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A GENERAL OVERVIEW OF SILICOSIS

Silicosis is an occupational disease. It is one of the pneumoconioses, a generic group of lung diseases associated with dusty occupations, and is not an infectious disease. Pneumoconiosis is an abbreviated version of the term pneumonokoniosis - meaning 'dusted lung' - coined by the German pathologist F.A. Zenker in 1866. The specific lung disease silicosis, derived from the Latin word, 'silex', meaning a flint was delineated by Visconti in 1870. This disease is caused by free silica dust inhaled by men or animals.

Apart from asthmatics most dusts inhaled by men or animals are innocuous to them and produce only pigmentation or slight chronic inflammatory reactions. However certain forms of mineral dust, which are non-toxic in the ordinary sense of the word, are fibrogenic. This means that if they are inhaled continuously over long periods they are capable of penetrating to the deep lung tissue and provoking the production of fibrous tissue - that is produce fibrosis - with consequent impairment of lung function. Exposure to these dusts is almost exclusively occupational and the manifestation of resultant diseases vary according to the type of irritant. Inhalation of dust from silica, coal, asbestos and other silicates, and beryllium leads to the development of silicosis, coal workers' pneumoconiosis, asbestosis and silicatosis, and berylliosis respectively. Mixed dust pneumoconiosis can result from inhaling normally inert dust admixed with small amounts of silica. Tin dust and barium dust can cause stannosis and baritosis; but these are benign forms of pneumoconiosis. They produce radiological changes without fibrosis.

Dust diseases of the lungs have been traced far back in history and it has been suggested that paleolithic flint miners died of the oldest occupational disease, silicosis. As industrialisation progressed it was realised that numerous kinds of dusts provoked lung diseases and those were given graphic names according to the kind of occupation or trade involved. For instance 'grinders' rot' and 'potter's rot' were both classical forms of silicosis. But the situation was puzzling because of the widespread incidence of pulmonary tuberculosis - long recognised as a disease of the market place, synonymous with civilisation and increasingly endemic in the overcrowded working and living conditions of the industrialising 19th century Europe. 'Miners' phthisis' was the incorrect term applied to the disease-stricken gold miners of the Transvaal, who in fact had silicosis. Its derivation is understandable but at the same time it must be clearly recognised that in clinical and pathological terms this definition of the disease is imprecise, if not totally meaningless. Phthisis, like consumption, was the descriptive symptom of the wasting away of the body caused principally by disease of the lungs, notably tuberculosis. Even after 1882, when Robert Koch identified the bacillus responsible for tuberculosis, both dust diseases and tuberculosis continued to be grouped together as consumption or phthisis. Gradually distinctions were drawn between the two types of disease. There was 'dust phthisis' or 'tubercular phthisis' according to whether or not there was occupational dust exposure; and 'miner's phthisis' became a term indiscriminately applied to many kinds of mining, whether or not silica dust was present or not. The term 'phthisis' too continued to be loosely used, and could include many kinds of incapacitating pulmonary conditions.

Although their causes are different and the pulmonary lesions produced by silicosis and tuberculosis are distinctive, the tissue response of the lungs to silica particles and the tubercle bacillus are similar. This caused a number of doctors, including Voch, to designate what is now known as the silicotic nodule the pseudo-tubercle. This similarity became even more pronounced when the two diseases presented simultaneously in the same lesion. This led many authorities to believe that the two diseases were inseparable - that they were both in fact tuberculosis - and while silicosis was recognised as a specific industrial disease, it merely altered the course and appearance of tuberculosis. At the turn of the 20th century, South African doctors did not believe that the majority of silicotic cases were tubercular, although an 'infective' variety was recognised. This was because of the migratory nature of the mining population many of whom returned home overseas to die. Their theories about the non-tubercular nature of silicosis were not proved, and controversy about the disease continued to exist, both in South Africa and Britain, concerning its cause. There was disagreement as to whether silicosis or tuberculosis came first. On the whole, however, it was agreed that silicosis could not present without tuberculosis; the element of tuberculosis was always necessarily present either in the original development of the nodule or it had to be superadded. Thus the terms silico-tuberculosis and tuberculo-silicosis were coined.

In 1926 two South African doctors, F.W. Simson and A.S. Strachan, began their investigations and by 1935 had proved conclusively that nodular fibrosis produced by lung tissue as a simple response to silicious dust did not necessarily have to possess an element of tuberculosis in the original development of the lesion. Despite these findings controversy continued to exist about tuberculosis and its association with silicosis. An American doctor, LeRoy U. Gardner, believed that when the two diseases co-existed neither could be classified as silicosis or tuberculosis. He suggested that it was a disease entity of its own. As late as 1947 A.G. Heppleston, one of the foremost British authorities on pneumoconiosis, especially coal-workers pneumoconiosis, regarded all cases of complicated silicosis (progressive fibrosis) as tuberculous in origin. It is for these reasons that a basic understanding of silicosis and tuberculosis is necessary.

Silicosis is a man-made disease. From the earliest times man's ingenuity in manipulating the products of his environment has brought him into contact with silica dust. As his technology and technical skills have evolved so his ability to use silica products has increased. In addition, the development of technical aids and the use of high-powered machinery has enabled him to increase the speed of his productive output. The result has been a concomitant increase of silica dust and an increased incidence of silicosis on a world-wide scale.

The element silicon (Si) is widely distributed in nature. It comprises approximately 28 per cent of the earth's crust and is second in abundance to oxygen (O_2). Although silicon is found in practically all rocks and many sands, clays and soils, it does not occur in its uncombined form. It either combines with oxygen to form chemically free silica (SiO_2 , that is silicon dioxide) - and this is the cause of silicosis - or with oxygen plus other elements to form more complex chains called silicates; and asbestos, for instance, is one of these silicates. It is the ubiquitous presence of silica minerals and their importance in a great variety of industries which create the health hazard of silicosis for a great number of workers.

Silica occurs as a pure mineral in a number of crystalline forms. Of the macrocrystalline forms quartz is by far the most commonly encountered. It occurs *inter alia* in sand, sandstone, quartzite and certain granite formations. Tridymite and cristobalite are uncommon varieties of macrocrystalline silica found in meteorites and certain volcanic rocks; while coesite and stishovite are even rarer types found

in meteorite craters. It is believed that the crystalline structure peculiar to silica accounts for its potential for lung damage. In its microcrystalline form silica is found, for example, in chalcedony of which true flint is a form. In amorphous or cryptocrystalline forms - that is those with hidden crystals - silica is found in the gemstone, the opal, and in diatomaceous earth which is composed of minute skeletons of fresh-water or marine aquatic plants. The amorphous forms of silica are not regarded as having fibrogenic properties. However they may acquire them if heated and melted (asked).

Quartz has special qualities which makes it valuable in a number of industries. Its peculiar crystalline structure enables it to resist fracture or cleavage giving it the required industrial hardness. It is ubiquitous and relatively cheap. It is of great importance in the refractory industries because of its high melting temperatures, its low co-efficient of expansion and the inertness of its high temperature forms.

Here are listed some of the occupations in which workers are exposed to the hazards of silica. Sand, the essential ingredient of concrete and mortar, consists largely of quartz. It is also used for sand-blasting, glass-grinding and the manufacture of miscellaneous abrasives. It is interesting to observe that the use of scouring powders and abrasive soaps, which contain flint and quartz, has been recently discontinued in Britain because of the hazard to workers in this field. Gannister, a variety of sandstone shale, and is referred to in the early medical literature, is combined with clay in the construction of furnaces, whilst other forms of silica are used in iron and steel foundries. Under these high temperature conditions quartz can be converted into the more dangerous forms of tridymite or cristobalite.

'Glass sand' or 'potter's flint', derived from grinding or crushing rounded flint pebbles (as are certain abrasives) is highly sought after in the manufacture of optical glass, china, earthenware and other pottery products, such as tiles and ceramics. This is because of its pure silica content and freedom from other impurities that could stain the products. The dry grinding of the calcined pebbles creates a serious silicosis risk, as does dusting off the loose sand from the finished product which has been kiln-fired at very high temperatures. Vitreous silica or quartz glass, used for making precision instruments, also falls into this category.

Sandstone and quartzite are used for the manufacture of building stones, and in road and railway constructions. The use of sandstone and granite in stone cutting, polishing and the cleaning of monumental masonry exposes workers to high concentrations of silica, a hazard compounded by the fact that the workers are in close contact with their materials. Workers using high powered pneumatic handdrills are exposed to silica if they should remove their protective clothing in order to inspect their work more closely. Workers are also exposed to high concentrations of silica dust in quarrying, tunnelling and mining. Workers who quarry granite, quartz, sandstone and slate, or who perform a variety of related tasks, such as rough-hewing the blocks, cutting, shaping and crushing the stone, at the face of the mine or quarry or in its immediate vicinity, are exposed to varied but high concentrations of silica dust. While wind can be beneficial to outdoor workers, it is not always possible for them to be on the windward side of the dust-production point or to avoid dust generated by their fellow workers. It is possible that

historical buildings in Edinburgh (and the Union Building in Pretoria) were costly in terms of silicosis of the workers.

Despite modern methods for dust measurement, suppression and prevention, as recently as 1956 tunnellers working on the vast hydro-electric scheme in the Swiss Alps, known as La Grand Dixence, were contracting silicosis. Water was used to suppress dust in the larger tunnels, but dry drilling methods were used in the smaller tunnels.

It is in the gold mining industry that silicosis is of most importance to this study. Gold which is mined on the Witwatersrand is embedded in a quartzite matrix containing 60 to 80 per cent quartz. Mining procedures produce airborne dust with high concentrations of silica which in 1958 measured just under 40 per cent.

It was originally believed that coal mining did not present the hazard of a dangerous pneumoconiosis. But this was disproved and in 1943 legislation in Britain awarded compensation for this disease. There are two schools of thought about the causation of this disease. The first school believes that coal by itself can cause the damage, while the second school maintains that damage is caused when there is an admixture of silica in significant quantities. This is found to be the case, inter alia, of the 'hard-headers', who tunnel and branch to reach the anthracite deposits in South Wales and in Somerset (in South West England). Coal dust by itself, with small quantities of admixed silica, does not cause serious damage, although incautiousness can result from massive depositions of dust in the lungs. The disease however becomes very serious if an infectious element, such as tuberculosis, is superimposed.

This discussion on the possibility of coalworkers contracting silicosis is relevant to the early period of gold mining in the Transvaal. Medical authorities realised that many of the Cornish tin-miners may have had silicosis before they arrived in South Africa, because the incidence of silicosis in Cornwall was high. However they do not seem to have considered that coal-miners too may have contracted the disease in England prior to their advent in the Transvaal.

Dust is everywhere in the atmosphere of the earth and, ^{if inhaled} the respiratory system of man has highly efficient mechanisms for its elimination. The penetration of quartz rock and the manipulation of its products (in an ever increasing number of industries) leads to the production of dust clouds. These vary in concentration from industry to industry, from mine to mine, and even in working places (both on the surface and underground) in a single mine. It is interesting to observe that in desert sand-storms (as in some industrial processes) the free silica in the airborne dust may exceed concentrations of 60 per cent. Yet these desert dwellers and workers do not develop silicosis because the dust particles are coarse and are not inhaled. From this it is clear that in a mine or industrial enterprise only a certain portion of the total airborne dust constitutes a health hazard.

The toxic fraction of the airborne dust must be able to remain suspended in the air for a reasonable length of time and the dust particles must be sufficiently small - less than seven microns in diameter - (a micron is equivalent to $\frac{1}{100\ 000}$ of a millimeter)

so that dust can not only be inhaled but also be retained in the lungs. The silica particles which penetrate to the alveoli in the depths of the lungs, and which cause silicosis, are those of approximately one to three microns in diameter. It should however be noted that the coarser fraction of the respirable dust should not be overlooked or underrated in terms of its damaging potential, and may be of significance in causing chronic bronchitis, a chronic obstructive airways disease. The visibility of a dust cloud is caused by particles which do not constitute the respirable fraction. However, it is important to realise that the air may be contaminated by particles less than ten microns in diameter without any evidence of haziness.

A variety of environmental and occupational and even biological differences may affect each individual's susceptibility to or relative immunity from occupational disease. There is a great deal of research being conducted at present on epidemiological lines on the toxic parameter of a dust cloud, using both animals and human volunteers. Many workers have spent a considerable number of years working in dusty occupations, but have died from causes other than dust diseases. However dust diseases have directly caused the deaths of thousands of other workers. Individual immunological variations have been ascribed to dust clearance and deposition mechanisms of the respiratory system which may explain why some workers in an identical occupation contract disease whilst their fellow workers do not.

Other factors also need to be explained. The relationship between dust dosage and host response is important. It is not clear, for instance, whether short periods of exposure to intense dust concentrations are more dangerous than exposure to lesser dust concentrations over prolonged periods of time. Individual patterns of breathing may be of significance. Certain strenuous jobs cause workers to breathe through their mouths thereby bypassing nasal filtration mechanisms; and this may affect the rate and concentration of the dust deposited. Recent experiments have also indicated that the nose is a far more effective filter than was previously thought, and epidemiological studies in this field are also being conducted. In assessing all these variables, epidemiological studies must also assess the length and nature of a worker's previous occupations.

Experiments have been undertaken to find out whether the effects of tobacco smoking, especially cigarettes, modify the deposition or clearance of dust particles. Tobacco smoking and environmental pollution are major causes of chronic obstructive airways disease, such as chronic bronchitis and emphysema. Research has not yet conclusively shown whether chronic obstructive airways disease will cause an increase in the number of particles trapped in the larger airways, and hinder their clearance by tobacco-or smoke-impaired cilia of the larger air passages. Many authorities believe that tobacco smoking is not in the interests of miners. Smoking, apart from the risk of causing cancer, can damage the surface cell layer of the respiratory tract and increase the worker's susceptibility to infection and/or disease.

Of great significance is the result of recent epidemiological studies conducted over a ten year period. These have shown for the first time that long term dust exposure can contribute to the development of chronic bronchitis although less so than tobacco smoking; but this in itself will not necessarily

predispose the individual to silicosis. There is disagreement as to the relationship between occupational dust exposure and chronic bronchitis. I. Webster is unconvinced of the connection on the South African gold mines while G. Leathart suggests keeping an open mind on the question.

A great deal of further epidemiological work is needed to define the toxic fraction of a dust cloud. This definition may well have to include the coarser particle fraction of the airborne dust cloud since this may be one of the causes of a specific dust disease, chronic bronchitis. Not only mass, but also particle surface may be more important than microscopic size. There is also the need for epidemiological studies, over long periods, in the areas of environmental pollution and the effects of humidity. Once all these and other factors have been explored, present day dust measuring devices may have to be modified by dust control engineers to cater for individual responses to dust, based on new criteria for the assessment of its respirable and toxic fraction.

In the period under discussion (in the paper that follows) the idea gained acceptance that dust particles were responsible for silicosis. But it was not until the 1930s, when the thermal precipitator was used to extract and to measure accurately quantities, size and frequencies of the dust produced by the various underground occupations, that a real understanding of the complexity of the dust-measurement problem began to emerge.

In order to determine the degree of risk of disease in the industrial environment, instruments and different methods have been devised to measure the degree of exposure of workers to respirable free silica. This is an important aspect of disease control as it can help establish (and thereby help minimise) the relationship between the environment and the occurrence of disease.

Dust sampling instruments include the modified konimeter, based on the principle of the model devised in 1915 by Robert Nelson Kotze, the South African government mining engineer. This instrument uses a modified method of impingement. Originally dust particles were sucked onto a plate covered with a thin film of adhesive, such as petroleum; and the number of particles collected within a specified time on that 'spot' was examined and calculated with the aid of a microscope. Readings taken by this method, however, were tedious and not always accurate. Particles were fragmented on impingement causing readings to be too high and thus exaggerating the dust hazard. Newer types of high velocity midget impingers, which can reduce particle fragmentation by the use of water or alcohol for the collection of particles, have improved the konimeter's accuracy. But it still cannot efficiently measure particles less than one micron in diameter. Newer instruments include modified thermal precipitators, the use of electric precipitation and filtration, and personal gravimeter dust samples, the efficacy of which depends upon the co-operation of the miner.

Control of dust concentration is very important; but its suppression is even more so, and this must remain the basic measure in the ~~restoration~~ ^{control} of occupational pulmonary disease. Ventilation is of primary importance. It must assist in dust suppression, but it also provides workers with comfortable temperature and humidity working conditions. Methods of mining

also play an important part in dust allayment. Sufficient time must elapse after blasting before miners are permitted to return to the blasting face; and improvements in the techniques of using explosives, in order to reduce their toxic dust fraction, must continue to be explored. Similarly machine operations and methods of mining need continual modification to reduce the proportion of fine dust produced.

The use of water, despite some of its deficiencies, has long been recognised in the control of dust in mines. It should be used wherever possible in drilling and other dust producing occupations to limit dust dispersion and to suppress airborne particulate matter. These include the wetting down of broken rock after blasting, both before and during transit.

Dust control by these means is the main factor in safeguarding workers. Nevertheless in certain circumstances it may be necessary to protect the individual further by providing him with specialized clothing, particularly a respirator. While R. Archibald claims that advances in the design of respirators have made them efficient and comfortable to wear, other writers throw doubt on their comfort when worn for long periods. The importance in the wearing of protective clothing lies in the individual responsibility of miners. Failure to comply with regulations can expose individuals to dust hazards.

Technological improvements in machinery have led to increased dust formation and dispersion in mining procedures, but better ~~constraints~~ in the environment and observance of modern standards of dust control and ventilation by management and workers, should result in workers being exposed to lower concentrations of silica. Already less noxious abrasive materials have been substituted, for example, in sandblasting operations. Attempts have been made to find prophylactic substances which will prevent the action of silica in the lungs. Aluminium powder has been tried, but not with total success, in Canadian goldmines, in conjunction with the use of other dust suppression methods. Aluminium delays but does not eliminate the production of silicosis. Webster demonstrates clearly that any prophylactic measure must be 'uniquivocally' successful in preventing fibrosis, otherwise miners or industrial workers will become lax in the use of dust prevention methods.

Finally, preliminary examinations of applicants for employment should become standard procedure. This measure would prevent workmen with defective respiratory physiques from entering dangerous employment. It would also help to detect workers with pulmonary tuberculosis who would both expose themselves to additional risks by remaining in dusty occupations and who could possibly infect their fellow workers.

In order to understand the disease process (namely the pathology) of silicosis it is necessary to have an elementary understanding of the anatomy and physiology of the lungs and their defence mechanisms. The lungs which are situated within the thorax are separated from each other by the heart. Each lung is enclosed by a two-layered membrane, the one layer being continuous with the other, to form an invaginated sac called the pleura. The inner lining adheres closely to the lung tissue which is called the lung parenchyma. The outer lining adheres to a number of adjacent organs such as the diaphragm and chest wall, etc. The main functions of the lungs are the exchange of gases which are

essential for the life processes, and the maintenance of the metabolism of the body through the formation and utilization of many substances necessary for a healthy existence.

The respiratory tract consists of a conducting zone and a respiratory zone. The functional unit of respiration is the lobule which comprises tiny ducts called bronchioles, each of which in turn opens into a varying number of alveolar ducts which terminate in minute irregularly shaped cavities known as the alveoli or air sacs. The alveoli constitute 90 per cent of the entire lung volume and the alveoli give the cut surface of the lung its familiar sponge-like appearance. This extraordinarily large surface area, where respiration takes place, explains the vulnerability of the respiratory zones of the lungs to substances from an increasingly hostile environment.

Lining the tubules of the respiratory tract is a layer of surface epithelium which rests on a basement membrane composed of a protein fibre called collagen. Deep to the epithelium is the smooth muscular membrane which plays an important part in the defence mechanism of the respiratory tract because it moves foreign material and secretions to the cough centres. The epithelial layer constitutes an important defence mechanism. It consists of basal cells situated on the basement membrane and other cell types are said to arise from these. The columnar cells, some of which have fine 'hairs' called cilia, line the hollow respiratory tubules and have an important defence role. Ciliated cells, although they have movement of varied rate and direction, have the propensity for continuous movement which wafts foreign matter along the tracheobronchial tree whence it is removed through bronchotracheal secretions and sputum. Loss of ciliary action can be caused by tobacco smoke, irritants or toxic gases (such as nitrous oxide) and fine suspensions of silica dust. These substances also affect the property of extensibility allowing the lungs to expand and relax as air fills them and is exhaled. Emphysema and other dust diseases affect and destroy this elasticity.

The development of the electron microscope has enabled a closer study of the alveolar walls and the identification of alveolar cells. There are two types of alveolar surface (epithelial) cells called pneumocytes type 1 and type 2. Pneumocyte type 2 is a larger cell than type 1, but is less numerous. It is probably the source of the surfactant, lining the lower respiratory tract. Without surfactant the alveoli would probably collapse, for the alveoli are devoid of muscle and cartilage. Surfactant is a substance which reduces surface tension. It thereby neutralises the difference in surface tension between the alveolar spaces and capillaries. This prevents the plasma and red blood cells being pulled into the alveolar spaces and causing the alveoli to collapse.

Mention must also be made of the free-lying alveolar macrophage (or phagocyte) cells. These are found in the alveolar spaces. For many years it was believed that these 'dust' cells were derived from the epithelial cells, but certain present day views hold that they may be derived from the blood monocytes. Monocytes in the blood and macrophages (or histiocytes) in the tissues possess similar structures and functions and belong to the class of cells called nonnuclear phagocytes. These cells constitute one of the body's defence lines against invading organisms; and both blood monocytes and tissue histiocytes are

capable of phagocytosis.

In phagocytosis the cell membrane depresses to engulf a foreign body. The cytoplasm of the cell then flows out and around the particle encircling and enclosing it. The upper portions of the depressed part of the cell membrane eventually break, all the free edges of the membrane join; and within the cell is to be found a tiny phagocytosed particle enclosed within a little membrane.

Within the cytoplasm one of the important tiny structures, called organelles, is the lysosome. This is bounded by a membrane and contains many enzymes which help digest the foreign particles. It is believed that the membrane of the ingested particle fuses with the membrane of the lysosome to form a phagosome. Enzymes are released and the particle may then be digested or killed. However in some cases the ingested material is resistant to the enzymes and when the cell dies the former is released. This occurs sometimes in the case of the tubercle bacillus. In other cases the ingested material may be toxic and will kill the cell causing its release. This is believed to be the explanation when phagocytosis of silica particles occurs.

As gas exchange is one of the important functions of the lungs it is necessary to understand how this occurs, also how the lungs are supplied with oxygenated and nutrient blood, and finally how the drainage by the lymphatic system of the lungs helps augment their defences. Venous blood carrying carbon dioxide is supplied to the lungs by the pulmonary artery which leaves the right ventricle of the heart. This blood supply reaches the broncho-pulmonary segments of the lungs by the dichotomous branches of the pulmonary artery. In direction and size these vascular branches eventually closely follow those of the bronchial tree and ramify to form a thick capillary network. This venous network alone serves the alveoli and comprises a surface area approximately the same as that of the alveoli, namely 70 to 80 square metres. The blood in these capillaries absorbs oxygen through the fine walls of the alveoli and returns carbon dioxide, the waste product of combustion, to the alveoli for exhalation by the process of respiration. Oxygenated blood from the lungs returns to the left atrium of the heart by means of the capillaries and branches of the pulmonary venous trees. It is then conveyed to the tissues of the whole body which can neither live nor function without oxygen.

Similarly the lungs too must receive oxygenated blood for the nutrition of the bronchi and bronchioles. This is provided by the branches of the bronchial arteries which leave the aorta. The capillary network of this source of supply terminates at the respiratory bronchioles and the alveoli receive only venous blood.

The lungs are also equipped with a mechanism for drainage called the lymphatic system. The lymphatic system consists of a network of minute thin-walled vessels which commence blindly in the tissue spaces. These are called lymph capillaries and are even finer than blood capillaries. Their walls however are permeable to substances of greater molecular size than blood capillaries. They are permeable to fluids and permit the free passage of mobile phagocytes, such as alveolar macrophages, into and out of their vessels and nodes. Inside the lymph vessels there circulates a clear colourless fluid called lymph. These vessels form pathways for the collection of particulate matter from the tissue spaces.

and ultimately they empty their contents together with the lymph into certain veins.

The lymph capillaries join to form larger trunks; and within an organ they usually traverse one or more lymph nodes before entering the venous blood stream. The lymph nodes are small, oval, bean-shaped bodies, and consist of small solid masses of lymphoid tissue into which the vessels, along their course, pour their contents. Within the lymph nodes foreign bodies and particulate matter such as dust, noxious substances, bacteria and micro-organisms are digested or deposited. There are two sets of lymphatic vessels in the lungs both of which largely drain into the tracheo-bronchial lymph nodes. The first traverses the surface of the lungs in the tissue of the pleura. The branches of the second system of lymphatics closely follows the course of the bronchi and blood vessels.

Having earlier discussed the toxic parameter of a dust cloud, it is important also to establish the reasons why silica dust causes fibrosis of the lungs, and why the magnificent defence mechanisms of the lungs (some aspects of which are still poorly understood) are able to be breached. Notwithstanding the fact that every man is characterised by his own individual umbrella, (a fact which makes him more or less susceptible to environmental agents producing lung diseases) the defence mechanisms of man's respiratory tract are similar in many respects. In this analysis the emphasis will be placed on inhaled particles, although the inhalation of gases and the entrance of microorganisms inhibit the lungs' efficacy (to some degree) in dealing with particulate matter.

Inhaled particles meet with formidable resistance. The incoming airflow containing particulate matter is subjected to turbulence and sharp directional changes in the nasal passages, pharynx and tracheobronchial tree. Inertial forces and gravitation play an important role at this stage. Particles have their own velocity and this together with changes in the direction of the air flow (in the nose and at the branching points of the larger air ways) lead to the impaction and deposition of the larger particles. By these means the respiratory membrane is protected from the deposition of particles larger than two or three microns in diameter. These larger particles are usually cleared within two to three hours by the ciliated epithelium. Particles smaller than 0,2 to 0,3 microns in diameter do not appear to be deposited and are subsequently exhaled. However there is an intermediate size range of particles which is able to bypass the aerodynamic filtration system and to reach the alveolar region. This is particularly evident in industrial processes which produce high concentrations of dust with a varied range of particle sizes; and it is at this stage that gravitational forces begin to exert a prominent effect.

Within this pulmonary region particles may be isolated within phagocytic cells, alveolar macrophages and scar tissue. The particle-laden cells may then be physically transported out of the lungs by the ciliary movement, or incorporated into the alveolar or small airway walls and eventually reach the lymph nodes via the peribronchial or perivascular lymphatics; or they may be totally digested in the lungs. Physical transport agents appear to act in conjunction with local detoxification agents or processes, like that of phagocytosis (particularly by the alveolar macrophages), with the result that inhaled foreign bodies may be killed,

digested, detoxified or neutralised whilst they are being transported, thereby maintaining pulmonary sterility. Clearance of such particulate matter - whether it be by mechanical barriers, detoxification agents and transport agencies - takes considerably longer than ciliary clearance from the tracheobronchial tree. Theories have also been postulated about the possibility of local immune processes assisting pulmonary sterility, although these theories have not been conclusively proved. However, it is important to realise that the influence of toxic gases and heavy particle loads may diminish the competence of the phagocytes to ingest the particles and hinder their ability in digesting them. Similarly physiological damage to airways caused by epidemiological factors such as tobacco smoke and nitrous oxide may inhibit the efficacy of the alveolar macrophages, or suppress other forms of pulmonary defences. The incidence of a wide variety of detrimental environmental agencies may alter each individual's personal immunity and may predispose him to the development of chronic obstructive airways disease, which in turn may affect his immunity to silicosis.

While not discounting individual immunity to silicosis it should be observed that the reaction to silica in the lungs is out of all proportion to the amount of foreign material present, small quantities of silica producing large amounts of fibrous tissue. A number of theories have been promounded concerning the mechanism by which the damage is done and the reasons why silica should stimulate this severe biological activity in the lungs in contrast to its low chemical activity. Although many of the theories have been discredited, it should also be borne in mind that the macrophage theory (described below) and which is currently accepted by many medical authorities, may not be the full explanation of the fibrogenic activity of silica dust.

For many years the fibrogenic mechanism of silica was attributed to its physical or mechanical properties. It was believed that the lung lining was injured by the hardness and sharpness of the mineral particles; and through mechanical abrasion and laceration of the tissues the way was opened for infection. This theory was held at the beginning of the 20th century when the mortality caused by silicosis first caused concern on the Witwatersrand gold mines. An American doctor, Leroy U. Gardner, was one of the experimental scientists responsible for disproving this theory. It was shown that carborundum and diamond dust, although equally angular and sharp, did not possess the toxicity of silica. It was also demonstrated that while non-crystalline amorphous silica produces fibrosis, stishovite, a crystalline form, does not.

In 1935 a British doctor, P. Heffernan, suggested that freshly fractured quartz had greater fibrogenic action than old quartz. This theory was also invalidated.

A more sophisticated physical theory - previously rejected - suggests that the piezo-electrical charges, which manifest themselves when quartz crystals are subjected to strain, are responsible for the toxicity of silica. This theory has been recently revised by the Russians, but nothing definitive has emerged from their investigations.

From 1922 investigations were dominated by theories involving the chemical properties of silica. One of the major theories, later invalidated, was the 'Solubility' or 'Chemical Toxicity Theory of Silicosis'. This theory, supported by prominent investigators

including W.E. Gye and W.J. Purdy in 1922, and later by Professors M.H. Kettle and H.J. King, assumed that silicic acid liberated from silica, especially quartz, which has a high solubility, would gradually dissolve in the tissue fluids and would stimulate fibrosis. But later King himself, amongst others, showed that the same amounts of released silicic acid bore no correlation with the degree of fibrosis produced. In 1957 a modified version of this theory was expounded by a chemist, Dr. P.F. Holt. Apart from emphasising the importance of the surface of the silica particle in the fibrogenic process, Holt also contended that released silicic acid produced greater amounts of collagen than other dusts thereby aggravating the fibrotic process. However Holt was unable to explain why tridymite, with a low silica solubility, produced more collagen than other forms of quartz with higher solubility rates. Other chemical theories involving in vitro (or tissue models) have so far proved interesting but inconclusive. Such theories include the supposition that carbon dioxide, in conjunction with other components released from quartz particles, causes damage to the tissues.

Five experiments (in vivo) with animals have been performed as models to formulate immunological theories to explain individual susceptibility to disease. Such theories postulate that in certain individuals silica alters the structure of the macrophages in such a way that allergens (or foreign bodies) are produced. These in turn combine with circulating antibodies to produce fibrosis. It is difficult to use animal models as substitutes for humans as their dust dosages differ from those of humans and their environments are dissimilar to the industrial and habitational ones of workers.

Improvements in tissue culture techniques and the use of the electron microscope in the study of cell changes have led to great weight being placed on the validity of the macrophage theory propounded by A.C. Allison and his co-workers. An earlier description of the defence mechanisms of the lungs indicated that inhaled particles which penetrated and were retained by the alveolar regions were ingested by macrophages in which they formed phagosomes. However, in the case of silica, instead of the enzymes of the lysosomes destroying the particles, the lysosomes are themselves destroyed or altered as well as the cell membranes of the phagosome and lysosome. The membrane of the phagosome appears unable to remain intact and to retain its particulate load. Thus when the macrophage dies it releases its contents including the silica particle. Fresh macrophages congregate in the dust foci, ingest the silica and in turn are killed. Thus it is the death of the macrophage which becomes the prerequisite for the subsequent fibrosis that takes place.

While the majority of macrophages containing silica are coughed out, the remaining macrophages remain either in the interstitial (supporting) tissue of the respiratory bronchioles or move to the interstitial tissue around the smaller blood vessels and the lymphatic nodes or nodules close to the smaller veins. With the death of the macrophages and the release of their contents a chronic inflammation is set up. This stimulates a healing reaction. Interstitial cells called fibroblasts, are activated. These form a network of reticulin - a substance closely related to collagen which surrounds the clusters of dust-laden macrophages. On maturation the reticulin fibres form collagen, a type of protein fibrous tissue. These fibrous tissues appear as concentric,

usually symmetrical, collagenised nodules around and near the arteries and bronchioles. Once nodules have formed there is permanent damage to the lung parenchyma. The final process is the hyalination of the collagen in which the tissue fibres become embedded in a glassy matrix; and sometimes assume an egg-shell calcification. These collagenised nodules of fibrous tissue are known as islets and in the simple stage of silicosis are approximately five millimetres in diameter. They are often grey to black in colour owing to the presence of carbon dust deposited with the silica. The fibrotic nodules can cause damage to the blood vessels and bronchioles by creating blood clots or obliterating the inner passages.* Distinctions have been drawn between simple (or chronic) silicosis and complicated silicosis. Uncomplicated or simple silicosis can be distinguished by three kinds of tissue reactions - chronic, accelerated and diffuse. In each case, respectively, exposure to silica varies from moderate - over a period of twenty years - to accelerated - which shows a concentrated increase over a period of five to fifteen years - to diffuse - which shows a heavy concentration over a period of less than five years. It is important to realise that these tissue conditions are produced by the presence of silica alone, a theory proved by the South African doctors F.J. Simson and A.G. Strachan, who started their investigations in 1926.

In the early stages of the disease only isolated nodules are formed and the disease may never progress beyond this stage if the individual is removed from dust exposure. Continuous exposure to silica, however, causes their increase in number. In South Africa x-ray examinations enable the diagnoses of slight, moderate and marked silicosis according to whether there are fewer than fifteen, fifteen to thirty, and more than thirty islets respectively. In addition, other pulmonary disability, such as chronic obstructive airways disease (including chronic bronchitis and emphysema) are also taken into account in making these diagnostic assessments and awarding compensation. It is interesting to observe that disablement of the latter kind was not considered in the diagnosis or compensation of silicosis at the first International Silicosis Conference held at Johannesburg in August 1930.

Chronic fibrosis is usually encountered in occupations where the proportion of silica in the respirable dust is less than 50 per cent. Conditions produced by such forms of silicosis may cripple but need not necessarily be fatal. However if the proportions of silica in the dust increase or exposure is heightened, as in accelerated and diffuse forms, the disease itself may rapidly change its course. Nodules may proliferate and enlarge encroaching upon the lung parenchyma to form a continuous mass of 'stony' fibrous tissue which is composed of a multitude of compressed nodules. These usually appear bilaterally, often close to the pleura, and invariably in the upper lobes. When gas exchange becomes difficult and there is insufficient ventilation, or right heart strain supervenes, the condition may become fatal. Much of our present day knowledge on accelerated silicosis has been obtained from studies of sandblasters.

Chronic silicosis may however turn into or become complicated by progressive massive fibrosis, the cause or causes of which have not yet been fully explained. This type of lesion is encountered in other forms of dust diseases including coal workers pneumoconiosis and in the lungs of haematite miners. Webster suggests that exposure to one or a combination of the following factors

* New paragraph.

may lead to its development in a person who already has silico-
silicosis. They are massive retention of siliac dust in the
lungs, bronchial obstruction with partial lung collapse, and
superadded tuberculosis. These complications are more common
when silicosis is accelerated or diffuse. The nodules of
progressive massive fibrosis, although slow in evolution, vary
in extent and number, but are usually more than two and a half
centimetres in diameter. As they increase in size, nodulation
becomes less marked and regular and may even disappear in the
fibrous masses matted together and containing obliterated blood
vessels and bronchi. Obliteration of the blood supply can cause
death and alteration of the tissue, namely necrosis. The result
is that necrotic material often liquefies and is evacuated
through the bronchus leaving behind a ragged cavity.

In advanced cases the lung structures may become distorted. The
majority of these massive fibrotic structures occur in the upper
lobes (where dust clearance is poorer) causing them to contract;
but they may also be found in the medial and lower lobes and have
been observed to cross the interlobular fissures. Progressive
massive fibrosis is also related to emphysematous changes. Such
changes may be linked to bronchial failure, partial lung collapse
and obstructive pulmonary disablement, although there is a great
deal of controversy about the latter complication.

Narrowing of the lung airways is called the obstructive syndrome
and is typical of chronic bronchitis and emphysema. In this
connection mention must be made of the vital capacity of the
lungs. Vital capacity is obtained by asking the patient to
breathe in maximally and to expire slowly and completely. The
tidal volume so obtained is the sum of the inspired and expired
volumes. Forced vital capacity is obtained by asking the
patient to inhale maximally and then to exhale as rapidly and
strongly as possible, such exhaled measurements then being
recorded. Both the vital capacity and the forced expiratory
volume indicate the ventilatory capacity or sufficiency of the
individual. Pulmonary obstruction will be indicated by a low
value for the forced expiratory volume expressed as a percentage
of the vital capacity. In the first two decades of the 20th
century silicosis was diagnosed by measuring chest expansion,
but confusion existed as to the exact role played by emphysema
in the course of the disease. There are many forms of emphysema;
but the disease is only seriously disabling when an obstructive
element is present concomitant with obstruction in the bronchial
tree and alveolar region.

Irritants such as dust, exacerbated by atmospheric pollution
and tobacco smoking, cause the bronchi and bronchioles to
become narrowed; and there is damage to the elastic membrane.
On inhalation the increased pressure of air causes the respiratory
bronchioles to dilate. Air entry is allowed, but the lungs have
great difficulty in expelling air. This in turn causes the
dilation and destruction of the third order of respiratory
bronchioles, alveolar ducts and alveoli. When dyspnoea, that
is difficulty in breathing, is present, as in silicotic subjects,
the expiratory bronchial collapse is intensified and patients
must literally 'squeeze' the air from their chests. The
patient exerts pressure on the chest in an effort to force air
through the flattened air passages, but merely causes them to
flatten further, and degeneration extends from the unsupported
bronchioles to some of the cartilagenous rings of the trachea
and bronchi.

Emphysematous change does not necessarily mean that all portions of the lung are affected similarly, and in obstructive emphysema a wide regional variation of pathological changes may be found many of which tend to become progressively worse especially in the presence of massive portions of lung collapse. In terminal cases there is extensive fibrosis and there may be severe restriction which prevents gas change as well as obstruction. Superimposed bronchial infection is not fatal in the healthy lungs of a normal person; but is an additional factor in the development of right heart failure in a patient with silicosis.

The superimposition of tuberculosis on silicosis is a complex problem which has been a puzzle for many years. The cause of this association of silicosis with tuberculosis is not yet clearly understood but its existence has been shown repeatedly by various epidemiological, pathological, statistical and in vivo and in vitro experimental studies.

It was only in the second half of the 19th century that the causative bacillus, *Mycobacterium tuberculosis*, was discovered by Robert Koch. The most common site of tuberculosis is the lungs but it may spread to or affect any organ or tissue of the body. The organism is usually spread from one person to the next by the inhalation of organisms coughed or sneezed into the atmosphere; but transmission may also be by stools or urine. In addition, the bacilli are resistant and may remain viable for months in dust or on articles of daily use. Also a person who has had tuberculosis or has been exposed to the tubercule bacillus is at risk of a flare up of his old tuberculosis when he is exposed to silica dust for any length of time.

The portal of entry of the bacillus is usually the respiratory tract. The inhaled bacilli produce lesions of a different kind to silicotic nodules and tuberculosis and silicosis may be present simultaneously in the lung as separate lesions. However when tuberculosis becomes associated with silicosis - and this can occur in the simple or complicated forms of the latter disease - it can be extremely difficult to identify the tuberculo-
tic element. Even the typical silicotic nodules are not recognisable in progressive massive fibrosis. The nodules are no longer well defined. They show evidence of necrosis and cannot be sharply distinguished from the surrounding inflammatory tissue. On the other hand the characteristic pathological features of tuberculosis are modified or absent in the lesions of progressive massive fibrosis. Although the term silico-tuberculosis is coined to apply to this condition, complicated silicosis is considered to be a more acceptable term today. In this context it is important to destroy the popular concept that pulmonary tuberculosis is the terminal feature of silicosis. Respiratory failure is the most common result of complicated silicosis as the whole pulmonary arterial system is often extensively destroyed. The association of tuberculosis with silicosis nevertheless still remains a controversial question, and as R.A. Steele suggests, it remains 'too frequent to be coincidental'.

It is very important to diagnose silicosis in its earliest stages, because unless the worker is removed from exposure to free silica the disease will pursue its relentless course (in its simple or complicated forms) leading to total pulmonary incapacity and/or right heart failure - but not to cancer of the lungs. There is no specific treatment for the disease; and it

is doubtful whether the formation of nodules is reversible. The removal from exposure to free silica does not necessarily halt the formation of nodules either. There are many cases in which pronounced nodules have been radiologically observed some years after patients had left their dusty occupations with only a slight degree of nodulation.

The difficulty with the clinical features of silicosis is that the principal symptoms develop late, after the disease is already well advanced. In chronic silicosis the symptoms are usually mild at the outset; but in the advanced stages and in complicated silicosis, characterised by progressive massive fibrosis, the major clinical manifestations are more severe. They include abnormal breathlessness (dyspnoea) on effort, constriction of the bronchioles which leads to wheezing (bronchospasm), shortness of breath on lying flat, and cough with or without sputum. The latter condition may be related to a history of tobacco smoking from youth. Other symptoms may include fatigue, indigestion, chest pains, diminished chest expansion and lessened fitness for work.

All these symptoms will probably be more severe if the disease is complicated by tuberculosis. Also, the presence of chest pains, night sweats, coughing up blood (haemoptysis) and weight loss will probably be indicative of associated tuberculosis. Despite this however, there may be no radiological evidence of tuberculosis, while significant radiological lesions may produce negative tuberculosis results in sputum, culture and biological tests. Also in cases of positive sputum (that is proven tuberculosis) treatment for tuberculosis may be ineffective in the presence of silicosis. Tuberculosis seems to be declining in cases of simple chronic silicosis - and this is true of the South African gold mines - but it still remains high in the accelerated and diffuse forms.

It is generally accepted today that chronic bronchitis (clinically defined by chronic cough with expectoration) and emphysema are not complications of silicosis. There is however a considerable controversy as to whether they constitute a specific occupational disease - chronic obstructive airways disease - related to dust exposure. Bronchiectasis is extremely rare in simple silicosis but occurs frequently in complicated silicosis accompanied by lung collapse. Bronchiectasis is an inflammatory condition causing the dilation of the bronchi or bronchioles, and is characterised by the coughing up of a great deal of purulent sputum. In a number of instances, the 'Old Rand Miners' were observed to cough up calcified bodies together with blood. There is little evidence to prove that other infections, such as pulmonary pneumonia, are complications of silicosis unless they supervene as terminal features.

The clinical symptoms are not definitive in establishing a diagnosis of silicosis. Similarly the early physical examination and assessment including measurement of chest expansion, chest palpitation and percussion and auscultation - the use of the stethoscope to detect changes in breath sounds - were, and are, equally valueless. Also negative lung-function tests do not exclude the presence of the disease. As a definitive diagnosis of silicosis can only be given by histological examination (pre- or post-mortem) radiological evidence and epidemiological data (including family and occupational histories) play a very important role in the diagnosis of the disease. It was only after 1945 that biopsies could be used for definitive pathological diagnosis. Before then definitive and accurate diagnosis could only be obtained on post-mortem pathology, although radiology was a helpful aid.

Radiology has its limitations and too much reliance must not be placed on it. For instance, the detection of nodulation may prove difficult in the presence of changed pulmonary patterns especially in the case of progressive massive fibrosis. In borderline cases other forms of pneumoconiosis may mimic silicosis. Much depends on the skill and experience of the radiologist. Sometimes early pulmonary structural changes may escape notice; while on other occasions radiological evidence may not be confirmed by pathology. Where occupational history has played an important part in establishing diagnosis, values obtained from periodic dust sampling may be important in indicating the attendant risks in a given situation.

Compensation to the miners with silicosis is a controversial question. Considerable disagreement exists amongst medical authorities as to what precisely should be compensated. In South Africa, in contrast to many other countries, legislation for the award of compensation for pneumoconiosis and for silicosis has from its earliest inception, in 1911, never been related merely to loss of money wages. The presence of the disease itself and the degree of disability have always been important criteria in awarding compensation, in spite of the difficulties in determining and measuring disability. The most recent legislation, that of 1962, allows for payment of compensation solely on the criterion of disability, the basis for which is the impairment of cardio-respiratory functions. Compensation also is defined to include the diagnosis of any radiological symptoms of nodulation including prenodular changes. This latter point is in itself controversial because in the prenodular stage it is very difficult to distinguish the deviant from the normal lung pattern of a particular individual. Compensation is also awarded for tuberculosis even though sputum tests may not be positive and radiological evidence may be absent.

Finally the degree of disability is also evaluated. If there is a lowering of working capacity by 20 per cent, on lung function tests, compensation will be awarded. Although chronic obstructive airways disease is closely related to smoking, in South Africa it is accepted that if service of a worker is of sufficient duration that some of the disability might have been caused by dust, he will be duly compensated.

In this paper the emphasis has been placed on understanding silicosis and its manifestations in a wider context than that of the Transvaal gold mines. There are several reasons for doing so. First, it is important to realise that, when the hazards of silicosis on the Witwatersrand were recognised, doctors in the Transvaal, the majority of whom were of British origin, had to draw on their experience and understanding of the problems from Britain and Europe, as well as British overseas territories, particularly Australia and Canada, where numerous ores were mined.

Second, doctors practising in the Transvaal were to play a major role in bringing about an awareness and understanding of the problems of silicosis to the medical world at large. The first international conference on silicosis, organised by the International Labour Office at Geneva, was held in Johannesburg in August 1930. Here representatives of many nations converged to exchange their views with South African experts who had accomplished so much in the field of silicosis. At this

conference South African medical and mining experts brought a wealth of experience to bear on the problem. Of the twenty-six papers read at the conference, nineteen were presented by South Africans who had examined many of the aspects of the problem encountered in the Transvaal.

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