hospital for German measles, the true diagnosis being reached only when the eruption did not clear up in the time usual for measles. Chancres of a terminal phalanx are usually diagnosed as whitlow until a rash appears. The worst error of this kind I ever saw was in a medical officer who was looking after syphilis cases during the war. He allowed his finger to be treated for whitlow, and his tonsils to be cauterized for ulcers, and then went peacefully to hospital for measles. It dawned on him that he had accidentally contracted syphilis, when the measles rash was succeeded by a profuse papular syphilide.

The error of excluding syphilis may be made from the absence of any history of a primary sore. A rather remarkable case of this kind was a nurse who pricked her finger in cleaning up after an operation. She dipped the finger in antiseptic, and no sore appeared, as I have known to happen after the application of an antiseptic in other cases. An axillary adenitis appeared on the side of the pricked finger, and it was diagnosed as tuberculous. Subsequently a general, rather painful adenopathy appeared, and she was treated with ultra-violet rays about three months after the accident. The application of the rays was followed by a rash, which was said to be due to an overdose, but a significant thing was a discoloration of the neck, which appeared at the same time, and was easily recognizable fifteen months later, when we first saw her, as a syphilitic leucoderma. This dated the infection. Naturally, all other sources of infection were excluded by careful examination.

A common mistake is to assume that in a patient with positive serum reactions all abnormalities are themselves syphilitic.

An unusual case was one in which a patient came to me with a history that he had been treated for secondary lesions between the toes. He had previously suffered from a gumma on the chest wall, but the peculiarity of secondary lesions succeeding tertiary did not seem to have struck his medical adviser. He had received a course of treatment, but the lesions remained unaffected, so the patient thought he was not being properly treated. A little examination showed that the trouble was due to trauma, and from one of the sores between the toes I picked out a small spicule, which I could not understand until the patient told me he used a loofah in cleansing his feet.

Tertiary syphilis of the tongue has led to many epitheliomata there being overlooked, and I would caution against being misled by a temporary improvement following administration of arsenobenzene preparations in these cases. The tendency to regard all lesions on the genitals as venereal in origin sometimes leads to an epithelioma of the penis being diagnosed as gumma.

Gummata of the testicle have fairly often been removed under a diagnosis of new growth. In one case which I remember I feel sure that the practitioner and also the pathologist were misled by the blameless life of the patient, and it was not until many months later, when an undoubted gumma occurred elsewhere, that the blood was tested.

I think the tendency in nervous diseases is to think of syphilis and in cardio-vascular disease to forget it. At least quite a number of cases of nervous disease due to causes other than syphilis have been sent to me for treatment, and I have seen a fair number of cases of syphilitic myocarditis in which I wished my predecessors had thought of syphilis earlier and left me with less of a wreck to treat.

In questions of treatment it is always a source of wonder to me that there are so many people who give only partial treatment if there has been a doubt about the diagnosis. They would probably agree at once that a thing cannot both be and not be, but yet they act as if it could.

In patients under antisyphilitic treatment failure to diagnose certain skin rashes as due to arsenic and continuance of the treatment has led to most tragic results; so much so that one would emphasize with all one's strength the advice to take serious notice of any dermatosis in a patient undergoing arsenobenzene treatment, however trivial the skin affection may seem. One should consider most seriously whether or not this is arsenical, and decide on continuance of this form of treatment only on firm conviction that it is not. Otherwise the result may be a dermatitis from which the patient may die, or recover only after a long and trying illness. I ought to mention here that bismuth sometimes causes a troublesome dermatitis, though it is very uncommonly so acute as can be one due to arsenobenzene.

I have thus attempted to set out some of the commoner errors in diagnosis in questions of venereal disease. The subject could be expanded to far greater length, but even if space would permit of this the result would only emphasize still more the principle that, in venereal diseases as in others, one must look for and gather all the evidence which will bear on the diagnosis. And, having gathered the evidence, one must sift it with judgement and common sense. If this discussion leads to some reduction in the wretched victims of wrong diagnoses—the poor devils who go through life under the shadow of an anxiety for which there is no real reason—it will have done some good.

THE PRESENT POSITION OF SILICOSIS IN INDUSTRY IN BRITAIN.*

BY

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DURING the last two decades the subject of silicosis has become increasingly popular as a topic for contributions to medical literature, and the treatment of the subject has been undertaken from so many different angles that it seems to be an act of supererogation to return to this topic at the present time. There are, however, one or two aspects of the subject which I have felt should be brought directly to the notice of the medical profession, and these are concerned with (1) the occurrence of the disease in the various industries throughout this country; (2) the manifestations of the disease as it presents itself to medical practitioners; and (3) the legislation which has been brought into being within recent years.

For the purposes of this paper I will assume a general knowledge of the etiology, symptomatology, and pathology of the disease, but those who have made a special study of the subject will pardon me if I review briefly a few points on this aspect of the subject.

ETIOLOGY.

Silicosis is produced by the inhalation of particles of silica breathed in with the atmospheric air in the course of a person's employment. In silicosis, properly so-called, the silica is in the form of free dioxide of silicon (SiO_2), in the form of quartz, flint, chert, etc. The particles to produce the disease must be inhaled into the ultimate pulmonary tissue—that is to say, into the alveoli—and must therefore be of a degree of minuteness which will permit of their free passage in the mid-current of the air through the upper respiratory passages and the finest bronchial tubes. Many of the particles return with the expired air, but a proportion of them will adhere to the alveolar walls, as well as to the walls of the bronchi. In the latter case particles are caught in the mucus and removed by the action of the cilia. Those particles which fall on the walls of the alveoli are taken up by phagocytes, which move towards the lymph channels and interspaces in an effort to remove the silica. Where the amount of dust is small the action of the phagocytes is likely to be effective, but where it is great this action is imperfect, the phagocytes, overloaded with dust, becoming detained in their progress. The silica acts on the cells so that they lose their motile power. The result is aggregation of these cells in the lymphatic channels, especially the perivascular and peribronchial lymphatics. A deposit of these cells produces a reaction in the surrounding tissue; fibroblasts are produced which change ultimately into fibrous tissue, and so long as the action of the silica proceeds the formation of fibrous tissue continues, from a centre towards an extending periphery. The fibrous tissue undergoes condensation and contraction, and, pulling on the remaining lung tissue, produces distortion with loss of function. It seems likely that blood vessels are obliterated by compression from the perivascular fibrosis before the corresponding bronchi are obliterated, and I have thought that this might explain the absence of cyanosis in chronic forms of silicosis, in

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contradistinction from respiratory diseases of a more acute type, in which the vessels remain patent after the corresponding area of lung tissue has ceased to carry out its aerating function.

PATHOLOGY.

The pathological characters of a silicotic lung vary according to the progress of the disease. When this is of a very chronic type there are apt to be large areas of dense tissue, extremely hard and black on section, and showing no remnant of lung tissue. These areas may be two or three inches in extent, especially about the upper or middle zones of the lungs, and they are associated with extensive pleural thickening and adhesions. The remaining lung tissue between the nodules or solid masses of fibrous tissue is usually in an extremely emphysematous state. In such lungs it is common to find small foci of tuberculosis embedded in the dense fibrous tissue. These may show caseation or cavitation, but in some cases this is the only macroscopic manifestation of the disease. In other cases tuberculosis of the usual ulcerative progressive type is found, and this may be the ultimate cause of death. In still other cases no massive fibrotic areas are found, but throughout the whole of both lungs nodules are present, varying in size from one or two millimetres to about a centimetre in diameter. On the pleural surface the nodules are represented by minute elevations, like sago grains, covered with a whitish layer owing to the thickened pleura. They feel shotty to the touch and are intensely hard. Where the lungs are so free from pleural adhesions that they can be removed from the chest cavity, these nodules on the outer surface of the pleura present an almost characteristic appearance. In fatal cases of this diffuse discrete type of nodular silicosis, where tuberculosis has not supervened and proved to be the cause of death, there are usually found areas of broncho-pneumonia and congestion. There seems no reason to doubt that silicosis may prove fatal without the intervention of tuberculosis.

Microscopic examination of lung sections shows the nodules to be formed in a characteristically whorled arrangement of the fibrous tissue. The oldest tissue is at the centre of the nodule, and often shows a laminated structure with pigmented particles between the fibres. When the particles or masses are large enough—that is, about 5μ —they can be demonstrated by polarized light. Lymphatic glands from the roots of the lungs are enlarged, intensely hard, and on section are black and show fibrous tissue change, and the presence of pigmented particles on microscopic examination.

SYMPTOMATOLOGY.

The inhalation of silica dust is not usually accompanied by evidences of irritation. When free from other dusts of an irritating kind, silica may be inhaled by the workers without any suspicion that they are exposed to dangerous dust, and, indeed, this fact accounts for some of the indifference shown by workers in neglecting reasonable precautions to protect themselves from the risk of inhaling silica dust, and it likewise prevents an employer from suspecting that danger is being incurred by the workers. With regard to symptoms, we must now distinguish between what we might call the chronic form of the disease and the acute form of the disease, but in describing these it must be understood that there is no hard-and-fast line between the two types of cases, so far as we know at present. This is a matter on which light is badly needed. The chronic form is by far the more common. The first symptom is usually dyspnoea, and this may be noticed any time from about five years to forty years or longer after the commencement of the time of exposure. In the production of this symptom two well-known characters of silicosis are important. The first is the cumulative action of silica—that is, each successive dose of silica produces a summation of effects in the anatomical changes of the lung. The second is the latent period which is necessary for those changes to take place. This means that exposure to silica dust is not followed by symptoms immediately, but after a lapse of time, varying from, say, two years to forty years or more, during which fibrous tissue is formed and the function of the lung is interfered with, the symptoms begin to appear. It means also that when a period of exposure to silica dust has occurred these anatomical changes will

proceed even though the subject is removed from further inhalation of silica dust. These two facts together are of the first importance in the prognosis of silicosis and in its control. If it be possible to detect the disease at such an early stage that the amount of silica dust already acting on the lung is less than that necessary to produce disability even when the whole of the latent period is passed, it would be possible, by periodic examinations, to remove from the risk persons with this degree of involvement, so that the disease would never develop to a disabling extent. One of the problems remaining to be solved is just that. Is the disease recognizable at a stage when removal from exposure to silica dust will not be followed by disablement? An important factor in this, of course, will be the intensity of exposure—that is, the concentration of dust in the atmosphere. If the dust cloud be dense, a large amount of silica may be inhaled into the lung before the anatomical changes are produced, and in such a way as to render extensive and severe anatomical changes inevitable.

Apart from dyspnoea, other symptoms are those of any respiratory disease, and they depend on the character and degree of secondary infection and the changes resulting in the lung tissue, the bronchi, and the upper respiratory passages. There is one possible exception to this, and that is in what we have called the "acute form" of silicosis. A few such cases have been noted recently. The onset of symptoms occurred from two and a half to four years after first exposure to inhalation of the dust, and, commencing with dyspnoea, there was rapid development of general constitutional disturbance, with pyrexia and acute illness lasting over a few weeks and terminating in death. In these cases cyanosis was a definite well-marked physical sign, and the whole picture of the illness closely resembled that of miliary tuberculosis. It is possible that the presence of an alkali mixed with the silica dust had something to do with the course of the disease in these cases.

DIAGNOSIS.

Three groups of facts must be available to make a diagnosis of silicosis which can be regarded as satisfactory and final. First, the employment history. Silicosis does not occur without exposure to the inhalation of silica dust. It is necessary that the employment history should be exhaustively studied and that no period of employment should be allowed to escape notice, because a comparatively brief period of exposure may be the only determining factor in a case of silicosis, provided that the latent period is accounted for subsequently.

A clinical examination of the patient can never be dispensed with. The symptoms and course of the disease, as stated by the patient or his relatives, are of the utmost value in making a diagnosis. The clinical examination by inspection of the chest, percussion, and auscultation is never so devoid of result as some of the writings on the subject would lead one to believe. The more one studies the subject the more one is inclined to regard the physical signs as being not only detectable but important, especially in prognosis.

In the radiological examination the technique has now become a science in itself, and so many medical men in the tuberculosis service have made themselves masters of this technique that I leave the subject to be dealt with by those who have modern practical experience. The pulmonary radiograph requires different methods from that employed in connexion with other applications of x rays. The test of a good x-ray film lies in the film itself, and provided that it complies with a certain high standard of technical excellence, the interpretation may safely be left to anyone who has an intimate knowledge of the disease and the appearances which the radiograph provides.

PROGNOSIS.

Prognosis depends on (1) the amount of dust inhaled, (2) the period of time over which this acts, (3) the occurrence of infective processes—tuberculous and non-tuberculous. If diagnosis can be made at such an early stage in the disease that sufficient silica has not been inhaled to produce irreparable damage, and so much damage has not been done as to produce disability, then it seems that the disease can be arrested. I know of no curative procedures

other than those to control secondary infections. When symptoms have become established and the patient is still exposed to the inhalation of dust, it seems inevitable that the condition should progress. This has been the uniform experience. The longer the period of exposure before symptoms have developed, that is, the more chronic the type of the disease, the slower will be its progress, and a period of several years may elapse without any appreciable advance being made in the course of the disease, even when partial disablement has resulted. Even in advanced cases, when dyspnoea is severe, a patient may carry on his work, especially work of a skilled and partially sedentary nature, until within a few days of death. In such extremely chronic cases death usually results from broncho-pneumonia or congestion of the lungs—it may be following influenza.

NOMENCLATURE.

Considering the wide distribution of silica in nature—55 per cent. of the earth's crust consists of free or combined silica—and the many uses to which it is put in industry, it is not surprising that the disease which we have under discussion should be widespread. I will briefly attempt to indicate the main industries in which silica is used in such a way that it may be inhaled by the workers and give rise to silicosis. Before entering on this, however, I would like to say a word on the nomenclature of this subject. Legislation has been brought into force within the last ten years in this country to provide compensation for silicosis, and in that legislation the term "silicosis" is restricted to the disease produced by the inhalation of silica dust. It is necessary, therefore, to restrict the application of the term "silicosis" to the disease condition produced by the inhalation of silica dust (SiO_2)—that is, free silica. This does not mean that the silica must be unmixed with other dusts, but it must occur chemically free, as it does, for example, in quartz, flint, chert, quartzose sands and sandstones, granite, and many rocks and deposits in which quartz or silica in another form is found in substantial proportion. There is a tendency to apply the term "silicosis" to fibrosis of the lung produced by various kinds of dust, and there is a danger that it will be assumed, because fibrosis of the lung is produced by the inhalation of dust, that that dust contains silica in some form or other. It seems to me that this is an entirely wrong method of proceeding. Fibrosis of the lung due to the inhalation of dust should be regarded as such, and the dust producing it should be referred to specially.

The word for fibrosis of the lung produced by dust happens to be "pneumoconiosis," and I feel that one reason why this is not used as much as it ought to be is because of its unwieldy size, and I am going to suggest that the word "coniosis" be used in the same sense, indicating fibrosis of the lung produced by dust. Seeing that there is no other system than the respiratory system which is essentially effected by dust, *qua* dust, the abbreviated term may be allowed to pass as a complete synonym of the word "pneumoconiosis." I would suggest, further, that to obviate the necessity for coining new words by putting "osis" on the end of every conceivable substance which may produce fibrosis of the lungs, it would appear to be more accurate to describe the condition as coniosis due to the inhalation of such and such a dust. This is almost essential when we consider that dusts are not inhaled in a chemically pure condition, that in some dusts, indeed, the chemical composition is not clearly known—they are mixtures; and above all, that in the course of his employment history one individual may have been exposed to the inhalation of several kinds of dust, each of which is capable of producing fibrosis, and he may at length suffer from a condition due to the cumulative effects of the inhalation of those dusts, and the resulting disease condition could not be represented by a single term signifying one substance.

INDUSTRIES IN WHICH SILICOSIS OCCURS.

Refractories Industries.—First amongst the industries in which silicosis is produced should be mentioned the refractories industries. This group of processes employs some 2,000 to 3,000 persons in this country, and is concerned with the manufacture of ganister and silica bricks for use in metallurgical and other furnaces to resist high

temperatures. For this purpose the bricks contain a high proportion of silica, sometimes considerably over 90 per cent. In defining this industry for purposes of compensation under the Compensation Act a restricting term was used, in which the expression "containing 80 per cent. of silica" is used, and by some this has been interpreted as an indication at which a danger line exists for the production of silicosis—in other words, that anything containing over 80 per cent. of silica will produce silicosis and that anything under 80 per cent. will not produce it. This conception is entirely wrong, as the term was used to define the silica-brick industry from the fire-brick industry, and was one of convenience for administration only. The refractories industries were brought by scheme under the Workmen's Compensation (Silicosis) Act in 1919, and by an amending Act certain special medical provisions have been made whereby a specialist medical board carries out periodic medical examinations and examinations for certificates for compensation of all the persons employed. The members of the board are whole-time medical men, with their administrative centre in Sheffield, which is the geographical centre of the industry. The medical board is on lines similar to that of the medical bureau in the goldfields of South Africa. Apart from the administrative value of such a medical board, the members of the present board, Dr. C. L. Sutherland and Dr. S. Bryson, have contributed valuable investigations to the subject of silicosis in industry since their appointment in 1925.

Sandstone Industry.—The sandstone quarrying and dressing industry represents perhaps the most widespread of all silicosis-producing industries. The sandstones include for this purpose the quartzose and quartzite rocks. It must be within the personal knowledge and experience of every one of you that the stonemason working on such rocks inhales almost continuously at his work minute quantities of the dust, and a stonemason's "phthisis" is well known in districts of the country in which sandstone is used as building material. At the present time machining processes are taking the place of much hand work, and as water and mechanical methods of dust suppression can be used, there is likely to be a diminution in the incidence of silicosis. It is still difficult, however, to protect the stonemason who dresses the stone with chisel and mallet. There has been introduced recently a device for the removal of dust from pneumatic hand tools for dressing stone, which is eminently effective. The sandstone quarrying and dressing of sandstone in quarries has now been brought under the provisions of the Workmen's Compensation Act by a scheme of compensation on the same lines as that for the refractories industries.

Granite Industry.—Allied to sandstone quarrying and dressing is the quarrying and dressing of granite. Granite is an igneous rock which consists essentially of quartz, felspar, and mica. In some native granites of this country they occur in about equal proportions. In many igneous rocks, however, the proportion of quartz is much less, or it may be even absent. A study of mortality records indicates that respiratory disease has been produced amongst granite workers to such an extent as to lead inevitably to the conclusion that the dust from granite was an important factor. This is particularly the case in the United States of America, and to a less extent in this country. The subject, however, is complicated because of the varying composition of the rock dealt with. A medical inquiry into the incidence of silicosis amongst granite workers is being conducted jointly by the Home Office and Mines Department at the present time.

Slate Industry.—Reference might also be made to the subject of slate quarrying and dressing. In 1927 reference was made by Dr. Wade of the Welsh Board of Health in a special report on the mortality rate from tuberculosis of the lungs in a slate quarrying and dressing district in North Wales. A medical inquiry was undertaken by the Mines Department, in which clinical and radiological examinations were made of a number of workers in that district. As a result of the inquiry there appeared to be evidence of the production of fibrosis of the lungs, but the occurrence of actual disability within the ordinary working

Lifetime of workers was not so clearly shown. It is possible that in this, as in other forms of fibrosis of the lung, tuberculosis may occur frequently and be the cause of death, while the underlying condition of fibrosis may not be easily recognized.

Grinding of Metals.—"Grinders' rot" is a name associated with the Sheffield trades and the manufacture of cutlery and edge tools, where steel implements are ground on wheels of sandstone. This industry has been notoriously associated with the production of silicosis, and especially the combined disease of silicosis with tuberculosis. The death rate in past years was appalling. One has only to consult the literature of the middle of last century to learn how this industry exacted its toll of the lives of those who took part in it. The grinding of metals is also done as a process in engineering and metal working generally throughout the country. The sandstone wheel or grindstone, while still being used to some extent in many of the processes, is being largely replaced by manufactured wheels of abrasives such as carborundum. One effect of this replacement will be the removal of silica dust from the atmosphere and the more easy control of the abrasive dust by the use of water and mechanical exhaust draught. Dry grinding on sandstone wheels is still carried out in certain industries as in the manufacture of textile machinery, but nowadays localized exhaust draught for the removal of dust is so effective that it is a comparatively safe process. The most serious process in this industry is what is known as "racing" the grindstone—that is to say, the trueing up of the face of the grindstone when it is first brought into use by the grinder. Formerly, no attempt was made to control the dust, and in the short time necessary to "race" the stone the operator could inhale enormous quantities of dust. These heavy pollutions of the atmosphere, even over such short periods, were enough to produce severe silicosis after the latent period, even without the continuous exposure at the grinding wheel itself. Recent devices have been introduced for removing the dust at this process of racing a grindstone, and with this improvement, combined with the extensive replacement of the grindstone by artificial abrasive wheels, we may look for a reduction at least in the number of cases of silicosis amongst metal grinders. A scheme of compensation has been made for metal grinders under the Workmen's Compensation Act. This scheme is on different lines from those for the refractories and sandstone industries in that it applies substantially the procedure under the Workmen's Compensation Act in regard to scheduled diseases. It provides for payment of benefits only in cases of total disablement and death.

Sandblasting.—Allied to the grinding of metals is the process of sandblasting of metals. This is carried on at metal works, and consists in playing a jet of sand under high pressure against a metal surface to produce certain effects. In some cases the worker is confined in a small room, and he is then protected by a close-fitting helmet with an air feed, much as in the case of a diver under water. In other instances the operator passes his hands into a cabinet, the dust being prevented from escaping from the cabinet by means of air suction. In spite of these precautions and a genuine attempt to maintain the plant in good order, cases of silicosis amongst sandblasters are being met with to-day. It is a process which is becoming more extensively used in industry, and it is a matter for serious consideration whether a substitute, such as iron or steel grit, could not be used to replace the siliceous grit as the abrading material. Sandblasting is also used in the etching of glass.

Pottery Manufacture.—Where does the potter get his silicosis or "potters' asthma"? In the manufacture of earthenware the body of the ware consists of an admixture of clays and ground flint. In the manufacture of china the body contains no ground flint, but ground flint is used to embed the articles of ware while they are being fired in the oven, and also for dusting the surfaces on which the ware may be put when in its wet state. In the case of earthenware, therefore, particles of the body which fall

about the benches and floors become dry and are crushed and give off dust which contains free flint. In the case of china, in the process of bedding the china in ground flint and of removing the ground flint after firing, the workers are liable to inhale dust containing flint and thus to contract silicosis. How far the other dusts to which workers are exposed in the manufacture of pottery increase or retard the action of the flint in producing silicosis we cannot say. This is another field in which research is needed. We know that silicosis does occur both amongst earthenware makers and amongst china makers, and in none of the processes in which there is exposure to dust is there absolute freedom of the workers from the risk of contracting silicosis. A scheme of compensation, known as the Various Industries (Silicosis) Scheme, 1929, covers certain workers in potteries, as well as workers in certain other industries and processes laid down in the scheme. These include practically all processes not covered by other compensation schemes where substantial exposure to silica dust occurs.

Crushing and Grinding of Silica, and Industries in which Ground Silica is used.—Ground silica is used in many industries. The silica used is commonly quartz crystal or a highly siliceous quartzite rock. This material is ground fine by machinery, usually dry, and with precautions as to the removal of dust by localized exhaust ventilation. The ground material is placed in sacks and sold in this form. Similar material is also imported into the country from the Continent. Ground silica is used for the manufacture of scouring powders for domestic use, when it is mixed with dry soap powder or alkali or other material, and filled into cartons or packets. It was in this industry that some recent cases of an acute type of silicosis occurred. The evolution of dust is controlled in these processes by the use of mechanical methods of handling, enclosure of machinery, and the use of localized exhaust draught to remove dust or to prevent it from entering into the air of the workroom. Ground silica is used in the manufacture of certain vitreous enamels. It has been used also as a filler in the furniture industry, in the manufacture of insulating materials, and in foundry use. Besides the finely ground silica, flint is sometimes crushed and used in the form of grit for poultry. This crushing process is also accompanied by the evolution of dust, and must be controlled by enclosure of the machinery and removal of the dust by exhaust draught.

Tin-mining in Cornwall has been recognized for many years as an industry with a high mortality rate from tuberculosis, and it has been shown that this was associated with the presence of silicosis. The tin ore occurs in a highly siliceous rock, and in former times drilling of this rock produced much dust, but nowadays drilling is done through hollow steel and with a water feed. Besides the dust in tin-mining itself, a factor in producing this high incidence of silicosis and tuberculosis in the past was the practice amongst tin-miners of working for a time in the gold mines of South Africa, where in former times the amount of silica in the atmosphere was very high and the incidence of silicosis was correspondingly great. Undoubtedly tin-mining in Cornwall can produce silicosis in persons who have not worked outside of this country.

Coal-mining.—Silicosis has been shown to occur amongst workers underground in coal mines. Those affected are the men employed on hard headings, cutting through stone to reach the coal seam. In some cases in which silicosis has occurred the employment on this work has been comparatively short, and it would seem to be the case that once enough silica has been inhaled to produce the nucleus of fibrosis, the resulting inhalation of coal dust into a lung in which the lymphatic drainage has been disorganized has the effect of increasing the fibrosis and producing disability. It seems likely that this type of silicosis is what was classically known as "anthracosis" in the past. We are now aware, however, that coal dust, without any evident exposure to silica dust, may produce a condition in the lungs which radiographically resembles the appearances produced by silicosis.

METHODS FOR DETERMINING THE CONCENTRATION OF DUST IN THE ATMOSPHERE.

A factor of great importance in the production of silicosis is that concerned with the physical properties of the silica, and especially with the fine state of division. In sections of a silicotic lung the particles of silica found are, with few exceptions, under 5μ , and a very high percentage of those are under 2μ , the average size of particles being about 1μ . From what has been said of the etiology of the disease it is obvious that a fine state of division of silica is necessary to ensure its reaching the ultimate pulmonary tissue. Particles under 2μ in diameter are not visible to the naked eye in the atmosphere unless present in such density as to produce cloud effects, and unless particles of this size are associated with the presence of larger particles, or aggregates, which are visible as dust to the naked eye, it is obvious that the visual appearance of an atmosphere is no criterion of its freedom from a dangerous state of dustiness. In investigating the conditions which give rise to silicosis it is necessary to introduce methods for the determination of the concentration of dust in the atmosphere, and several methods of investigating this have been brought into use. The commoner methods may be divided into two classes, depending on the ultimate method of estimation: they are the gravimetric method, and the numerical or counting method.

In the gravimetric method a large measured volume of air is passed through a filtering medium which retains the particulate matter. This is weighed by suitable means and the result calculated in terms of milligrams per cubic metre of air. It is evident that in this method particles of all sizes capable of floating in the atmosphere will be collected, and will be included in the resulting weight, and it follows that the larger particles, which have no pathological significance since they cannot enter the lung tissues, will be represented in the resulting weight in a far higher ratio than the minute particles, which are really injurious, although these are usually infinitely more numerous. The method, therefore, is suitable only for comparing atmospheric conditions as to dust content where the physical state of the dust relative to size is comparable.

The numerical method consists in the collection of the particulate matter from a small sample of air in such a way that it can be examined under the microscope, the particles measured and counted, and, by calculation, the number of particles in a unit volume of air determined. The advantage of this method is that it collects only the comparatively small particles, and by measuring and counting the particles an accurate estimate can be made of the concentration of injurious particles of dust at breathing level in the atmosphere. The drawbacks of the method are, first, only a small and momentary sample can be taken, and this may not be representative where conditions vary widely from time to time; and secondly, the presence in the atmosphere of soot and extraneous matter, which are liable to obscure the particles under investigation and to render counting somewhat difficult.

Both of these methods have been in frequent use in the Factory Department, and in the numerical method of counting Owens's dust-counter has been used. When one is able to estimate concentration of dust in an atmosphere the question arises, Is it possible to establish a standard of air purity within which there is no risk of injury to health? I may say at once that such a standard has not been suggested in this country. To provide such a standard would have certain advantages in that preventive measures for the suppression of dust could be standardized and, by periodic examinations of the atmosphere, such preventive measures could be controlled. The arguments against setting up a standard are, however, strong. In the first place, intensity of air dustiness varies widely, even from moment to moment, in the same process carried on in the same place, and when regard is had to the vast number of processes materially different in character and carried on under widely different conditions, it must be admitted that the factors influencing the results of tests taken in a brief period of time are so great that conclusions drawn from such results are as likely to mislead as to guide. In the second place, it is not known how much silica must be

inhaled before the disease is developed. In other words, there is no criterion of air dustiness which we can safely regard as indicating freedom from risk of producing the disease. Each of the various factors has its own weight. The concentration of dust in the atmosphere; the period of time over which exposure takes place; the intermittency of the process; and possibly also the presence of other dusts in the atmosphere, which may have either a restraining or an accelerating effect on the action of the silica itself.

Research is needed before such a criterion can be established. This must include: (1) collection of data regarding the incidence and production of silicosis in the various industries in which it occurs; (2) a study of the influence of other dusts and any modifying circumstances affecting the inhalation of the dust; and (3) the results of preventive measures for the suppression of the dust. These data can only be collected and made effective use of by the concerted action of all those whose work brings them into touch with the sufferers from the disease, and co-operation with the central body whose function it is to investigate and to ameliorate or suppress the conditions under which the disease occurs.

SOME NOTES ON THE BIOPHYSICS OF SILICA AND THE ETIOLOGY OF SILICOSIS.*

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AMONGST the main questions awaiting answers at the present time regarding silicosis would appear to be the following:

1. How is the characteristic fibrosis of the lungs which follows the inhalation of fine silica dust, and which is termed "silicosis," produced?

2. Are there any factors, apart from prevention of dust inhalation, which hasten, retard, or otherwise modify the development of the condition?

3. Is the fibrosis of the lungs which we speak of as "silicosis" produced by substances other than silica?

In attempting to answer these questions I would suggest that it will help if we review briefly some facts regarding the biophysical behaviour of silica and its use in therapeutics—facts which seem to have a bearing on the points at issue, and not to have received hitherto the attention which their importance merits.

In the *Journal of Biological Chemistry* for November, 1928, Earl J. King published the following table¹ giving the silica content of certain organs of the guinea-pig, rabbit, and dog, and of normal and silicotic lungs in man:

Organ.	Animal.	Mg. of Silica per gram of Organ.		Total in Organ.
		Dry.	Fresh.	
Normal lung	Guinea-pig	0.78	0.12	0.5
" " " " " "	Rabbit	2.02	0.30	2.93
" " " " " "	Dog	2.03	0.32	60.72
" " " " " "	Man	1.40		
Silicotic lung	Man	123.55		
Normal kidney	Rabbit	1.97	0.25	2.96
" " " " " "	Dog	1.12	0.21	7.28
Normal liver	Rabbit	1.70	0.32	10.50
" " " " " "	Dog	1.33	0.34	7.71
Normal spleen	Dog	0.66	0.16	

One would like to see these figures checked by those of other observers, but, as will be seen later, the evidence of the presence of silica in normal organs is convincing.

The remarkable similarity of the figures for the silica content of the organs of the rabbit, a herbivorous rodent,

* Read in the Section of Occupational Diseases of the Annual Meeting of the British Medical Association, Manchester, 1929.