevitable, namely, a localised necrosis of the fibrous tissue owing to deficiency of blood supply. This necrosis manifests itself first by a softening and then a liquefaction of the more central parts of a diffusely fibrosed region. When, as sometimes happens, the affected lung has escaped a destructive tuberculous infection the liquefied products of this necrosis often remain *in situ*, and thus no actual cavity may be formed during life. With the common complication of a tuberculous infection it is, however, the rule to find one or more empty cavities in the fibrosed lung. The emptying of such cavities during life is evidenced by the expectoration of a large quantity of dark grey sputum.

When the necrotic changes have occurred in any portion of the lung near its surface it becomes almost impossible to remove the organ without mutilation, owing to its adhesion to the parieties and the friability of the necrotic tissue. The vomica which results from the necrotic liquefaction of the dense fibro-cellular tissue of the fourth stage has, in the absence of a tuberculous infection, viscid, dark grey contents, very ragged walls, no definite boundaries, and usually extends into its surroundings by ragged clefts and fissures. The presence of

a defined wall of more condensed tissue, or of denuded blood vessels in the vomica, are both indications of the presence of a tuberculous process.

It is a very common experience to examine a lung which is neither typically tuberculous nor typically silicotic. In such specimens scattered areas of caseation, cheesy peribronchial deposits, and cavities with well-defined limiting membrane are found in pulmonary tissues which show at the same time the pigmentary characters of the early stages of silicosis or the silicotic "nodules" of the third stage. As already remarked, a chronic tuberculous process in the lung is liable to induce both abnormal accumulations of pigment and also fibrosis. The presence of tuberculous foci in an early silicotic lung is therefore usually accompanied by a localised hastening of the stages of silicosis. Thus we commonly find that when a single lobe is not uniformly in one stage of silicosis it is also affected by tubercle. In such circumstances it is usual to find samples of all the stages of silicosis, often accompanied by excavation, within the limits of a single lobe.

I have attempted to formulate a description of the appearances in the various stages of pulmonary silicosis because it has hitherto seemed almost impossible to record them briefly and accurately. This difficulty has been largely due to the fact that the disease is usually present in what are obviously different stages in various parts of the same lung. Were any other apology needed for a minute description of the macroscopic appearances of the various stages of the disease I would urge that the mistake is very commonly made of describing a miner's lung as "tuberculous" because it has circular, translucent, greyish white deposits in it, or because there is a ragged excavation in its more densely fibrosed parts. Without a full appreciation of details and the exercise of much patience such a mistake is almost inevitable. It is, indeed, advisable that a tuberculous infection of a silicotic lung should never be alleged unless the B. tuberculosis has been detected microscopically.

It is usually stated that the bronchial lymph nodes are markedly enlarged in cases of pulmonary silicosis, but my own experience leads me to think that

this is not a correct statement. Although an enlargement up to 15 or 20 mm. is common, yet I have rarely found them to exceed 30 mm. in longest diameter. They are usually very firmly embedded in the fibrosed lung, or in the indurated tissue of the mediastinum. On section they are found to be tough and to exhibit a dull jet-like pigmentation which is usually mottled with small areas of grey fibrous tissue. Even when tuberculosis is a prominent complication of the silicosis it is rare to find these lymph nodes showing any caseation.

The mucous membrane of the trachea and bronchi is nearly always thickened, œdematous, and congested to a dull Indian red colour. Small, necrotic ulcers of the tracheal or laryngeal mucous membranes are commonly found. The heart is almost always flabby and markedly dilated.

A few years ago Calmette published some observations on experimental pneumonokoniosis which were held to support the theory that pulmonary tuberculosis was usually due to the ingestion of tubercle bacilli rather than to their inhalation, and he showed that insoluble particles which enter the body by way of the stomach and intestine may be eventually found arrested in the lungs. As a sequel to these experiments some writers have maintained that pulmonary silicosis results from the swallowing rather than from the inspiring of siliceous dust, but many researches have since been published which disprove this view. My own observations go to show 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. Slightly enlarged and pigmented nodes are sometimes discovered in the gastro-hepatic ometum and are found to contain traces of pigmented mineral matter; I have not succeeded, however, in detecting the presence of siliceous particles in them.

Before passing on to describe the microscopic anatomy of the disease I wish to acknowledge the debt I owe to Drs. L. G. Irvine and G. A. Turner for their generous assistance in providing abundance of material for investigation together with carefully compiled clinical notes.

Histopathology.

In the foregoing section I have indicated how the medical man (knowing the occupation of the deceased) may recognise, by the unaided eye, the various stages of silicosis and even decide on the presence or absence of a tuberculous process. The examination of microscopic sections will, however, confirm his opinions, and will, moreover, reveal to him some very interesting specimens in histopathology.

The histological abnormalities in the lungs are essentially those of chronic, interstitial pneumonia associated with the presence of minute particles of siliceous mineral matter. The presence of these foreign particles gives rise to a proliferation of the connective-tissue. This proliferation is at first protective and moderate in character, serving apparently to incarcerate the foreign matter in the lymphatic channels and connective-tissue spaces of the organ; eventually, however, it becomes exuberant, and, quitting the immediate neighbourhood of the foreign particles it invades and replaces all the normal structures of the lung in a manner somewhat similar to that of a malignant tissue. It is also analogous to malignant new-growth in that it supplies itself very inefficiently with blood vessels, with the consequence that when it has reached its maximum development it inevitably undergoes necrosis.

In the first stage of silicosis—that in which a congested lung tissue is discretely mottled with pigmentary deposits—microscopic examination shows that the external, fibrous coat of the terminal lobular blood vessels, and to a less extent of the terminal bronchi, has undergone a fibro-cellular proliferation. In the interstices of the thickened adventitia carbonaceous and siliceous particles are found in great abundance, some of the particles being contained in phagocytic cells but most of them being free. The reason why the foreign matter is first noticeable in this situation is because it corresponds with the position of the lymphatic channels of the lung; these accompany the vessels—more particularly the veins—and are bound up with their outer coats. The

connective-tissue proliferation is also seen to affect the interlobular septa and the walls of the immediately adjacent air vesicles. The alveolar walls are thickened, partly by distention of the capillaries with blood and partly by the appearance around the capillaries of a nucleated fibroid tissue. The alveoli and terminal bronchioles are often flooded with serous fluid and usually contain a few catarrhal cells and leucocytes, amongst which siliceous fragments are often detectable. Similar fragments may also be seen in the interior of both varieties of cell, and the leucocytes are usually laden, in addition, with minute particles of other mineral and carbonaceous matter.

In the second stage, the perivascular and peribronchial fibrosis is more extensive and the lumina of the tubes are much diminished. The nucleated fibroid tissue in the alveolar walls has increased in amount and phagocyted and free mineral particles are now to be found in its interstices. The air-space of the alveolus is greatly diminished owing to the thickening of the septa and the increasing accumulation of cellular elements and cellular debris in the cavity. These intra-alveolar cells now form a compact mass which shows a definite tendency to become organised.

In the early part of the third stage many of the alveoli are found to have become entirely obliterated by the sprouting into them of the new fibro-cellular tissue in their walls and its coalescence with the organised catarrhal contents. The so-called "nodules" of the later part of the third stage are found to be merely extensions from the perivascular proliferation noticed in the first stage, a proliferation which now extends around the whole lobule and separates and permeates its component lobulets. This perilobular proliferation is seen to be relatively free from those deposits of extraneous mineral matter with which, whilst merely perivascular, it was associated. The vessels, terminal bronchi, and terminal bronchioles, in many of these "nodules" are now found to have become obliterated, the sites of their lumina forming the centres for somewhat concentrically arranged laminæ of fibres. A premature

obliteration of the bronchioles may lead to the collapse of any alveoli which may have remained functional and thus facilitates their invasion by the new tissue. A premature obliteration of the vessels probably explains the occasional presence of groups of alveoli which, with their catarrhal contents, have undergone necrosis, as indicated by their inability to become stained by the ordinary histological dyes.

In the fourth stage of the disease we find that the interstitial pneumonia has been carried to its farthest possible limits; the normal alveolar structure of the lung has practically disappeared, all the characteristic tissues, with the exception of the larger branches of the blood-vessels and bronchi, having been replaced by a fibro-cellular growth of low vitality. In the meshes of this tissue a few degenerating leucocytes may be found, often represented by little more than nuclei. Scattered throughout it are irregularly shaped spaces and clefts—the remains of alveoli—which enclose a few dust-bearing cells and free, foreign particles; the sites of obliterated vessels and bronchioles are now mainly marked by isolated accumulations of carbonaceous and mineral fragments.

Thus the various stages of pulmonary silicosis are found to be essentially characterised by different degrees of fibrosis—perivascular, peribronchial, perilobular, and diffuse.

The diffuse, composite, fibrotic tissue of the fourth stage has a marked tendency to undergo necrosis and disintegration, a change which, spreading from the sites of the obliterated vessels, gives rise to areas of amorphous material throughout which are scattered nuclear remains, particles of extraneous pigment, and mineral fragments. The leading microscopic character of this material is the absence from it of pus cells, or indeed of any formed elements; if pus cells be present in abundance, or if the necrotic change be found in the lung tissue in the earlier stages of silicosis, the process is almost certainly a tuberculous one. In the majority of instances (probably about 90 per cent. at the present time) in which the fibrotic tissue of the fourth stage is found to have undergone necrosis the B. tuberculosis will be detected in the structureless material; the

organism is seen, in scanty numbers, lying among the mineral particles and detritus of the disintegrated tissue. Beyond the presence of the bacillus, there are usually no histological signs of a tuberculous process; giant-cell systems, small-celled infiltrations, and deposits of tubercle in the lymph nodes or elsewhere in the body are absent. It is therefore somewhat misleading to say that the great majority of cases of silicosis is complicated in the last stage by "tuberculosis." Although the identity of the organism has been frequently proved by animal inoculation, yet the rôle which it plays in the final stage of pulmonary silicosis would be more correctly described as that of a saprophyte than a pathogenic parasite. The silicotic lung in all its stages is more liable to develop a tuberculous infection than a lung in which there are no fibroid changes. This is especially seen in the lungs of native mine labourers, in which it is very common to find the histological characters of pulmonary tuberculosis in association with those of both the early and the middle stages of silicosis. In these cases tuberculous deposits are usually found in other organs besides the lungs.

It is stated by certain authors that the bronchial lymph nodes in cases of silicosis are not affected by fibrosis. With this I cannot agree, for I have found that in all cases in which the disease has advanced to the third stage in one or more lobes of either lung the lymph nodes, though not greatly enlarged, show changes which are strikingly similar to those in the lung. There is a more or less complete replacement of the proper elements of the lymph gland by a newly-formed fibro-cellular tissue, in the clefts of which minute carbonaceous and siliceous particles are located. The siliceous particles appear to be smaller and more uniform in size than those seen in the lung tissues. It is also worthy of remark that the deposits of silica in the lymph nodes are usually very scanty, even in cases in which the lungs are laden with this material: this fact is doubtless the consequence of the early obliteration of the afferent lymph channels in the lungs. Although microscopic evidences of an early stage of necrosis in the new fibro-cellular tissue of the gland

are usually present, complete disintegration and liquefaction do not apparently occur.

In order to detect the primary cause of miners' phthisis in microscopic sections it is essential to employ a polariscope in conjunction with the microscope a word of caution is necessary as to the ex- by ordinary illumination. Silica, and in varying degrees the mineral silicates, rotate the beam of polarised light ; when therefore a section of a silicotic lung is viewed through crossed Nicol prisms the siliceous particles stand out as apparently luminous objects on an obscure background. By such means we are able to discover the anatomical distribution of the particles, their shapes, and—if we also employ a micrometer eye-piece—their dimensions.

To those unaccustomed to the use of the polariscope a word of caution is necessary as to the examination of sections. Fine particles of quartz are very common constituents of dust, and may have fallen on the section during its preparation or on the cover-glass after the specimen has been mounted. To avoid the latter source of error the coverglass must be very carefully dusted before the object is examined. Fragments of cotton fibre and the particles of "fluff" which separate from the surface of the filter-paper with which the section may have been dried are, like silica, optically active. Finally, if the section has been stained with certain aniline dyes, large tracts of the tissue may appear partially luminous through the crossed prisms.

The siliceous particles in a miner's lung are seen in what appears to be the greatest abundance in the earlier stages of the disease, and before the familiar features of a lung-section have been abolished by the exuberant fibro-cellular growth of the later stages. When the fibrosis has advanced to the fourth stage the particles are found to be confined to isolated areas separated by wide tracts of almost silica-free, new tissue. If the patient has survived for many months after cessation of work the amount of silica detectable in the lung by microscopic examination is much reduced from that found in more acute cases, and if the survival has extended over several years it may, apparently, entirely disappear.

The exit of the siliceous particles from the fibrosed lungs of those who survive long enough after leaving the mines appears to be accomplished by the agency of phagocytic leucocytes. These cells enclose the particles and transport them not to the lymph nodes —for the passages to these appears to remain occluded for a long time—but to any of the air sacs which retain communication with patent air tubes. The evidences in favour of this theory are—that for many months after the patient has left his work phagocytes, bearing siliceous and carbonaceous particles, are found in those alveoli which are still functioning; and that for long after cessation of work siliceous and carbonaceous particles are present in the sputum. The same clearing process is apparently carried on in the lungs of the subjects of anthracosis, for the sputum of such people contains carbonaceous particles for many months after they have ceased to inhale coal dust. The factors which, in the majority of cases, prevent this clearing process from restoring the patient to health are—the grave anatomical disorganisation of the lung tissue and the greatly enhanced liability to a tuberculous infection.

In a case in which death occurred from right-heart failure two years after cessation of work, I found that microscopic sections of the less affected parts of the lung showed but little interstitial deposit of silica; a few particles of the mineral could, however, be detected in the alveoli. In the fibrosed lungs of a middle-aged man who died from adherent pericardium and who had been a miner "all his life," but had been entirely incapacitated from work by "silicosis" for a period of four years, I could detect no siliceous fragments at all in the lungs.

The characteristic shape of the fragments of silica which are found in sections of lungs has already been referred to. Dr. A. I. Girdwood has been patient enough to make sketches and measurements of one hundred consecutive fragments as viewed by polarised light in lung sections, and I have reproduced his sketches to scale. The two largest of these particles were not actually in the lung tissue but in the cavities of bronchioles or alveoli. It will be seen that the majority of the fragments vary

from about 2μ to 12μ in maximum diameter. It must be remembered, however, that particles having a diameter below about 1μ are incapable of rotating the beam and therefore remain invisible by polarised light. That such very minute particles are, however, in the great majority in the silicotic lung has been found by Dr. J. McCrae. His observations in this matter will, I hope, be published, but I have his permission to show this sketch of the microscopic characters of the silica and refractory silicates separated from a silicotic lung by acid-digestion of the organic matter. In order that the shape of the larger particles might not be obscured, Dr. McCrae has intentionally omitted an enormous number of minute siliceous specks, all of which were less than 1u in diameter.

The appearance of a section of a silicotic lung, in the early stage, is of striking interest when viewed by polarised light, and one of the first questions which rises in the mind of the observer is—how did the particles of silica reach the positions in which they are seen?

Some physiologists have maintained that the air in the smaller bronchioles and pulmonary alveoli is renewed by "diffusion" only, and that the oscillating current of tidal air due to the alternating movements of inspiration and expiration does not extend much below the bifurcation of the trachea. We know that 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. Other 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. This is evidently a matter upon which there is still want of agreement, at all events with respect to the terms used. Study of

the subject which we have now in hand shows that 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, as we have seen, the particles in the lung tissue are practically all of very small dimensions. We must, for the present, therefore, suppose that some particles of inhaled dust are carried by the air current right into the air sacs, although their passage may have been opposed by the action of the ciliated epithelium which extends as far as the respiratory bronchioles.

The next consideration which presents itself is that the entrance of foreign particles into the lung tissues must be due, to some extent, to a vital process. It is difficult to believe that particles floating in the inspired air can penetrate mechanically into the tissue unless the air be under considerable pressure—which it is not. Dr. Adami suggests that the foreign matter is carried into the lung tissues by scavenging leucocytes and by the phagocytic activities of the epithelium, and this is a theory which, in view of what we have already said about phagocytic transportation, must commend itself to us. We have seen that the particles in the pulmonary alveoli and in the connective tissue spaces are very often enclosed in phagocytic cells, and that when free in the lung tissue they are very rarely, if ever, of too great a size to have been carried thither by such agency. Phagocytic cells which contain mineral fragments are, moreover, quite commonly seen, in silicotic sections, to be undergoing dissolution, depositing their burdens where they die. Particles of foreign matter are also found in abundance in the new tissue immediately beneath the respiratory epithelium; as the air sacs are not provided with any lymphatics it is obvious that such particles cannot have been carried from other parts of the lung to this situation through lymphatic channels.

Another fact which may be held to favour the theory of the absorption of the extraneous fragments being due to a vital process is that the particles in the lung appear to have been selected from the dust

which has entered the air passages. If we examine the dust generated by drilling or blasting we find that the elongated acicular forms so common in the lung are enormously outnumbered by those of other shapes—the commonest being mutilated cubes and tetrahedra.

That the foreign particles do not enter the lung tissue via the epithelium of the bronchioles is suggested by the fact that deposits of such particles around the bronchioles are neither so early nor so abundant as those around the blood-vessels.

This very brief discussion of the histology of the disease may be concluded by referring to indications of repair and restoration of function. It has been pointed out that the lungs of those who survive long enough after abstention from mining show a diminution or even complete removal of the siliceous particles. In other such cases indications of the regeneration of pulmonary alveoli can be found, and the original fibro-cellular tissue is seen to have provided itself with capillary vascular channels.

The conclusions to be drawn from this histological survey of the disease are :—

That complete recovery is possible when the disease has not advanced beyond its first or second stages, and if the occupation be abandoned for an open-air life.

That partial recovery from the disease in its more advanced stages is certainly possible, but must necessarily be associated with great and permanent impairment of function.

That recovery in either case is contingent upon the successful adoption of scrupulous precautions against infection by the Bacillus tuberculosis.

Relations Between Silicosis and Tuberculosis.

I have remarked that pulmonary tuberculosis cannot be legitimately classed with the industrial diseases; the association between it and silicosis on the Rand is nevertheless so intimate that a brief consideration of the relations between the two diseases is called for.

It has been recognised for many years that the incidence of pulmonary tuberculosis is specially high amongst those who follow dusty occupations. Of all classes of industrial workers in England and Wales tin-miners, copper-miners, scissors-grinders and file makers suffer the most from pulmonary phthisis, and agricultural labourers the least. Considering the relative rarity of post-mortem examinations in England and Wales it is but reasonable to suppose that in a large number of such instances of industrial phthisis the disease is, in reality, a pulmonary fibrosis due to pneumonokoniosis, and that the tuberculous infection is a secondary phenomenon.

The old name "miners' phthisis," which still officially prevails with us, indicates that the two diseases—pulmonary silicosis and pulmonary tuberculosis—were formerly held to be practically indistinguishable; their recognition as distinct diseases has, indeed, only come about in recent years. I have already pointed out that the apparently inevitable necrosis of the fibrotic lung-tissue in the fourth stage of silicosis is generally associated with the presence of the B. tuberculosis in the disintegrating material, but that signs of tuberculosis either elsewhere in the lung or in other parts of the body are absent. I have also stated that pulmonary tuberculosis is extremely common amongst the native mine-labourers of the Rand, and it may be further alleged that in a large proportion of these cases of pulmonary tuberculosis signs of silicosis, in its early stages, are also present. With such facts before us we are naturally led to enquire why the silicotic lung is unusually liable to a tuberculous infection, and what is the source of such an infection?

A very suggestive light is thrown upon the first question by some recent experiments by D. Casa-Bianchi (Zeitsch. f. Hyg., vol. 73, p. 166). Two series of animals were inoculated with the B. tuberculosis, and those in one of the series were caused to inhale dust for several hours daily. It was found that those infected animals which inhaled dust showed a special liability to develop pulmonary tuberculosis.

In the silicotic lung it is the most densely fibrosed and air-less parts which are most commonly affected with tubercle. This is a peculiarity which lends decided support to the theory, at least when applied to this form of pulmonary tuberculosis, that the bacillus gains access to the lung by way of the blood-stream and not by direct inhalation into the air passages. Given a source for systemic infection, we find it easy to understand how any tubercle bacilli which have been carried by the blood-stream to a lung damaged by fibrosis would be most readily arrested and permitted to proliferate in those regions in which the blood and lymph circulations were most impeded. It is probably by the free movement of lymph from capillaries to tissue-spaces, from tissue-spaces to lymph vessels, and from lymph vessels to lymph nodes that the normal lung delivers itself from any pathogenic organisms which may reach it from time to time.

The liability of silicotic lungs to tuberculous infection was found by the Miners' Phthisis Commission of 1912 to increase rapidly with the extent to which the organs had been damaged by fibrosis. Thus in the early stages 6 per cent. of the patients were found to have become infected, in the middle stages 12 per cent., and in the late stages 44 per cent. This increasing liability is, I think, due to an increasing interference with the local lymphatic circulation; this interference, however, whilst it progressively facilitates the lodgment and proliferation of Koch's bacillus, also reduces, *pari passu*, the likelihood that the infection, when once established, will become disseminated to other parts of the body.

We have now to enquire what is the source of the tuberculous infection to which silicotic miners become increasingly liable to fall victims. Whatever the source may be, it is highly probable that it is also responsible for the marked, and probably increasing,* prevalence of tuberculous diseases amongst the native mine-labourers, for White and Black are

*From a report by Dr. G. A. Turner (Octr. 1913) it appears that natives repatriated by the W.N.L.A. on account of tuberculous diseases in the years 1910, 1911, and 1912 comprised 6, 11 and 18 per thousand of the total number of natives employed in these respective years.

intimately associated in their underground work. This common source of infection appears to be continuous and prolific, and for these reasons we may at once exculpate tuberculous meat and milk, for both are very rarely found on the Rand.

In a recent Report by the County Medical Officer of Health for Shropshire (*B.M.J.*, 1912, I., 694), Dr. Wheatley refers to the marked liability of the Grinskill hard-stone quarrymen to ultimately die of "phthisis," and he points out that as there is no excess of phthisis amongst the wives of these men the element of infection is probably absent. The Miners' Phthisis Commission of 1912 was also of the opinion, stated however with caution, "that infection in the mine itself is not the chief source of infection."

I have already pointed out that the increased liability of the silicotic patient to tuberculous infection is primarily due to interference with the mechanism by which blood-borne organisms are normally removed from the lungs; the increased liability to infection does not, therefore, necessarily connote any increased exposure to infection from without. In the circumstances of underground mining, however, there are more than common facilities for coming in contact with infection-bearing material, and there is, therefore, an actually increased exposure to infection from without.

Dr. G. A. Turner has repeatedly called my attention to a very interesting form of tuberculosis which he has frequently found in mine Natives, and Dr. D. Macaulay has also sent specimens illustrating the same condition. In these cases the lesions are either entirely confined to the spleen and the mesenteric lymph nodes, or are far more numerous and advanced in these parts than elsewhere. The spleen is enlarged, sometimes to an enormous size, and is studded throughout with large, discrete, stone-coloured areas which are very sharply demarcated from the surrounding tissue by a crenated margin; the mesenteric glands are enlarged, discrete, firm and elastic. Microscopic examination of both spleen and lymph glands shows a proliferation of the proper elements, with some fibrosis, and a widespread condition of early necrosis. The tubercle bacillus can

be detected in scanty numbers and with much difficulty.* I have endeavoured to reproduce this form of tuberculosis experimentally, but have so far concluded only one observation. Three, male Vervet monkeys, reared in captivity, in good health, and housed in separate cages, received, by the mouth, one minim of human tuberculous sputum on every second day. The drop of sputum was concealed by injecting it into the centre of a banana. Within eight weeks of the first administration two of the animals had died of abdominal tuberculosis. In both cases there was a tuberculous ulceration of the small intestine and the mesenteric glands and spleen, except for obvious caseation in the glands of one animal, were strikingly similar to those seen in the abdominal tuberculosis of natives already referred to. The third, an aged animal, survived a fortnight longer and died primarily of acute phthisis. There was no ulceration of the intestine. There were, however, many tuberculous nodules in the omentum, liver, kidneys and spleen. In none of the animals was there any intracranial lesion. It may be provisionally concluded from this meagre experiment that if human tuberculous sputum be ingested in small quantities and at short intervals, a tuberculosis, mainly of the abdominal type, is originated.

The possibility of the mine natives ingesting, from time to time, small quantities of tuberculous sputum now becomes a very important consideration. Of 250 consecutive samples of expectoration collected from the underground workings of various mines on the Rand, 38, or 15 per cent., were found to contain the Bacillus tuberculosis. This sputum is deposited in all manner of places—on the walls, floors and timbering of the workings, on the steps and hand-rails of the ladderways, on the sides of the

*The report by Dr. G. A. Turner, already referred to, shows that of 309 deaths which occurred in the W.N.L.A. Compound Hospital during the six years 1908-1913, and which were found by post-mortem examination to be due to various forms of primary tuberculosis, 140 were pulmonary, 92 generalised, 33 peritoneal, 33 splenic, 7 hepatic, 3 meningeal, and 1 renal.

The same remarkable prevalence of abdominal lesions was also noted by Dr. G. D. Maynard in a paper published in the *Transvaal Medical Journal* (Octr. 1912, p. 72) and the conclusion was ventured that infection by the alimentary canal prevailed among such natives.

tubs which carry the rock, on the inner surface of the sides of the cages, and—by those who lubricate their hands in a primitive manner—on the handles of hammers and the shafts of jumpers. When working underground men are indifferent, not only as to where they expectorate, but also as to the cleanliness of any surface upon which they lay their hands. That traces of specifically infected sputum may, from time to time, be ingested by underground workers is at least probable from the fact that each man takes down with him sufficient food, usually in the form of a loaf of bread, to provide him with one meal during his shift; this meal, needless to say, he eats with unwashen hands.

The matter of the infection of underground workings by tuberculous sputum is one which is now engaging the serious attention of the Miners' Phthisis Prevention Committee. The most systematic and painstaking search for, and the rigid exclusion from the workings, of every expectorator of the tubercle bacillus—be he White or Black, manager or hammer-boy—would, in my opinion, reduce the liability to tuberculous infection of those whose lungs are damaged by silicosis to a minimum. Not only would this most desirable end be accomplished, but I venture to think that an inflexible proscription of the "carrier" from both the compounds and the workings would practically banish all forms of tuberculosis amongst the Native labourers.