

Silicosis and Asbestosis

BY VARIOUS AUTHORS

EDITED BY

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PREFACE

THE industrial age has complicated the scheme of life in many ways, some of them directly affecting the public health; among these are the diseases due to the inhalation of certain kinds of atmospheric dusts arising directly from industrial operations. Ill effects due to dust contamination of the atmosphere have been recognized from earliest times.

This book has for its purpose the presentation of the medical and public health aspects of silicosis and asbestosis, two definitely industrial dust diseases. Silicosis looms larger in the picture than asbestosis because the former is much more widely spread, affects a much greater number of the industrial population, has been recognized and studied longer and, consequently, more is known about it.

The form of the generic term pneumoconiosis, rather than pneumonoconiosis or pneumonokoniosis, has been adhered to throughout the book. The International Labour Office and both British and American editors have adopted it as preferable.

The editor wishes to express special thanks to Miss C. M. Bresnan for reading proof and for making the index and to acknowledge with gratitude the help received from many sources and from many individuals. To them and particularly those working in the field of industrial hygiene, this book is dedicated.

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INTRODUCTION

PATHOLOGICAL conditions of the lungs, due to the inhalation of excessive quantities of dust in certain occupations, have been known to exist for many centuries. It is also true that the frequency of infection, particularly pulmonary tuberculosis, as a complication has been realized for a long time. Pneumoconiosis is a comprehensive term, designating the effects upon the lungs of the inhalation of excessive quantities of dust, manifested by structural changes in the lung tissue and entirely distinct from the action of poisonous dust, such as lead or mercury, in which case the lungs act merely as the point of entrance into the body without definite local influence. At first it was associated chiefly with mining but with the advancement of the industrial age, dusty industries multiplied.

It is customary to speak of the dust inhalation effect upon the lung tissue by the name of the chief constituent of the dust breathed, as silicosis when silicon dioxide (SiO_2) is the principal ingredient and asbestosis when asbestos is the offending agent. Silica dust, when the particles are sufficiently small, has the peculiar property of inducing a fibrosis of the lung tissue. This effect is characteristic, as is the resulting roentgenologic appearance. In asbestosis, the pathological process is quite different as is the roentgenologic appearance. These matters are thoroughly dealt with in the text. There is still, however, much to be learned with respect to both conditions. We know the results of the inhalation of these dusts but we are not sure how they are produced.

There are two ways in which the pulmonary infections, tuberculosis and pneumonia, may affect or complicate pneumoconiosis. First, there may be infection before the exposure

to dust. It is an undecided question whether such infection can so change the pulmonary structure that the later dust effect is more pronounced than it would otherwise be. There seems to be reason to believe that such an enhancement is to be expected, especially if the previous infection were tuberculosis, but this must be left to the histopathologist for final proof. Secondly, do such infections upon an already dusted lung become more severe or aggravate the dust effect or otherwise change its character? There is even more evidence that any of these effects may result but here again the histopathologist must be called upon to tell us the exact truth. The first intimation that occurred to us that the character of a silicotic lesion could be so changed was when we were searching for an explanation as to why certain individuals with silicosis showed massive consolidations of fibrosis in the lungs and others in the same occupation and apparently exposed to the same amount of dust of the same variety showed a more evenly diffused nodular fibrotic process. It was a great satisfaction to hear a well known pathologist say that a comparatively recent infection like lobar pneumonia might be the answer but that at present there was no definite proof of such theory being correct.

It was gratifying to learn that most of us were thinking along similar lines. That most cases of silicosis die of an intercurrent infection, especially tuberculosis and possibly pneumonia, is evidence that silicosis may exert some effect upon the progress of some pulmonary infections. It has not been proven as yet that the same is true of asbestosis.

A comprehensive knowledge of the effects upon the lungs of excessive inhalations of dust is not to be quickly gained. The combined efforts of the histologist, pathologist, clinician, roentgenologist, chemist, physicist and other technological experts are necessary. The legal profession deserves a certain share of commendation for stimulating the study of

occupational dust diseases and has made a thorough knowledge of pneumoconiosis a necessity among many of the medical men. Were it not for the demand for exactness from outside the medical profession, it is difficult to say how soon curiosity or medical research would have achieved our present accuracy of knowledge with respect to the etiology, symptoms, and pathology of pulmonary dust diseases and their modification by certain infections.

A large number of us have, no doubt, taken up the study of pneumoconiosis from the standpoint of interest. Following the experience with silicosis in South Africa, starting several decades ago, text books for the medical student simply mentioned it in a more or less cursory manner as an uncommon condition. We are sure of the necessity of many angles of approach to the subject. The first comprehensive report of silicosis in the United States was made about twenty years ago by one of the authors. It described what was occurring among miners in the south west. Almost simultaneously and without any knowledge of this report, it occurred to a small group of medical men in the east that the best way to study the differences in the appearances of trunk shadows in the roentgenograms of the lungs might be to study the chests of persons engaged in various dusty occupations. The main point learned in this investigation was that organic dusts do not induce lung changes but it further stimulated the study of the effects of inorganic dusts, particularly silica, and later, asbestos. This study has continued up to the present time.

Some of the authors of this book were among the pioneers in silicosis and asbestosis ; they have blazed the trail for future investigations. It can be truthfully stated that those who studied these occupational diseases in North America and the British Empire have always had the good of the affected workers in mind. Their researches have saved life and have made many occupations safer for the worker.

HENRY K. PANCOAST, M.D.

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SILICOSIS AND ASBESTOSIS

f.D.

I. HISTORY OF SILICOSIS AND ASBESTOSIS

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1. HISTORY OF SILICOSIS

ALTHOUGH to the general public silicosis is a disease of recent origin, the history of dust diseases reaches far into the past, and the first recognition of a definite relation between exposure to dust and pulmonary infection is lost in the mists of antiquity.

As Collis¹ states, 'In historic times references are to be found in the works of ancient writers who, however, seldom distinguish between the various forms of respiratory disease, but rather refer to a general connection between lung affections and dust inhalation.'

Probably the earliest recorded reference to the harmfulness of dust exposure is Pliny's description of the devices used by refiners to prevent inhalation of the 'fatal dust.'² The symptoms of a metal digger, as noted by Hippocrates, who lived in the fifth century B.C., are comparable to those observed among present-day miners suffering from the effects of dust inhalation.³ Other early writers discussed dust phthisis, and it is believed they had in mind the type of disease described by Celsus,⁴ a medical writer of the first century, who stated :

'By far the most terrible form of emaciation is that which the Greeks call phthisis. It spreads to the lungs. On top of this ulceration occurs and a slow fever which at times disappears and at other times reappears.'

In 'De Re Metallica,' published in 1556, Agricola described the perils of mining and the 'pestilential air' breathed by miners. He also relates how 'the constant dust enters the blood and lungs, producing that difficulty of breathing the Greeks call asthma. When the dust is corrosive it ulcerates the lungs and produces consumption.'⁵

Lung diseases of miners, smelters, grain workers, etc., were discussed and described by a number of early writers. Among these, Paracelsus, a Swiss alchemist and physician, was the first to list the occupational diseases of miners and smelters. His book on industrial medicine was published in 1567.⁶ It is said that Dimerbroek, in 1649, made the first section of a stone-cutter's lung which revealed 'lung vesicles completely clogged with fine dust.'⁷ Ramazzini, in his book published in 1700, noted the effect of dust on the respiratory organs, and reported numerous cases of fatal dust disease. He obtained his information by personal inspection of the trades he discussed, and his writings display a recognition of the relationship between dust inhalation and consumption.⁸ According to Collis, Thomas Benson, of Newcastle-under-Lyme, was granted a patent for grinding flints by a wet method. Previously, flints were pounded dry, the process proving 'very destructive to mankind insomuch that any person, ever so healthful and strong, working in that business, cannot possibly survive over two years, occasioned by the dust sucked into his body by the air he breathes.'¹ A number of special inquiries into the prevalence of dust-phthisis were made, for example, by Professor Allison⁹ and Hugh Miller,¹⁰ who wrote of the progress of such disease among stone-cutters.

Thackrah,¹¹ in 1831, began a study of the effect of the dusty trades on British workmen's longevity, and contributed valuable information, including the fact that not all dusts shorten the lives of the workers exposed, as evidenced by the experience in various mines. He noted that, while sandstone workers generally died before forty, there seemed to be little or no unusual incidence of lung diseases in brick and limestone workers.

Dr. Greenhow,¹² an instructor in public health, became the first state medical factory inspector in England and made personal investigations of health conditions in industries throughout the country. He collected lung specimens of workers, who had died from dust diseases, for pathological study and he was perhaps the first to use polarized light to identify microscopically the tiny particles of silica in the lungs of a metal grinder. As a result of his work, the first Royal Commission was appointed in 1861 to make a thorough investigation of health conditions in mines.

In 1862, Dr. Peacock,¹³ who had interested himself in the lung diseases of buhrstone cutters and who had detected particles of the stone in their lungs, was made a member of the Commission. In his report on the medical aspects of mining, he distinguished between the conditions of the lungs known as 'miner's phthisis' and the ordinary type of tuberculosis, noting that the miner's disease occurred in otherwise healthy men with healthy families and that it appeared in them at an older age and was a more chronic type than the usual infective tuberculosis.

Although medical opinion at this period attributed the disease among miners more to bad hygienic conditions than to the dust to which they were exposed, some of the Scottish and English physicians noticed that even in mines where miner's phthisis was prevalent, drillers of rock suffered from more rapid onset of the disease than other miners. This, to-

gether with other similar data, gave rise to the supposition that the character of the rock influenced the occurrence of the disease.¹⁴

The effects of various dusts on the lungs were reported by other writers, the names given to the resulting diseases being descriptive of the dusts causing them. Zenker¹⁵ provided the anatomical and Kussmaul,¹⁵ the chemical proof of the deposit of inhaled dust, especially siliceous dust in the lungs. The name 'pneumonokoniosis' was devised by Zenker, who described in detail the anatomical picture of the dusty lung.

Visconti, as reported by Rovida,¹⁶ in 1870, is accredited with being the first to use the term 'silicosis' to denote the pathological condition of the lungs resulting from inhalation of silica.

Commencing in the latter part of the nineteenth century, great interest in the health of miners was being shown throughout the world. A summary of development in some of the principal countries follows.

SOUTH AFRICA

Too much credit cannot be given to the South African authorities and investigators for their contributions to the whole subject of pulmonary dust diseases. The publications and reports of the Miner's Phthisis Prevention Committee, the Miner's Phthisis Medical Bureau, and the South African Institute for Medical Research have been an inspiration for officials and research workers all over the world. Their influence on both the clinical and compensation aspects of silicosis has been enormous.

Gold mining started in South Africa on the Witwatersrand in 1886. It was not long before the introduction of machine drilling and the depths of the mines began to produce health problems. The quartz content of the ore bearing rock was high, containing as much as 80 per cent to 90 per cent free

silica. In 1902, the Government mining engineer noted that miner's phthisis seemed to be prevalent among rock drillers and a Commission of Enquiry was appointed. Physical examinations were made of 3,000 men, the roentgen ray being used in South Africa for the first time in 300 cases.

Compensation was inaugurated in 1912 and in 1916, the Miner's Phthisis Prevention Committee issued a comprehensive report. Pre-employment and periodic physical examinations were initiated by the Medical Bureau and engineering methods to improve ventilation and allay dust by the use of water were instituted. The estimation of the amount of silica dust in various working places underground was developed, Sir Robert Kotze having devised an instrument for this purpose, the Kotze Konimeter. A threshold limit of 300 particles per cubic centimeter (about 8,500,000 particles per cubic foot) was set as a reasonable (at that time) standard of safe practice.

The Miner's Phthisis Medical Bureau defined diagnostic standards and regulations for compensation. The Bureau established the classification of silicosis into four stages — ante-primary, primary, secondary, and silicosis with tuberculosis. Watkins-Pitchford and the other medical members of the Bureau — Watt, Irvine, and Stewart — made notable contributions to clinical knowledge. The importance of tuberculous infection as an accelerator of lung fibrosis, the marked susceptibility of silicotics to tuberculosis, the progressive nature of silicosis, were among the important aspects of silicosis which this group described. From the Institute for Medical Research came the important pathological studies of Mavrogordato and Simson. No one interested in the medical, economic, or engineering phases of silicosis can afford to overlook the publications that have come from South Africa for the past twenty years.

GREAT BRITAIN

In 1902, fifty years after Peacock's investigations, a governmental commission was appointed to study the persisting high mortality among the tin miners of Cornwall. Dr. Haldane, one of the members of this commission, definitely ascribed the disease of these miners to rock dust and stated that the dust itself was what caused the primary injury to the lungs.¹⁷

Investigations among slate workers showed that slate dust exposure was related to phthisis, particularly in Wales, where the slate is high in quartz.¹⁸

From this time on, frequent and intensive investigations of many of the dusty trades in England were made, and numerous reports containing valuable information and suggestions were issued by the British Home Office. Among the industries studied were slate, refractories, sandstone, potteries, and coal mining.

Dr. Collis, in 1915, in the Milroy Lectures, reviewed the status of pneumoconiosis in England at that time. He clearly expressed the theory that 'free' or crystalline silica is the causative agent in nearly all dusts that produce serious lung injury and susceptibility to tuberculosis.¹

In 1917, an investigation was made of the refractories industry, where a species of sandstone shale with a high free silica content is used in making bricks and in 1919, the first scheme of compensation for silicosis was set up for this industry. Dr. Heffernan has given an account of the problems connected with this industry.¹⁹

The situation in the English pottery industry had existed for many years, Wedgewood having found that his workers employed in grinding flint lived only a couple of years. Several Royal Commissions were appointed to investigate the potteries industry; one by Sutherland and Bryson for-

mulated a scheme for including the potteries in compensation laws.²⁰ One report of the British Factory Inspectors states that regulations now enforced for controlling dust in this industry has been so effective that no process in English potteries is now so hazardous as to produce even an early stage of silicosis in less than ten years.

The granite industry was also studied repeatedly. In 1930, out of about 500 workers examined, 17 per cent showed evidence by roentgen-ray of silicosis, mostly in the early stages. The free silica content of granite is lower than that of sandstone, usually not over 30 per cent, but the fact that pneumatic tools were generally used was thought to cause more dust than hand methods.²¹

A very comprehensive report on the metal grinding and casting cleaning industry was issued by two of the Factory Inspectors in 1923. It was shown that the occurrence of silicosis, many cases being complicated with tuberculosis, was present almost entirely among those who worked on sandstone grinding wheels. Silicosis was more frequent and more advanced in the wet grinders examined than in the dry grinders. Sand-blasting was rated as potentially a very hazardous process, unless enclosed, or the workers protected. Counts of the dust particles in the air were made with an apparatus developed in 1922, called the Owens' jet dust counter. Petrographic determinations were made and particle size measurements estimated for the dust present in the various processes. Detailed requirements were drawn up for the improvement of working conditions. This report helped to establish the fact that metal particles or hard mineral particles from corundum or emery were not the source of true silicosis; that only work on sandstone wheels or on sand cleaning or with other exposure to free silica dust is capable of producing the disease.²²

*Collis and Yule, in 1933, endeavored to determine the ef-

fects of exposure to silica dust on mortality by comparing the death rates for a group of occupations having exposure to dust containing free silica with a group having equal exposure to dusts not containing free silica, working conditions being similar. Comparison was also made with the rates for all occupied and retired males, with correction for age distribution. The authors concluded from this analysis that silica dust tends to be a poison to the whole body and that, although it exerts its important influence on the respiratory tract, especially with respect to tuberculosis, it also impairs the circulatory, nervous, and digestive systems and the glands, kidneys, and liver.²³

The chemical aspects of the action of silica in the tissues have been painstakingly studied by Gye, Purdy, and Kettle. Kettle has concluded from his experiments that silica, after it has entered the body, dissolves in a colloidal form in the weak alkalis of the lung fluid and is very gradually absorbed, the soluble portion then acting as a tissue poison. He thinks that through its toxic action it paralyzes the phagocytes so that they lose their phagocytic ability and also their power to deal with the tubercle bacillus, thus leaving the lung unprotected.²⁴ Kettle, Gye, Purdy, and S.Lyle Cummins have all proved by experiment that far from being 'poisoned' by silica, the tubercle bacillus is stimulated by silica.²⁵

Extensive studies have been made on the health of coal miners. Dr. Middleton, in a five year study of silicosis deaths reported in British industries, showed that during the period from June 1, 1931 to December 31, 1935, there were 147 certified deaths from silicosis and silicosis with tuberculosis and 511 total disablement cases among 139,228 employed coal miners in South Wales and Monmouth. In all the rest of England and Scotland, there were only 22 deaths and 70 total disablement certificates for silicosis in coal miners, in a total of over 600,000 employed.²⁶ Physicians practicing in the

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South Wales coal fields believe the silicosis observed there is of the 'infective' type; 'infective' is Dr. Irvine's term for the type of extensive fibrosis found in silicotic lungs which is suspected of being produced by the aid of the tubercle bacillus, but in which the bacillus cannot be demonstrated.¹⁴ Investigations in the iron mining industry disclosed a rather high silica content in hematite miners' lungs at autopsy and Stewart and Faulds felt justified in naming this lung condition 'sidero-silicosis.'

In 1933, W.R. Jones brought forward the theory that mineral sericite (a hydrous silicate of aluminum and potassium) was the chief factor in the development of silicosis.²⁸ However, this has not been substantiated. Silicosis has been demonstrated in experimental animals and in human cases in the absence of sericite and where pure quartz was the only causative factor. It is interesting to note that many attempts have been made to find an 'antidote' dust to neutralize or delay the harmful action of silica. Carleton,²⁹ in experiments with combined flint and coal dust on guinea pigs, found that coal dust appeared to cause enough initial immediate irritation to promote elimination of some, but not all, of the flint dust inhaled. No definite conclusions have as yet been reached in this work which is still being conducted.

Compensation for workers disabled as a result of silicosis or silicosis and tuberculosis has been provided for in England by amendment to the compensation law. From 1925 to date, five schemes have been put into operation dealing with silicosis in various industries, including the refractories, metal grinding, sandstone, tin mining, coal mining, potteries, granite working, and others. Processes not involving exposure to silica dust are exempted, in some cases, the limits of the silica content being specified for the purpose of the law. The most important feature of these schemes is the provision for medical certification of physical fitness for work in such exposure,

for medical certification of disability with limited compensation for non-disabling stages of uncomplicated silicosis, and for post-mortem examination in death claim cases.

GERMANY

In Germany, Paracelsus was first to deal with injury to the lungs of miners caused by inhalation of dust and to designate such injury as an occupational disease. In the nineteenth century, German pathologists and clinicians made considerable progress in regard to research connected with diseases caused by dust. Arnold succeeded in producing experimental dust fibrosis of the lung and in distinguishing clearly between that and tuberculosis. Eulenberg definitely affirmed that silicic acid causes serious fibrotic disease of the lungs. The connection between inhalation of dust with quartz content and tuberculosis has been shown statistically by various writers: Oldendorff, Sommerfeld, Moritz and Röpke and others showed the high mortality rate from tuberculosis among metal grinders; Lewin, Hirt, Wilbrandt, and others drew attention to similar conditions among pottery workers.

Baumler described the symptoms of silicosis and stressed the cirrhotic character of the accompanying tuberculosis. Staub-Otiker described in detail the roentgen-ray appearance of the silicotic lung as found in metal-grinders and also distinguished the various stages of the disease. Stähelin, Ickert, Jötten, Arnoldi, Lehmann-Engel-Wenzel have all given comprehensive expositions of this subject. Koelsch and Kaestle designated the appearance of fine mottling on roentgenogram as the first stage of silicosis, associated with slight dyspnea after effort. While it is admitted that very often tuberculosis is associated with dust fibrosis, the majority of German investigators are of the opinion that fibrosis due to dust is a disease in itself and to be distinguished from tuberculosis

and that dust inhalation, as such, may lead to extensive pulmonary fibrosis without associated tuberculous infection. Schmorl, Staub-Otiker, Arai, Koopmann, Böhme, and Schridde describe such cases. Merkel has concluded from his anatomical findings that simultaneous inhalation of dust and tubercle bacilli occurs and that these exert a double action in the production of the majority of dust fibrosis. A great deal of experimental work has been done in Germany regarding silicosis by Lehmann, Gross, Jötten, and others.

In 1929, 'severe pneumoconiosis' (silicosis) was included in the list of occupational diseases subject to compulsory compensation. In the case of severe dust fibrosis combined with tuberculosis of the lungs, tuberculosis is regarded as pneumoconiosis (silicosis) for the purposes of compensation.

Up to the beginning of the present century, all changes of the pulmonary tissue caused by different kinds of dust were considered as fairly similar. However, the observations of the last few decades, particularly the thorough investigations effected in the last twenty years, have fundamentally altered the former theories. Today it is recognized that the various types of dust must be judged in a different manner and that the decisive factor regarding effect is not the morphological-physical activity of the various types but rather the content of free crystallized silicic acid (quartz) in the dust. Koelsch began some years ago to investigate conditions in dusty industries and in the last few years, over 1,200 workers in various branches of industry involving exposure to mineral dust were examined clinically and roentgenologically. It was found that the most dangerous occupations in regard to pneumoconiosis, as related to dust exposure, were granite and sandstone working. The statistical morbidity and mortality returns, as well as the clinical-roentgenological examinations,

show clearly the fundamental principle that the greater the quantity of free silicic acid in the dust, the more dangerous it is for the lungs.¹⁵

AUSTRALIA

The rapid increase in Australia's population in the latter half of the last century was due in large part to the discovery of rich alluvial gold fields in several parts of the Continent. This stimulated prospecting and brought about the discovery of other metalliferous deposits. A large portion of the migrants came from mining districts in England and other countries.

Silicosis has been demonstrated in many of the mining fields and a high death rate from pulmonary diseases, including tuberculosis, has been evidenced in connection with the industry.¹⁵

In 1902, while a system of sewers was being dug in Sydney, Australia, a Sewer Works Ventilation Board was appointed to inquire into the working conditions and to recommend means of improvement to render the work less hazardous. The board found that miners employed in this type of work suffered from a disease known as 'sewer disease' but which was strongly suspected of being due to dust. It was the board's opinion that although fumes from explosives, poor ventilation, etc., were contributory to the high mortality, the sole cause of the disease was probably the dust from hammering, drilling, and use of the pickax.

The Australian government has made many investigations of the mining industry throughout the country and vigorous efforts have been made to control unhygienic conditions. The chief metal mining sections in which health conditions have been surveyed are: Bendigo, Victoria; Broken Hill, New South Wales; Kalgoorlie, Kimberley, and Coolgardie in Western Australia.¹⁴

In 1912, Armstrong investigated a reported epidemic of pneumonia at Broken Hill and found the death rate from pneumonia among underground miners in that locality during 1910-2 was nearly 4 times as great as that for all males in New South Wales. In 1914, the report of a Royal Commission investigating the mining industry at Broken Hill stated that although the prime cause of pneumonia was sudden changes in temperature, dust inhalation in any form would predispose to disease.³⁰

In 1919, the Technical Commission constituted to examine the miners at Broken Hill used the roentgen-ray, for the first time in Australia, as a means of diagnosing silicosis as an occupational disease. The Commission made an elaborate report in 1921 and set up standards for diagnosing the different stages of silicosis.³¹

In 1924, a very complete investigation of the sewer system situation was made by Dr. Charles Badham, Industrial Medical Officer of the New South Wales Department of Health. Dust counts were taken and it was found possible to set a standard for permissible dust concentrations of 200 particles per cubic centimeter (approximately 5,664,000 particles per cubic foot).

A special scheme for the compensation of silicosis for Sydney sandstone workers was put through in 1927 and the whole field was checked again; surveys were also made among sandstone masons and quarriers in the neighborhood. These were found to be exposed to much the same hazard as the tunnelers. Open sheds and wet methods were recommended for controlling the dust hazard.¹⁴

Since 1926, the mine employees in Western Australia have been examined annually by the Commonwealth Department of Health. Of 2,290 men previously classed as normal, 1.3 per cent were found on re-examination in 1927 to be suffering from uncomplicated silicosis and 0.5 per cent had tubercu-

losis only ; of 491 diagnosed as silicotic, 86 (17.5 per cent) had silicosis with tuberculosis. In 1929, of 2,293 normals re-examined, 100 were diagnosed as silicotic. Silicosis was not found in any case under forty years of age or with less than five years of underground work.³⁰

ITALY

Italian research workers have furnished interesting contributions to the knowledge of respiratory diseases resulting from the inhalation of dust. While experimental work was done by Biondi, Tommasi-Crudeli, Feliziana, and others, the report of Devoto and Cesa-Bianchi in 1911 must be considered especially notable. In their experiments with guinea pigs, they found that animals subjected to the inhalation of very fine limestone dust are less susceptible to tuberculosis than those which have inhaled silica dust.¹⁵

Among other workers who have studied the occurrence of pneumoconiosis in various Italian industries are Pesenti, Rota, and Finzi, who reported on health conditions of workers in lime, cement, and plaster ; Frongia, who noted that the cause of death among Sardinian miners was most frequently an acute infection (pneumonia) ; Bianchi, who made careful clinical and radiological examination of workers employed as sculptors, hewers, modelers, and workers in grinding rooms. In regard to the importance of silica dusts as the cause of pneumoconiosis, a report by Giglioli is of real importance. Pieraccini had already remarked that among the workers in the Cornacchino mine, there occurred a high incidence of broncho-pulmonary affections and pulmonary tuberculosis ; a later inquiry by Puccinelli and Ginanneschi confirmed this fact. Giglioli found that in many of the miners at Cornacchino, and in almost all who worked where the air is rich in silica dust, there occurred, after two to three years of work, broncho-pulmonary affections, with coughing,

breathlessness, emphysema, and in general, after a more or less lengthy period, all the signs of pulmonary tuberculosis. There is almost always a well-marked, pre-tuberculous period, which may evolve slowly without tuberculous complications corresponding to so-called miner's phthisis. More recently, Mazzi, in 1913, on the basis of animal experiments and clinical observations, has come to the conclusion that silicon (in the special form of silicates) causes chronic intoxication of the system and acts especially on the blood in exercising a general anemia producing action.¹⁵

CANADA

In 1875, Sir William Osler, in one of his lectures, demonstrated two specimens of miners' dusted lungs. His description of the pathology of this disease compares favorably with modern descriptions of silicosis. Pneumoconiosis or silicosis or miner's phthisis has been scheduled as a compensable occupational disease in quarrying, stoneworking, metal grinding or polishing, and mining for a number of years in Ontario, Alberta, and Saskatchewan.

Silicosis has been studied from time to time in the gold fields of Ontario, at Porcupine, Kirkland Lake, Cobalt, and Sudbury. Granite cutters in Ontario were examined in 1928. Other examinations have been made among foundry workers and among sandblasters, as well as workers in marble, talc, cement, brick, grain elevators, and artificial abrasives. These investigations were carried out by the Industrial Hygiene Division of the Ontario Health Department, where Cunningham, Riddell, and their associates have done work on correlating clinical, roentgen-ray, and pathological findings in lungs of workers. Belt of Toronto University has made comparisons between the silica content of the ash of silicotic lungs and the number of particles to be seen in the same lungs with a polarized microscope. The method brings out 'invisible

silica' so often predicated by pathologists working on silicosis.

Outstanding experimental research on silicosis is being carried out at the Banting Institute of the University of Toronto, under the direction of Banting, by Doctors King, Fallon, Irwin, Stantial, Franks, and others. Irwin has developed a micro-incineration method which makes it possible to determine amounts of silica in tissues with considerably greater accuracy for the purpose of relating it quantitatively to the degree and location of fibrosis present in the lungs.¹⁴

Recent experimental work by Denny, Robson, and Irwin showed that animals dusted with quartz, to which less than 1 per cent of metallic aluminum dust had been added, showed practically no fibrosis.³²

UNITED STATES

As early as 1887, Dr. Frederick Peterson¹⁴ of Poughkeepsie,³³ New York, presented a report of an autopsy upon a foundry worker which unmistakably indicated silicosis and called attention to the prevalence of this type of disease among fellow employees of his patient. In the same year, a series of patients employed in a cutlery factory were presented to the Franklin District Medical Society by Dr. F. J. Canedy of Shelburne Falls,¹⁴ Massachusetts.³⁴ Dr. William W. Betts of Nevada, in 1899,¹⁴ described³⁵ a series of cases of pulmonary disease among employees of a gold crushing plant.

Not until 1915 was the first study of silicosis and allied disorders initiated by the United States Bureau of Mines and the United States Public Health Service, jointly, in the lead and zinc mining district of southwest Missouri.³⁶ This investigation was followed by similar studies in hard rock metal mining regions in other parts of the United States.³⁷

In 1924, the United States Bureau of Mines established a clinic in the Tri-State Lead and Zinc mining district (Missouri, Kansas, and Oklahoma) which was later (1927) ex-

panded into a co-operative enterprise by the Bureau of Mines with the Tri-State Producers Association and the Metropolitan Life Insurance Company. This clinic was maintained for nine years. Over 60,000 complete physical examinations of 27,000 individuals were performed at this clinic while it was maintained under Government auspices, including roentgenogram of the chest and a blood Wassermann or Kline test. The results are partially summarized in Technical Papers 545 and 552 of the United States Bureau of Mines.³⁸ The general tenor of these studies in the United States was in close conformity with those of South Africa and England.

Attention was later directed to the anthracite coal mining industry, in which a disabling pulmonary disease, locally known as miner's asthma, had long been recognized. In 1933, the United States Public Health Service, in co-operation with the coal mine operators, the United Mine Workers of America, and the Pennsylvania Department of Labor and Industry, undertook a survey of pulmonary disease in the anthracite mines, the results of which were published in Bulletin 221 of the United States Public Health Service.³⁹ This study confirmed what many had suspected — that disabling pulmonary fibrosis among coal miners was due to silica and not to coal dust, although the coal might modify the clinical and pathological picture.

Other studies of the United States Public Health Service in bituminous coal mines failed to establish a disabling fibrosis in the absence of silica. Brundage of the Public Health Service had demonstrated from mortality records that bituminous coal miners had a low rate of tuberculosis. To the condition found in the anthracite mines, where silica bearing rock is often adjacent to the coal. Sayers and his colleagues gave the name anthraco-silicosis and demonstrated that in the older age groups, the incidence of tuberculosis as a complication was very prevalent. In the age group forty-five to fifty-five,

the tuberculosis rate was five times that of the general population and above age fifty-five, ten times as large.³⁹

About the same time, Gardner and Cummings of the Saranac Laboratory started their extensive studies of pulmonary disease in the iron ranges of northern Michigan and Wisconsin. The general conclusions of all these field studies indicated that the incidence of silicosis and the resulting clinical picture were directly related to the severity of exposure to quartz dust and to the presence of infection and that other substances, in combination with the silica, might and often did modify the action of the silica.

These conclusions are supported by the results of investigations of foundries. Foundry studies were conducted at various times by the United States Public Health Service,⁴⁰ by the Industrial Hygiene Division of the Metropolitan Life Insurance Company,⁴¹ by the Saranac Laboratory and the American Foundrymen's Association,⁴² the State of Connecticut,⁴³ and the State of Massachusetts.⁴⁴

A number of investigations were also made in the granite industry, notably in 1918 by the Committee on Mortality from Tuberculosis in the Dusty Trades,⁴⁵ under the leadership of Dr. Frederick L. Hoffman, and later, in 1929, the important study by Russell of the Public Health Service.⁴⁶

The sandstone industry was investigated by Hayhurst in 1926;⁴⁷ the marble industry by Dreessen of the Public Health Service in 1934;⁴⁸ and the pottery industry on a number of occasions.⁴⁹

The cement industry also has been thoroughly studied, beginning in 1915 by Tucker,⁵⁰ and later by the Public Health Service⁵¹ and also by the Saranac Laboratory.¹⁴ These various investigations of the cement industry revealed little or nothing in the way of occupational pulmonary disease and served to confirm the so-far accepted principle that disabling

pulmonary fibrosis is dependent upon exposure to the dust of uncombined or 'free' silica and not 'silicates.'

The manufacture of abrasives received considerable attention as did metal grinding. The United States Public Health Service,⁵² the United States Department of Labor, Dr.C.-E.A. Winslow⁵³ in Connecticut, Dr.W.Irving Clark⁵⁴ in Massachusetts, Dr.H.H.Kessler⁵⁵ in New Jersey, and others have reported on the abrasives industry; and Winslow and Greenburg,⁵⁶ W.H.Drury⁵⁷ and others on metal grinding.

During the years that these investigations were carried on, much laboratory and clinical research was under way. In Philadelphia, Stengel, Pancoast, Landis, T.Grier Miller, and Henry Field Smyth, interested in tuberculosis in industry, had conducted studies in the relationship of industrial dusts to pulmonary disease; numerous articles and reports came from this group, both collectively and individually, commencing about 1914.

During the years 1914-30, in association with the Bureau of Mines and later on their own behalf, the Public Health Service combined field studies with laboratory research in an effective manner. Miller in the Public Health Service demonstrated the technique of intraperitoneal injections of silica and other dusts to determine the tissue reaction.⁵⁹ Gardner and his associates at the Saranac laboratory demonstrated that attenuated tubercle bacilli in the presence of quartz dust would, in experimental animals, produce fatal tuberculosis without increasing the virulence of the bacilli.⁵⁸

In 1922, Greenburg and Smith introduced the impinger, a dust sampling device, which resulted in the impinger method becoming the standard for the United States Bureau of Mines and the Public Health Service.⁶⁰

The intimate relationship of roentgenological diagnosis to the silicosis problem was recognized early. In 1926, Pancoast

and Pendergrass published the first book to appear on this subject.⁶¹ These authors, together with Sampson and others, have carried on experimental work in technique and in elaborating standards of diagnosis.⁶²

The determination of disability in silicosis has proved vexatious to both clinicians and workmen's compensation officials. At the University of Rochester, McCann and his associates have been working on physiologic methods of estimating pulmonary impairment.

Harrington of the United States Bureau of Mines, Philip Drinker at the Harvard School of Public Health, Cummings at Saranac, Bloomfield and Dallavalle of the Public Health Service, Fehnel and his associates at the Metropolitan Life Insurance Company, have contributed a very large amount of work to the engineering and chemical methods of dust determination and control. Here again, the reports and publications are far too numerous to detail and the reader is referred to the original sources.

In addition to all the publications made available by all this field and laboratory work, the Harvard School of Public Health, the Saranac Laboratory, and the Division of Industrial Hygiene of the United States Public Health Service have undertaken direct educational activities. A recent organization is the Air Hygiene Foundation, sponsored by various industries to encourage research into atmospheric pollution and pulmonary disease and to carry on educational work.

2. HISTORY OF ASBESTOSIS

Asbestos was mined and the fibres were separated from the mineral and used in the making of fire-proof fabrics several centuries B.C. Attention is called by Cooke⁶³ to a cremation cloth described by Herodotus in 450 B.C. Materials made of asbestos have been used ever since but in the past three decades, the industry has grown tremendously owing to the

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greatly increased use of fire resisting and insulating materials as well as brake linings in the automobile industry.

Asbestos is a magnesium silicate, containing a small amount of iron and a trace of aluminum. The fibres are long and flexible and for many purposes are combined with cotton to undergo regular textile processes of spinning and weaving. Most of the asbestos used in the United States comes from Canada.

The first report of a death resulting from asbestos dust inhalation was presented by Murray in 1900.⁶⁴ A second case was reported by Cooke⁶⁵ in 1924 and again in 1927.⁶³ At that time, Cooke described the unusual and characteristic asbestosis bodies. In 1927, Oliver⁶⁶ reported 2 cases, to be followed by McDonald⁶⁷ and Seiler.⁶⁸ In 1928, Simson⁶⁹ reported 2 cases in South Africa. Other cases were reported by Wood and Page⁷⁰ in 1929, Haddow⁷¹ in 1929, and Wood and Gloyne⁷² in 1930; in that year appeared the comprehensive report of Merewether and Price.⁷³

In the United States, Lynch and Smith⁷⁴ reported the first cases in 1930. The first investigation of the asbestos industry in the United States was made in 1930 and 1931 by Lanza, McConnell, and Fehnel.⁷⁵ In the last few years, much attention has been given both to the clinical aspects and diagnosis of asbestosis and to its pathology. In 1936, Shull reported a roentgenological review of 71 cases.⁷⁶ Recently the United States Public Health Service⁷⁷ has published several reports of dust control methods in asbestos plants and of the industry in general.

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II. ETIOLOGY, SYMPTOMS, DIAGNOSIS OF SILICOSIS AND ASBESTOSIS

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1. ETIOLOGY, SYMPTOMS, AND DIAGNOSIS OF SILICOSIS

Silicosis has been variously defined : Dorland (Dict. 16th ed. pp.68) 'Pneumoconiosis due to inhalation of the dust of stone, sand, or flint ;' (Webster's New International Dictionary, 1930, p. 1956) 'An affection of the lungs occurring in stone cutters, caused by the inhalation of quartz dust.'

The Committee on Pneumoconiosis of the Industrial Hygiene Section of the American Public Health Association, recently defined silicosis as 'a disease due to breathing air containing silica (SiO_2), characterized anatomically by generalized fibrotic changes and the development of miliary nodulation in both lungs, and clinically by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis (some or all of which symptoms may be present), and by characteristic X-ray findings.' ¹

The compensation law of West Virginia defined silicosis as 'an insidious fibrotic disease of the lung or lungs, due to prolonged inhalation and accumulation sustained in the course of and resulting from employment, of minute particles

of dust containing silicon dioxide (SiO_2) over such a period of time and in such amounts as result in the substitution of fibrous tissue for normal lung tissues; and the term silicosis shall also include silicosis accompanied by tuberculosis of the lungs evidenced by the presence of tubercle bacillus in the sputum.²

While the foregoing definitions express somewhat different viewpoints, they have this in common — the cause of silicosis is silica or quartz. Although other dusts, when inhaled in sufficient concentrations over a long enough period of time, have been shown capable of producing a definite pulmonary fibrosis, nevertheless the pneumoconiosis characterized by nodular fibrosis has to date been shown clinically and experimentally to be associated only with the inhalation of dusts containing free or uncombined silica.

SILICA IN NATURE

Silica is the most abundant constituent of the minerals and rocks that make up the crust of the earth. It occurs in two forms, free and combined. The free silicas, as a group, are definite compounds in the form of SiO_2 . The combined forms are silicates. Of free silicas which occur in nature, that known as quartz is by far the most common. Quartz is a hard mineral and chemically resistant to reagents. It is an abundant constituent of granite, schist, and other rocks, and the chief component of sandstone and quartzite. Many ores are deposited in veins that consist nearly wholly of quartz. Probably the next most common form in which free silica exists in nature is the amorphous hydrated form known as Opal ($\text{SiO}_2 \cdot \text{H}_2\text{O}$). Opal is a silica of colloidal origin and occurs abundantly in the diatomaceous earths. It is less resistant to reagents than quartz. Another type of free silica is flint and with flint is found chalcedony, a waxy, translucent form of silica interpreted as consisting of fibres of quartz with a small

amount of interstitial opal. Other forms of free silica occurring less abundantly in nature are tridymite, cristobalite, and siliceous glass or vitreous silica.

OCCUPATIONAL EXPOSURE TO SILICA

As the earth's crust contains so large an amount of silica, it is obvious that those occupations concerned with mining and the driving of tunnels are associated with a silicosis hazard. Other occupations exposing the workers to this hazard are those concerned with the processing and industrial use of mineral products, such as smelting and refining, the use of sand and gravel for structural purposes; the cutting of stone, particularly granite; the manufacture and use of certain abrasives; and the processing of the various forms of free silica. According to Knopf,³ the most common forms of free silica used industrially are massive crystalline quartz, quartzite, sandstone, flint, tripoli, diatomaceous earths and silica sand. Table 1, from Ladoo,⁴ illustrates the great variety of uses to which silica is put in industry and the kind of silica adapted to each purpose.

TABLE I

USES OF SILICA	TYPES OF SILICA USED
Abrasive uses:	
In scouring and polishing soaps and powders	Quartz, quartzite, flint, chert, sandstone, sand, tripoli, and diatomaceous earth; all in finely ground state.
In sandpaper	Quartz, quartzite, flint, sandstone, and sand; coarsely ground and closely sized.
In sand-blast work	Quartz, quartzite, sandstone, and sand, crushed into sharp angular grains uniform in size.
Metal buffing, burnishing, and polishing	Ground tripoli and other forms of ground silica.

USES OF SILICA	TYPES OF SILICA USED
For sawing and polishing marble, granite, etc.	Sharp, clean sand graded into various sizes.
As whetstones, grindstones, buhrstones, pulpstones, oilstones, etc.	Massive sandstone from very fine to moderately coarse grained.
Tube-mill lining	Chert, flint, and quartzite in dense solid blocks.
Lithographers' graining sand	Medium to fine sand or rather coarsely ground silica and tripoli.
Tube-mill grinding pebbles	Rounded flint pebbles.
In tooth powders and pastes	Various forms of pure silica finely ground.
Wood polishing and finishing	All forms of silica ground to medium fineness.
Refractory uses:	
In making silica fire brick and other refractories	Fairly pure quartzite known as ganister; not less than 97 per cent SiO_2 , nor more than 0.40 per cent alkalis, tightly interlocking grains desired.
Metallurgical uses:	
In making silicon, ferrosilicon, and silicon alloys of other metals, such as copper	Moderately pure sand, massive crystalline quartz, sandstone, quartzite, or chert.
As a flux in smelting basic ores	Massive quartz and quartzite.
Foundry-mold wash	Ground sandstone, quartz, and tripoli.
Foundry parting sand	Fine sand and ground tripoli.
Chemical industries:	
As a lining for acid towers	Massive quartz or quartzite.
As a filtering medium	Massive diatomaceous earth and tripoli, sand, finely granular quartz or quartzite, finely ground tripoli, diatomaceous earth, and other forms of silica.
In the manufacture of sodium silicate	Pure pulverized quartz sand, pure tripoli, and diatomaceous earth.
In the manufacture of carborundum	Pure quartz sand.
Paint:	
As an inert extender	Finely ground crystalline quartz, quartzite, and flint; also finely ground sandstone, sand, and tripoli.

USES OF SILICA	TYPES OF SILICA USED
Mineral fillers :	
As a wood filler	Finely ground crystalline quartz, quartzite, flint, tripoli, and other types of ground silica.
In fertilizers	
In insecticides	
As a filler in rubber, hard rubber pressed and molded goods, phonograph records, etc.	Finely ground silica of all types.
In road asphalt surfacing mixtures	
Ceramic uses :	
In the pottery industry as an ingredient of bodies and glazes	Flint, tripoli, and chert, and other amorphous silica preferred ; also all other forms of very pure silica, all finely ground.
In the manufacture of ordinary glass	
In the manufacture of fused-quartz chemical apparatus such as tubes, crucibles and dishes	
Decorative materials :	
In the manufacture of gems, crystal balls, table tops, vases, statues, etc.	Rock crystal, amethyst, rose quartz, citrine quartz, smoky quartz, chrysoprase, agate, chalcedony, opal, onyx, sardonyx, jasper, etc.
Insulation :	
Heat insulation for pipes, boilers, furnaces, kilns, etc.	
Sound insulation in walls, between floors, etc.	Massive and ground diatomaceous earth.
Structural materials :	
Sand-lime brick	Moderately pure, sharp, angular sand, preferably finer than 20-mesh, together with a small percentage of finely pulverized silica.
Optical quartz :	
For the manufacture of lenses and accessories for optical apparatus	Clear, colorless, flawless rock crystal or massive crystallized quartz.

In a recent survey,⁵ carried on in a large manufacturing center, it was found that about 9 per cent of the industrial workers were employed in occupations where the silica hazard required consideration. According to the census from 1930, there were gainfully employed in the manufacturing and

mechanical industries in this country approximately 14,000,000 persons. If the above survey can be accepted as representative of the occupational distribution of these workers, it would appear that there are nearly 1,200,000 individuals possibly exposed to a silicosis hazard in the manufacturing and mechanical industries alone. Lanza and Vane,⁶ in their discussion concerning the prevalence and effect of silicosis, state: 'Our very rough, but obviously conservative estimate of the number of workers exposed to silica dust to a harmful degree in the United States is, therefore, upwards of 500,000.'

FACTORS INFLUENCING THE ACTION OF SILICA DUST PARTICLES AS THE EXCITING CAUSE OF SILICOSIS

Although it has been accepted that silica is the exciting cause of silicosis, there are certain factors which must be considered as influencing its action. Early workers were inclined to consider that the injury produced by the dust particle was due to the mechanical irritation of its hard and cutting edges. Gardner⁷ has shown experimentally that the inhalation of finely divided carborundum dust, which has sharp edges and a greater hardness than silica, does not produce the nodular reaction characteristic of silicosis. Collis⁸ was one of the early workers to draw attention to the chemical action of dust. Gye and Kettle⁹ have shown that silica in solution or non-crystalline form exerts a toxic action upon the tissues which leads to the proliferation of fibroblastic cells. Lately, Miller and Sayers¹⁰ have reported results of experimental studies and have classified the reaction of peritoneal tissues to certain dusts. Only the silica containing dusts have uniformly produced the proliferative harmful reaction. Other dusts have been either completely absorbed, leaving no scar tissue, or have remained unaltered in the form in which they were injected. These latter reactions are classed as absorptive and inert.

Since dust, to exert its harmful action, must enter the finer divisions of the lung, its particle size bears a definite relationship to its injurious effects. The silica must be present in the air in particles small enough to enter the finer air spaces and of such dimensions that the phagocytic cells may engulf them. The natural defenses of the respiratory tract probably prevent many particles larger than 10 microns from reaching the finer divisions of the lung and such as do are likely to be expelled with the bronchial secretions. The solubility of the silica may play a definite part in the production of disease and the size of the particle also affects the rate of solution as the smaller the particles, the greater the total surface area exposed to the action of solvents.

Table 2 shows the size distribution of various industrial dusts as compared with the dust particles observed in the outdoor air in the general atmosphere. About 70 per cent of the particles found in industrial dusts generally are between 0.5 and 3 microns in diameter. There are, no doubt, a great many particles too small to count by the method used but experimentally it has been shown that such sub-microscopic particles are not retained in the lungs but pass out with the expired air. Sayers¹¹ has shown that less than 15 per cent is retained when finer particulate matter, such as lead in the form of fumes, is inhaled. The majority of particles found upon microscopic examination of lung tissue also fall within the limits of from 1 to 3 microns.

Another reason for considering the size of the particles as affecting the harmfulness of the dust, is that the larger ones settle out with comparative rapidity from the atmosphere while the rate of falling for the smaller particles is very slow. Figure 1 illustrates graphically this difference ; particles under 1 micron falling at the rate of from 1 to 3 feet per hour, varying with the specific gravity, while particles 5 microns in diameter and of specific gravity 7 fall about 60 feet per hour.

TABLE 2
SIZE-FREQUENCY DISTRIBUTION OF VARIOUS INDUSTRIAL DUSTS AS COMPARED TO OUTDOOR DUST
[Average frequency in per cent]

Kind of dust	Number of Samples	Median	Size Group in Microns											
			0-0.49	0.5-0.99	1-1.49	1.5-1.99	2-2.49	2.5-2.99	3-3.49	3.5-3.99	4-4.49	4.5-4.99	5-5.49	5.5-5.99
Outdoor dust	179	0.5	56.0	41.0	2.5	0.5	—	—	—	—	—	—	—	—
Sandblasting	9	1.4	1.4	19.7	34.7	20.3	12.6	5.2	2.8	1.6	1.1	0.2	0.2	0.2
Granite cutting	4	1.4	2.0	19.0	33.6	24.5	10.4	4.6	3.1	.6	.9	.3	1.0	—
Trap-rock milling :														
Crusher house	1	1.4	—	13.0	39.0	33.0	10.5	2.5	2.0	—	—	—	—	—
Screen house	1	1.3	2.0	31.5	33.0	16.0	10.0	4.5	2.5	.5	—	—	—	—
Disc crusher	1	.9	10.0	48.0	31.0	6.0	3.0	1.0	1.0	—	—	—	—	—
Foundry parting compound	2	1.4	.5	22.0	42.0	17.3	9.2	5.0	1.5	2.0	.5	—	—	—
General foundry air	1	1.2	—	26.0	48.0	17.0	8.0	1.0	—	—	—	—	—	—
Talc milling	1	1.5	—	16.0	32.0	20.0	13.0	7.0	5.0	2.0	2.0	2.0	1.0	1.0
Slate milling	1	1.7	1.0	13.0	29.0	17.0	14.0	14.0	6.0	4.0	1.0	1.0	1.0	1.0
Marble cutting	1	1.5	—	12.0	37.0	21.0	10.0	11.0	3.0	1.0	1.0	2.0	2.0	1.0
Soapstone	2	2.4	1.2	16.0	19.0	13.0	11.0	6.0	6.5	4.5	5.5	3.3	2.5	11.5
Aluminium dust	1	2.2	3.0	8.0	20.5	14.0	11.5	9.0	6.5	3.0	3.5	4.0	7.0	10.0
Brass dust	1	1.5	1.0	12.0	33.5	25.0	21.0	6.0	1.5	—	—	—	—	—
Anthracite-coal mining :														
Breaker air	2	1.0	7.0	51.0	26.0	8.0	3.0	3.0	2.0	—	—	—	—	—
Mine air	1	.9	11.0	60.0	17.0	7.0	3.0	1.0	1.0	—	—	—	—	—
Coal drilling	1	1.0	1.0	55.0	34.5	7.5	1.5	—	.5	—	—	—	—	—
Coal loading	3	.8	11.5	56.3	24.3	5.5	1.5	.7	.2	—	—	—	—	—
Rock drilling	1	1.0	4.0	49.0	25.5	12.5	5.5	1.5	.5	1.5	—	—	—	—

Particles of more than 10 microns settle out in a relatively short time.

Thus we may say that the harmfulness of a given dust is directly influenced by the number of particles it contains of free silica less than 10 microns in diameter and that probably the most damage is produced by those between 1 and 3 microns.

The relationship of dust concentration and duration of exposure is closely associated in their etiological significance. The rate at which silicosis will develop, excluding certain factors considered as predisposing, depends upon the dosage

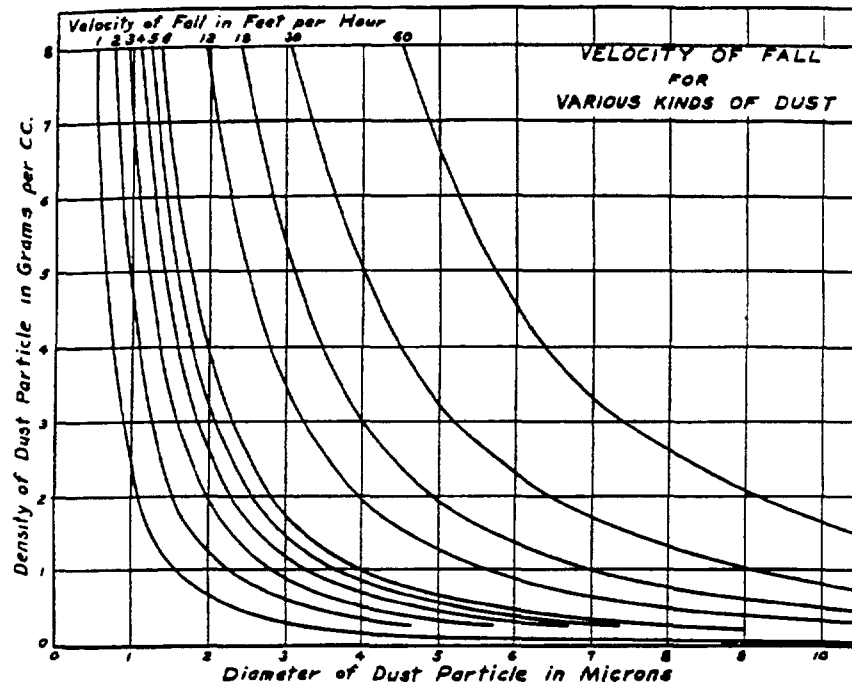


FIGURE 1

- Diameter of Dust Particle in Microns — Density of Dust Particle in Grams per CC.

of free silica. This dosage is obviously dependent upon the amount of silica in the air inhaled and the duration of exposure. In this lies a point of great practical importance which explains the apparent discrepancies between the reports of different investigations with respect to the time necessary for silicosis to develop.

In 1902, a committee, of which Dr. J. B. S. Haldane¹² was a member, reported upon an investigation made to determine the cause of excessive mortality rates from tuberculosis among Cornish tin miners. These investigators pointed out that it was evident that the inhalation of stone dust by these miners was the cause of permanent damage to the lungs. Furthermore, they noted that the condition developed gradually in the case of the ordinary miner but rapidly in the case of the machine workers who were exposed to greater amounts of dust. Dr. Watkins-Pitchford¹³ and Dr. Mavrogordato,¹⁴ as well as other South African investigators, emphasize the relationship of the concentration of dust and duration of exposure to the degree of lung changes produced. Intermittent employment has been suggested as an aid in preventing silicosis but aside from the fact that it does delay the ultimate result, it has not been shown that such interruption of work can safely be relied upon where harmful concentrations of dust are involved. In the United States, the reports of the Picher Clinic¹⁵ have indicated the effects of varying periods of exposure to silica in the mines of that district. Similar studies of the health of granite workers^{16 17} likewise stress the duration of exposure necessary to produce definite degrees of silicosis.

Various investigators have stated that the presence of other substances in silica containing dust may influence the action of silica tending either to aggravate or retard the usual reaction. The question was raised by Heffernan¹⁸ in 1926 as a result of a study of a group of brick makers in Derbyshire. He

concluded that the clay present in the silica dust was responsible for the absence of silicosis in this group of workers. Gardner is strongly of the opinion that the action of silica is modified by other substances which may be in combination with it and this is supported by the recent Canadian reports with reference to the action of metallic aluminum.¹⁹ What the implication of this may be with respect to specific dust problems in various industries remains to be determined.

Cases of so-called acute silicosis, resulting from the presence in highly siliceous dust of alkali in a fine powdered form, have been reported by Chapman,²⁰ Kessler,²¹ MacDonald,²² Kilgore,²³ and others. Some have suggested that the action of silica is made more rapid by the presence of these alkalis because silica is more soluble in alkaline solutions. Kettle²⁴ failed to demonstrate such action experimentally and states that proof is still lacking that any action of the kind may occur. The effects of inhaled coal dust in conjunction with silica have been reported at length by the Public Health Service²⁵ in their study of anthraco-silicosis among hard coal miners.

Taken all in all, the available data indicate that other components of silica laden dust are not merely inert diluents whose effect is to reduce the amount of silica inhaled. Some of these substances may act in this manner; others apparently aid in removing the silica particles from the atmosphere; and others may accelerate or retard the action of silica. Moreover, when inhaled with silica, these latter tend to alter the anatomical form of the silicotic lesion. The degree of modification is determined by the character and amount of the adulterant dust.

OCCUPATIONAL HISTORY

Most important in determining the significance of a given dust exposure is a complete, carefully taken occupational

history, whereby the total dust exposure may be estimated and compared with other cases of a similar nature. Tables 3, 4, and 5 illustrate a practical method of analyzing and recording occupational factors in the study of a dusty industry.²⁵

TABLE 3

OCCUPATIONAL RECORD

Division of Industrial Hygiene — United States Public Health Service

Dated : August 8, 1933		Office : Industrial Hygiene	
Name K. M.		Present age : 49	
Age began work : 15		Number of years worked : 34	
	<i>Specific Occupation</i>	<i>Specific Industry</i>	<i>No. of years in Hard Coal Non-dusty</i>
Present	Section foreman	Anthracite coal	5
Preceding 1	Contract miner (chamber)	" "	15
2	Miners' laborer (chamber)	" "	3
3	Mule driver (dry mine)	" "	3
4	Patcher (dry mine)	" "	2
5	Slate picker (dry breaker)	" "	2
6	Farm laborer (Pa.)	Agriculture	1

Remarks : Estimating the total time idle during working life — 3 years

Table 4 shows the occupations and associated dust exposure of chamber miners and laborers. Each occupation engaged in by these coal miners was studied separately. Experience has shown that the various occupations comprising the activity of any dusty industry are usually associated with dissimilar dust exposure.

Table 5 shows a method of analysis used in estimating an individual's total dust exposure.

With such occupational histories available and the knowledge of the percentage of total dust exposure revealed by detailed occupational analysis, it was found possible in a recent study to predict in 9 cases out of 10 approximately the

TABLE 4
DUST EXPOSURE OF CONTRACT MINERS AND HELPERS
(LABORERS)
Chamber Mining

<i>Occupation</i>	<i>Number of Samples</i>	<i>Number of Hours in Activity</i>	<i>Average Dust Count (millions of particles per cubic foot of air)</i>	<i>Millions of Particle- Hours Per Cubic Foot</i>
Jack-hammer drilling	23	1	575	575
Hand loading	22	2	1,138	2,276
After firing	7	$\frac{1}{4}$	834	209
Taping and wiring	2	$\frac{1}{4}$	40	20
Setting up props, and in main airways	8	$2\frac{3}{4}$	15	41
Totals	62	$6\frac{1}{4}$	—	3,121
3,121 million particle-hours per cu.ft. $6\frac{1}{4}$ hrs. = 480 million particle-hours per cu.ft.				

pulmonary condition that would be found upon physical examination, insofar as changes due to dust inhalation were concerned.

A statement on the effect of variations and concentrations and composition was developed by the Committee on the Prevention of Silicosis through Medical Control.²⁶ An abstract of this report follows.

The effects of the variation in concentration of silica in the air breathed must be judged by data from those field and laboratory studies which have reported results that can be compared. The following concentrations have been accepted as permissible for particular industries in the localities indicated.

*It is believed that the effects of other substances associated

with silica in atmospheric dusts are sufficiently marked and well defined to preclude comparison of silica hazards merely on the basis of the amount of free silica in the atmosphere. It is concluded that atmospheric concentration of silica, which may have proved incapable of producing silicosis in one industry, will not necessarily be harmless under other conditions. The reverse is equally true. If these premises are as

TABLE 5

ESTIMATION OF THE INDIVIDUAL'S TOTAL DUST EXPOSURE

Name K..... M.....			
Occupation Foreman	Number of Years	Dust Concentration	
		in Millions of Particles Per Cubic Foot (average)	Millions of Particle-Years Per Cubic Foot
Slate Picker (dry mine)	2	380	760
Patcher (dry mine)	2	71	142
Mule driver (dry mine)	3	71	213
Miner's laborer (chamber)	3	480	1,440
Contract miner (chamber)	15	480	7,200
Section foreman	5	7	35
Total	30	—	9,790
$\frac{9,790 \text{ millions of particle-years per cu. ft.}}{30} = 326 \text{ millions of particle-years per cu. ft.}$			

sound as they seem, there can be no universal regulatory standard of permissible dust concentration at the present time. Separate standards must be set for different industrial dusts and processes. When all of the factors determining such standards have been more accurately measured and correlated it may become possible to use them as a basis for the general regulatory standard.

'It is recommended that clinical and engineering studies be carried out as rapidly as possible to establish the necessary

scientific data upon which we may base practical and effective regulatory standards.

The factors necessary to establish a safe concentration of atmospheric dust are the following :

1. The relative number and size of the free silica and the other particles suspended at breathing levels in the different parts of the industrial atmosphere.

<i>Industry</i>	<i>Percentage of Silica in the Dust</i>	<i>Permissible Maximum Safe Dust Concentration Millions per cubic foot</i>
(1) South Africa Gold Mines	80%	4½
(1) Ontario Gold Mines	about 35% (in the rock)	8½
(1) Australia Sandstone	90% (in the rock)	6
(2) Barre Granite	31% to 35%	10 to 20
(2) Pennsylvania Anthracite Mines	35%	5 to 10
	13%	10 to 15
	5%	50
(1) Broken Hill, Australia Lead-Zinc Mines	10% to 17%	14
(1) Based on engineering practice. (2) Based on clinical studies.		

2. The proportion of the workmen's time actually spent in the various dust concentrations.

3. The effects of exposure to environment, as revealed by the occupational and medical histories of old employees, by physical examination and by the roentgenological appearance of their lungs.

From the viewpoint of silicosis prevention, the standard of safe atmospheric dust concentration is the maximum in which persons can spend their entire industrial lives without

producing demonstrable evidence of significant reaction in their lungs.

'There is evidence that for prolonged exposure, a concentration of more than 5,000,000 particles per cubic foot of a highly siliceous dust is dangerous. Therefore, it is now considered good practice to hold concentrations of highly siliceous dust at 5,000,000 particles per cubic foot or less.

'Since standards of safe atmospheric dust concentration, based on medical findings, have been established but for a few industrial dusts, and in view of the fact that to establish such standards for other industrial dusts will require considerable study and investigation, some arbitrary standard to serve tentatively as a measure of good practice may be useful. This arbitrary standard should be based upon what is believed to be within the limits of good engineering practice and that which, from a medical viewpoint, it is judged will largely control the silicosis hazard for most industrial exposures. It has been suggested that the maximum permissible concentration of silica in the air breathed might be expressed by the following formula (Determination of dust concentration according to the technique described by the United States Public Health Service in Reprint 1520 from Public Health Reports, March 18, 1932) : Multiply the percentage of free silica by the total particle dust count. If the result is under 5,000,000, the condition may be considered permissible. If the result is over 5,000,000, the condition may be considered too high. For example, 10 per cent free silica with an average total dust concentration of 30,000,000 particles per cubic foot would give 0.10 times 30,000,000 which equals 3,000,000 (good practice) ; 30 per cent with an average total dust concentration of 50,000,000, would equal 0.3 times 50,000,000 or 15,000,000 (unsatisfactory). This formula is not applicable to any dust containing less than 3 per cent free silica.'

PREDISPOSING CAUSES

The wide distribution of silicosis indicates that all races are susceptible and that no nationality is exempt. Although available data show that the incidence of silicosis is higher among the younger miners in districts where the percentage of free silica is high and among older miners where the percentage of silica is low, age, in itself, is probably not a factor. The history of previous dust exposure and of previous respiratory infection is obviously of the utmost importance in the study of either an isolated case or of an industrial group. Individual susceptibility is frequently mentioned but the weight of evidence is that it may be considered an acquired and not a congenital condition.

Lehmann's ²⁷ experiments to determine the functional efficiency of the upper respiratory tract, in the removal of dust, suggest that abnormalities of the nasal passageways may affect the time element in the development of silicosis.

Infections of the upper respiratory tract may be of importance. Sinus infections may act by decreasing the efficiency of the upper respiratory tract in the removal of dust from the air passing to the lungs and also may be responsible for the spread of infection to the lower respiratory tract. Acute pneumonic conditions as well as the more chronic lung changes such as chronic bronchitis, bronchiectasis, and bronchiolectasis, emphysema, and pleurisy, all tend to lessen the ability of the lung to rid itself of foreign materials, through impaired lymphatic drainage and inability to force the bronchial secretions and foreign matter from the lungs.

SYMPTOMS

The Committee on the Prevention of Silicosis through Medical Control ²⁶ has summarized the symptoms associated with silicosis both in the presence and in the absence of infection, as follows (condensed) :

SUBJECTIVE SYMPTOMS

Dyspnea. The complaint most frequently mentioned is shortness of breath. Depending upon the extent of the involvement, this varies from slight dyspnea, following exertion, to marked dyspnea upon the least exertion or even when at rest. The shortness of breath noted in silicotics presents one peculiarity in that it is seldom accompanied by orthopnea, the individual being no more short of breath lying down than when in an upright position. This may not be so, however, when silicosis is complicated by cardiac disease or by true asthma.

Silicosis

Noted as a rule only after sudden or extra exertion. Seldom so marked as to interfere with routine duties. However, in cases with extensive pulmonary fibrosis, it may limit the individual's activities.

Silicosis with Infection

If complicating infection is not widespread, may be no more marked than in cases of simple silicosis but as infection and fibrosis increase, it becomes disabling.

Cough. Many silicotics complain of a troublesome cough. This cough differs from that resulting from simple irritation due to dust which clears up upon removal from exposure. When present, it is more pronounced in the morning or upon beginning work after a rest period.

Silicosis

The typical silicotic cough is dry and non-productive. It usually parallels shortness of breath in degree and may contribute to disability.

Silicosis with Infection

The cough usually becomes more troublesome and is productive. The sputum varies from thick, tenacious, mucous material to that of a foul, purulent or purohemorrhagic consistency.

Microscopic examination or animal inoculation frequently reveal tubercle bacilli or, in some cases, organisms of the fusiform spirochetal group. In advanced cases, coughing attacks are often of such severity as to leave the individual exhausted.

Chest Pain. This symptom is complained of by a majority of silicotics. It varies from a feeling of tightness in the chest to the sharp pain typical of pleurisy. (Since chest pain is offered as a complaint in many conditions, it cannot be stressed as especially characteristic of silicosis.)

Silicosis

A late symptom in cases of simple silicosis. Then seldom more than a sense of tightness or feeling of substernal pressure.

Silicosis with Infection

Pleuritic pain is suggestive of a complicating infection. It is increased by exercise and by coughing and may be distressing in advanced cases with extensive infection.

Hemoptysis. True hemoptysis seldom occurs. Frequently, however, the sputum may be blood-streaked following a severe coughing attack. Hemoptysis must always be considered as suggestive of tuberculosis.

Silicosis

Occasional blood-streaked sputum. May result from alveolar rupture following sudden exertion in advanced cases.

Silicosis with Infection

May be the first indication of tuberculous infection. May be consequent upon the development of pneumothorax. May occasionally be excessive if cavities are present.

General Complaints. Weakness, loss of weight, digestive disturbances, night sweats, insomnia, dizziness, and edema of the extremities are not characteristic of uncomplicated silicosis but are apt to be present if infection supervenes, especially when the infection becomes extensive.

OBJECTIVE SYMPTOMS

Changes in the general appearance are infrequent in simple silicosis unless far advanced. Such changes as are manifested are usually due to complicating conditions.

Silicosis

Early cases appear unchanged; in fact, it is common to find these individuals showing a slight increase in weight, possibly because they are less active. As the disease progresses, respiratory embarrassment is noticeable and there is a general loss of muscle tone.

Silicosis with Infection

The appearance sooner or later becomes that of chronic phthisis. The bony landmarks of the thorax become prominent and there is an increase in the anterior-posterior diameter of the chest, possible hypertrophy of the accessory respiratory muscles of the chest, and in the final stages, retraction of the supra and infra clavicular spaces. Cyanosis and clubbing of the fingers are not prominent except in those cases of long standing, with cardiac disturbances.

Chest Expansion. Decrease in the expansion of the chest may be demonstrated in cases with extensive pulmonary fibrosis.

Silicosis

In early cases it is usually not possible to demonstrate de-

Silicosis with Infection

Decrease in expansion may not be noted in early silicosis with

creased expansion. In advanced cases, expansion may be lessened by 20 to 30 per cent but remains equal on both sides.

slight infection but as the condition progresses, a definite decrease is readily observed. When infection is more pronounced in one area of the lung, expansion may be more markedly decreased on the affected side, particularly if there is pleural involvement.

Prolonged Expiration. In most cases, decreased chest expansion is preceded and later accompanied by a definite change in respiratory rhythm. Close observation reveals that even at rest there is a distinct tendency to prolongation of the expiratory phase, which, as silicosis advances, becomes more marked. Following exercise, the silicotic may breathe less rapidly than the normal person under similar conditions as the lungs cannot be emptied rapidly enough to permit more rapid respiration; but although the respiratory rate is not so rapid, as in the normal person, it will persist for a longer period.

Silicosis

Early in the development of simple silicosis, prolonged expiration may be evident only after exertion. Later the degree of prolongation usually parallels the increase in pulmonary fibrosis.

Silicosis with Infection

In cases of early silicosis with slight infection, prolongation of the expiratory phase may be no more marked than in simple silicosis. As the condition progresses and fibrosis increases, it may simulate characteristic asthmatic respiratory rhythm.

Signs Revealed by Palpation of the Chest. Except in the late stages of silicosis with infection, little is revealed by pal-

pation of the chest. However, where there is a measurable decrease in expansion, one may note by palpation that, upon forced inspiration, the anterior chest wall is lifted forward by the accessory muscles of respiration.

Silicosis

No change noted until extensive fibrosis has taken place, when there may be an increase in tactile fremitus generally.

Silicosis with Infection

When infection is extensive, tactile fremitus is increased and occasionally friction rubs may be elicited. Extensive thickening of the pleura or pneumothorax may result in a decrease or absence of tactile fremitus over affected areas.

Percussion. There is usually an impairment of the percussion noted over the whole chest but unless one is particularly observant, this may not be detected.

Silicosis

Since impairment in resonance is general over all lung areas, it is difficult to demonstrate until advanced fibrotic changes have developed. Decrease in diaphragmatic excursion may sometimes be revealed by percussion.

Silicosis with Infection

Increase in loss of normal resonance. When massive areas of fibrosis have developed, this may amount to absolute dullness over areas involved. Amphoric resonance may be elicited where there is pneumothorax. Decreased diaphragmatic excursion may readily be shown in advanced infection.

Auscultation. Breath sounds are usually decreased in intensity and the characteristic prolongation of expiration is readily noted.

Silicosis

Decrease in breath sounds general and more marked as the condition progresses. Sub-crepitant rales, which clear up after coughing, are occasionally heard.

Silicosis with Infection

Usually heard, some of the following: Persistent post-tussic crepitant and sub-crepitant rales; coarse rhonchi associated with productive coughing, wheezing or musical rales increased by exertion and coughing, amphoric breath sounds over cavities and areas of pneumothorax; pleural friction rubs occasionally.

DIAGNOSIS

The diagnosis of silicosis must be based upon information furnished by past occupational and medical history, the record of present complaints, and the evidence revealed by a careful physical examination of the chest, including a roentgenogram.

The past and present occupational history should, as far as possible, detail the specific jobs in each industry in which the worker has been employed during his entire working life and estimate the time spent at each job and the duration and nature of previous dust exposure.

The medical history should include all disabling illnesses and any history of contact with cases of active tuberculosis. The dates of all illnesses should be recorded.

Physical examination of the chest should be carried out in a systematic and thorough manner. Subjective symptoms and positive clinical signs are usually absent or difficult to elicit in early and uncomplicated silicosis; there are no signs or symptoms which may be considered as pathognomonic. Where the disease has progressed to the point when there is

decreased capacity for work, there will usually be clinical evidence of pulmonary changes.

Included in the diagnosis should be a statement whether there is evidence of tuberculous infection, either active or latent, or infection of other nature.

A positive diagnosis of silicosis should not be made without a good roentgenogram of the chest, revealing nodular shadows throughout the lung fields. Although it is not advisable to make a diagnosis of silicosis based upon roentgen-ray findings alone, nevertheless the chest conditions revealed by fluoroscopic and roentgenological examinations of the chest furnish the most convincing evidence of pulmonary fibrosis. Diagnosis by roentgen-ray is discussed in a separate chapter.

A fluoroscopic examination is of distinct advantage. Particularly is it possible, by such procedure, to study diaphragmatic movements and note the presence of areas of increased density which, of course, are better shown in detail by film examination. The general information regarding pulmonary density furnished by fluoroscopic examination is of benefit in deciding the particular technique to use to secure the best roentgenogram of the lungs.

Laboratory Examination

Laboratory examination of the blood, urine, and sputum are of value when correlated with the clinical manifestations of silicosis. There are no blood characteristic of simple silicosis. When infection is present, blood examinations are sometimes helpful in determining its severity and progress. Cases complicated by tuberculosis often show an increase in the sedimentation rate, paralleling the activity of the tuberculous complication.

Urinalysis offers little of particular value in silicosis. Uri-

nary silica determinations may show an increase in silica present, paralleling the silica exposure.²⁸

The chief value of sputum examination is to determine the presence of tubercle bacilli. As in the case of chronic fibroid phthisis, too much dependence must not be placed upon microscopic examinations of the sputum. Where many sputum examinations have been negative for tubercle bacilli, guinea pig inoculation or sputum culture may indicate their presence.

THE RELATION OF SILICOSIS TO DISABILITY

A diagnosis of silicosis does not necessarily mean disability. Since it is a slowly developing disease, roentgenological evidence of silicosis may exist for many years prior to the demonstration of any decreased capacity for work.

The term 'disability' is usually employed to indicate lessened capacity to do the work required of an individual in the course of his usual occupation. It may vary from partial to complete. Disability may persist as partial, but when the fibrosis is complicated by pulmonary tuberculosis, it becomes serious and permanent. Individuals with active tuberculosis are considered unsuitable for further employment in even a minimal silica exposure, regardless of whether or not they may also be silicotic.

The existence of marked or complete disability is relatively easy to establish. However, the clinician may find it possible to arrive at such a conclusion only after repeated examinations, made over such a period of time as to afford him an opportunity to determine definitely the nature and severity of infection and other complicating conditions.

The matter of partial disability is far more difficult to determine satisfactorily. The cause and extent of partial disability frequently requires careful examination to be ac-

curately gauged before proper placement of a worker can be effected or a fair settlement of a compensation claim achieved.

To declare whether or not a workman should be allowed to continue at his work is a grave responsibility and calls for the utmost care, as well as experience, on the part of the examining physician. Fitness for work may be expressed as follows :

I. Partial disability due to silicosis without infection :

- (a) May continue at usual occupation, if environment is satisfactorily controlled as regards dust concentrations and tuberculosis contact ;
- (b) Degree of fibrosis and rate of development may require that the individual seek less arduous employment and avoid even minimal exposures to silica-containing atmosphere.

II. Partial disability due to silicosis with complicating pulmonary infection :

- (a) Primary silicosis. Infection mild and non-tuberculous. In most instances, after successful treatment of non-tuberculous complicating infection, may be allowed to return to usual work, provided environment under which they are working is safely controlled.
- (b) Primary silicosis with pulmonary tuberculosis as complicating infection. Those individuals with silicosis who develop pulmonary tuberculosis are considered totally unfit for further employment in an industry affording even minimal exposures to free silica. This disability is obviously total, so long as active tuberculosis is present. Following successful treatment, these cases regain their health to the point where they can be safely employed at other work.

PROGNOSIS

There is but a limited amount of factual data regarding the progress of silicosis and the relationship of a previous or superimposed infection to the progressive nature of this condition must be considered. Nor is there adequate information as to the ultimate results to silicosis cases after removal from exposure, and the extent of their subsequent pulmonary infection.

It may be stated that the prognosis of the individual case of silicosis is almost as variable as are the industrial conditions which cause the disease. Where the hazard is not very serious and the resulting damage to the lungs of slight or moderate degree, with removal from further dust exposure, there may be little to apprehend either in the way of disability or shortened life. Tubercle infection occurring in such cases tends to become chronic and the patient may live for a long time with no marked disability as do other cases of fibroid phthisis.

Where the pulmonary damage from silica is considerable, the outlook is usually unfavorable and when, as so often happens, this type of patient becomes tuberculous, his prognosis is very grave. Everything considered, the silicotic who is removed from further dust exposure has a fairly good prognosis. When tuberculosis supervenes, he may go the quick route or the slow one. His chances of securing an arrest of his tuberculosis are much less good than if his lungs were not previously damaged by silica.

2. ETIOLOGY, SYMPTOMS, AND DIAGNOSIS OF ASBESTOSIS

Asbestosis has been defined by Merewether²⁹ as 'a specific occupational disease of the lungs caused by the inhalation of asbestos dust and characterized by progressive replacement

of the essential active functioning tissue of the lung by inactive non-functioning fibrous or scar tissue. It is essentially a pneumoconiosis, a fibrosis of the lungs, caused by the inhalation of dust and, therefore, is in the same category as silicosis, which disease it resembles in some respects, while differing considerably in others.'

Asbestos is a hydrated magnesium silicate. More than 90 per cent of the raw mineral used in manufacture in the United States and Great Britain is Canadian chrysotile. While most of the asbestos dusts in industrial plants are made up of very small particles, asbestos fibers exceeding 200 microns in length have been found in the lung tissue of asbestos workers.

Asbestos is subjected to two main manufacturing processes. It may be combined with diatomaceous earth and other similar inert substances to be used for packing, insulating and fireproofing materials, or it may be combined with cotton or other materials and submitted to the usual textile processes of cleaning, mixing, carding, spinning, twisting, and weaving. The asbestos cloth may in turn be subjected to further processes of impregnation with various substances for special purposes.

Merewether and Price,³⁰ in their comprehensive report, state that approximately 2,200 individuals are exposed to practically pure asbestos dust in the factories of Great Britain. They examined 363 workers and the following table gives the result.

<i>Years at Work</i>	<i>Cases Examined</i>	<i>Showing Fibrosis</i>	<i>Per cent</i>
0 to 4	89	0	0.0
5 to 9	141	36	25.5
10 to 14	84	27	32.1
15 to 19	28	15	53.6
20 and over	21	17	80.9

This table does not apply to any one group of workers in the factories. The authors have collected also, outside of these statistical cases, records of ten deaths resulting from asbestosis, nine of which were verified by autopsy and the tenth, by repeated clinical and roentgenographic examinations. Nine of these deaths occurred from 1928 to 1929. The exposure of the ten cases varied from nine to twenty-four years.

In a study commenced in 1929, Lanza, McConnell, and Fehnel³¹ arrived at the following conclusions:

1. Prolonged exposure to asbestos dust causes a pulmonary fibrosis different from silicosis and demonstrable by roentgenogram. Clinically, it appears to be of a type milder than silicosis.
2. Definite cardiac enlargement was frequently found to be associated with asbestosis.
3. A predisposition to tuberculosis, due to asbestos dust, was not indicated although it was not known how much asbestosis may add to the mortality from pneumonia and acute non-tuberculous pulmonary infections.

Page and Bloomfield,³² of the United States Public Health Service, have given a comprehensive report on dust control methods in an asbestos fabricating plant, and Dreessen, Dallavalle Edwards, and Miller,³³ of the United States Public Health Service, have made a recent study of the asbestos industry. This study included physical examinations of 541 asbestos workers and of a control group. No cases of asbestosis were found among workers exposed to dust concentrations below 2,500,000 particles per cubic foot of air. Three doubtful cases were found in the 2,500,000 to 4,900,000 particles range. The definite cases were found where the exposure had exceeded 5,000,000 particles. The authors suggest 5,000,000 particles per cubic foot of air as a threshold limit pending further investigations.

Carders showed the highest proportionate amount of as-

bestosis, next were mule and ring spinners, and then weavers. As with silicosis, it was found that the incidence of asbestosis increased rapidly with increasing dust exposure. A fifth of those with an exposure of 50,000,000 to 99,000,000 particle years and one half of those with more than 100,000,000 particle years, had asbestosis. No free silica was found in the dust of the factories studied.

SYMPTOMS

The onset is gradual and depends upon the nature and extent of exposure to asbestos dust. There is a general impression that asbestosis comes on more quickly than silicosis and whereas the roentgenogram may show considerable involvement in silicosis with few, if any, symptoms, the reverse is apt to be found in asbestosis. Cough is variable and expectoration little or none, unless there is an accompanying infection. Anorexia is a fairly constant late symptom and Haddow³⁴ regards it as an indication to stop work.

As in silicosis, dyspnea is the most striking symptom. It is progressive and severe and is due to impaired elasticity of the lungs and to interference with blood supply. Cyanosis and clubbing of the fingers are common late in the disease as is loss of weight and emaciation.

There is a typical skin lesion found in asbestos workers known as the asbestos corn. The fine asbestos fibers penetrate the superficial layers of the skin and produce small corns or hypertrophies of the epiderm. No asbestosis bodies have been found in these lesions.

ASBESTOSIS BODIES

Asbestosis bodies are, perhaps, the most interesting feature of pulmonary asbestosis. They have been described in detail by numerous authors, notably Cooke,³⁵ McDonald,³⁶ Simson,³⁷ Gloyne,³⁸ Merewether and Price,³⁹ and Lynch and

Smith.³⁹ The description given by Cooke is abstracted as follows :

The 'curious bodies' so characteristic of pulmonary asbestosis are found in the alveoli and bronchioles and in the fibrous and necrotic areas. They measure 20 to 100 microns in length. One or both ends are bulbous, giving a clubbed or dumb-bell appearance. The shafts are either homogeneous or segmented crosswise. They are golden yellow to brownish in color. They do not stain but give a Prussian blue reaction to iron. The Bragg roentgen-ray spectroscope showed that they were not altered asbestos fibers. Most of them are soluble in strong acids and alkalis and Cooke, McDonald, and Gloyne have been able, by this means, to identify a slender mineral core derived from the asbestos fiber. Lynch and Smith³⁹ were able to see this core as a central filament with slightly greenish tinge. These bodies are not essentially iron, but contain it. They are not found in the lungs of iron workers.

These curious bodies have been induced experimentally. Simson³⁷ states that Mavrogordato gave him for examination the lungs of a guinea pig which had been exposed to asbestos dust from the mines two hours a day for fifty days. The animal died from other cause. The microscopic examination of lung structure showed a slight generalized fibrosis and the curious bodies similar to those found in the human. Gloyne³⁸ injected asbestos dust into animals and after twelve days found foreign body giant cells but no curious bodies.

From a diagnostic standpoint, it is generally agreed that the 'curious bodies' signify exposure to asbestos dust but cannot be depended upon for a diagnosis of asbestosis.⁴⁰

DIAGNOSIS

The diagnosis of asbestosis must be predicated on the history of exposure, including the essential factors of length of exposure and its extent, as well as a characteristic roentgeno-

logical appearance, together with definite symptoms that cannot be ascribed to other organic disease. Because the pulmonary fibrosis of asbestosis tends to cause cardiac enlargement and impairment, caution should be taken to avoid diagnosing, as asbestosis, cardiac or cardio-renal vascular disease in an individual merely because he worked in an asbestos plant, even though there may be asbestos bodies in the sputum. With the complications of pulmonary tuberculosis or cardiac disease present, the occupational factor may be difficult to determine even by the roentgenologist after a series of films and a post-mortem may be necessary to ascertain to what extent asbestosis, if present, contributed to disability or death. The roentgenological diagnosis of asbestosis is discussed in another chapter.

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III. ROENTGEN-RAY DIAGNOSIS

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For many years, pneumoconiosis, in spite of its often rather spectacular roentgenographic appearances, was very largely regarded in the light of an interesting disorder. More recently, however, the extreme importance of roentgenograms as a means of accurate diagnosis in indicating the exact pathological condition present in connection with pneumoconiosis has received considerable recognition. At the present time, it is generally conceded that the roentgenographic examination, properly carried through, is the most precise method at our command for determining lung pathology in a case of suspected pneumoconiosis, or for differentiating this affection from another one that may be present.

The proper roentgen interpretation of pneumoconiosis cases is dependent upon certain important qualifications. These may be enumerated as ; (1) a knowledge of the anatomy of the chest and the many physiological problems associated with its anatomical constituents ; (2) a thorough familiarity with normal roentgenographic and fluoroscopic appearances and their permissible variations therefrom ; (3) an understanding of the histology of the lungs and more especially of the lymphatic system ; (4) a clear preception of the pathology of pneumoconiosis and of all conditions which may simulate its roentgenographic appearances ; (5)

an experienced intimacy with the roentgenographic appearances of pneumoconiosis and of those which may resemble it, based upon a fundamental knowledge of the pathology represented; (6) a keen insight of the physical factors involved in the production of the suspected or alleged pneumoconiosis; and, (7) the use of the proper technic to show, to full advantage, any or all of the abnormalities present. Technic may completely enlighten, may so modify appearances as to be confusing, or may mislead entirely.

THE HEALTHY CHEST¹

Familiarity with the roentgen appearances of the healthy chest is a prerequisite for anyone who attempts to properly interpret chest roentgenograms. Unfamiliarity with normal variants and with the modifying influences of a faulty technic not infrequently results in misinterpretation, with an attendant chain of difficulties. A common example of such an error is the diagnosis of pulmonary tuberculosis, pneumoconiosis, and silicosis based on the presence of prominent truncal shadows. Within recent years, improvements in certain technical factors and apparatus have created marked variations and changes in the roentgenographic appearances of some of the chest structures. In view of such facts, therefore, a review, and where necessary a revision, of the more pertinent aspects of the chest problem, both in health and disease, seems a necessity.

The various tissues demonstrable in the chest roentgenogram will be discussed individually and in the order in which they are taken up in our routine consideration of such an examination. To follow a definite system in the study of any roentgenograms is to prevent the overlooking of, in some instances, obvious conditions.

The Soft Parts. — The approximate development of fat and musculature can readily be determined from the roent-

genogram. The density of the axillary fold frequently serves as an index of the patient's state of nutrition. A loss, as well as a gain, in weight can easily be determined by comparison of the thickness of the soft parts in those instances in which repeated chest exposures have been made. Sometimes, apparent roentgen evidence of diffuse pulmonary haziness and lung pathology may be explained by unusually thick soft parts. Examples of such instances include the well-developed pectoral folds in men, and the large breast shadows in women.

The nipples of the breasts and pigmented moles may produce shadows in the roentgenogram that simulate those of metastatic nodules or other pathological processes. Other soft tissue shadows, such as calcifications of vessels, angiomas, tumors, thyroid, and lymph nodes, may be observed in the neck and axillae. Shadows of soft tissue masses (benign or malignant) and enlarged lymph nodes can be demonstrated in the axillae, neck, or supraclavicular fossae.

The Bones. — The bony thorax can be fairly well visualized in the chest roentgenogram in the anteroposterior, posteroanterior, and lateral projections.

In the *posteroanterior* and *anteroposterior* views, with the sternal ends of the clavicles equidistant from the spine, in order that the chest be symmetrically placed, one can see the clavicles, the upper end of the humeri and the scapulae, the ribs, the cartilages when ossified, and the alignment of the thoracic spine. In a few instances, one can see the irregular appearance of the rhomboid fossae on the under surface of the inner fifth of the clavicle. This may be unilateral or bilateral and should not be mistaken for a bone lesion. Occasionally, one can see the shadow of the foramen of the supraclavicular nerve, near the upper aspect of the middle of the clavicle.² The epiphyseal development of the inner end of the clavicles can be readily demonstrated, as can epiphyseal centers in the humeri and scapulae. The normal variants of

the ribs, such as bicipital and cervical rib, should be noted. A mental note should be made as to the density of the ossification of the bones. Not infrequently, decreased bone density, caused by some condition such as parathyroid disease, or even localized areas of bone destruction from metastatic malignancy, will assist in directing the attention of the clinician to the primary cause of the patient's complaint. Recognition of erosion of the under surface of the ribs may lead to a diagnosis of coarctation of the aorta. Ossification of the costal cartilages is of very little significance. We place no reliance on the old dictum that ossification is evidence of a chronic bronchitis. In general, ossification increases with age, but occasionally it is found well advanced in thin young women. In such instances, it may be difficult, at times, to interpret structures near the midline, due to the superimposed shadows of the costal cartilages. Oblique views are of great assistance in these patients. There seems to be very little difference in the width of the intercostal spaces in the absence of pleural involvement. In older individuals with increased aeration or emphysema, the intercostal spaces may be slightly wider, but the important manifestation is that the ribs assume a more right-angled position with the spine. The alignment of the thoracic spine can be readily demonstrated. Minor degrees of scoliosis do not interfere with study of the chest, but marked scoliotic changes, on the other hand, tend to obscure the shadows of the structures in both lung fields, due to the shadows of the spine on one side and the closeness of the ribs on the other.

In the *lateral view*, one can see the ribs, portions of the shoulder girdle, and the thoracic spine. The spine is readily visualized, but superimposed shadows of lung structures are confusing in evaluating bone detail. In adults, the body of the vertebrae may, or may not, show hypertrophic changes, and even calcification of the nucleus pulposus. Usually there

is a gentle, double curve to the spine, concave posteriorly above and slightly convex, or straight, below. In kyphotic individuals, the anteroposterior diameter of the chest may be greater than the transverse. The vertical diameter of the body of the vertebrae and the intervertebral discs increases slightly from above downward.

The Pleura. — The parietal pleura is so thin that it does not cast a shadow which can be seen in the roentgenogram unless it is thickened and diseased or obliterates some portion of the costophrenic sulci. Very slight thickenings of the parietal pleura may be suspected in those instances when there is interference with the costal expansion, especially when the process is unilateral.

In anteroposterior roentgenograms of the chest made with the Potter-Bucky diaphragm, the reflection of the pleura, ligaments and muscles over the vertebral column produces a straight line shadow on each side of the spine. Disturbance in this normal straight line may occur in spinal caries, bone tumors, some spinal cord tumors that emerge from the spinal canal, and pleural collections occurring in the vicinity of the vertebrae.

In this country particularly, where one finds many references to the apical cap (thickening of the visceral pleura over the apices of the upper lobes), it is a wise roentgenologist who recalls that there are other structures whose shadows create a similar appearance in a similar position. Subcostal muscles, in the oblique views, may be misinterpreted as thickened pleura.

The roentgen appearances of these various anatomical structures have been emphasized by Knutsson,³ whose observations are briefly abstracted. On the posterior portion of the thoracic wall, the internal surfaces of the ribs are covered with subcostal muscles. In the lateral aspect of the thorax, the muscle lining disappears. The narrow muscle layer on

the inner aspect of the ribs produces a shadow, seen only in the oblique view, with one exception, which gets thinner from above downward and disappears entirely in the region of the costophrenic sulci. In pleurisy, on the other hand, the pleural thickening usually gets thicker from above downward,

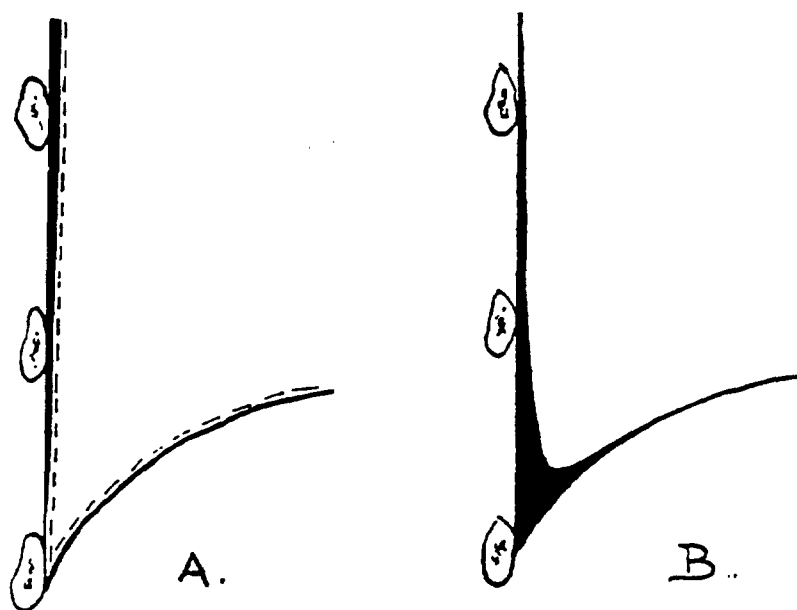


FIGURE 1

a. Schematic presentation of intercostal muscles demonstrating the decrease in the thickness of the muscles as they approach the diaphragm. *b.* Schematic presentation of pleural thickening which becomes more prominent near the diaphragm. (Knutsson.⁸)

and may obliterate the sulcus. (Figure 1) The thickness of the muscle shadow depends upon the muscular development of the individual. In the upper thorax, the muscles extend laterally, far enough to be seen in the posteroanterior and the anteroposterior projections. This structure is called the 'companion shadow of the second rib.' Mesial to the angle

of the second rib, there are no subcostal muscles, and the shadow here is produced by the connective tissue. The first rib, likewise, has a companion shadow which is produced by a sheet of connective tissue interposed between the posterior part of the dome of the pleura and the first and, to a certain degree, the second rib. The following structures are situated in this interspace; the last cervical ganglion of the sympathetic, the supreme thoracic artery and vein, and the first thoracic nerve. The 'companion shadows of the first and second ribs,' as well as those produced by the subcostal muscles in the oblique views, are symmetrical on the two sides. This fact should be of assistance in differentiating these shadows from those created by pleural thickening. The pleura does not calcify under normal circumstances.

The visceral pleura covers the different lobes of the lungs and ordinarily does not cast a shadow that can be seen in the roentgenogram, except in the instance of the azygos and other accessory lobes. At times, straight and very thin shadows of the interlobar lines can be seen in the chests of healthy individuals. We have felt that such pleural shadows represent a permanent thickening of the pleura from a pre-existing infection. Furthermore, such slight thickening of the interlobar pleura is capable of producing shadows because the roentgen rays are projected in their axial plane. This conclusion is supported by the frequency with which a shadow of the interlobar pleura is found in one roentgenogram of a stereoscopic pair and not in the other. Any interlobar thickening not observed in an axial plane will, nevertheless, produce shadows in the roentgenogram, which, when superimposed upon those of the lung fields, will create an increased density. If such a density is not recognized in its true relation, it may be mistaken for an intrapulmonary lesion. In disease, the visceral pleura rarely, if ever, becomes as thick as the parietal pleura. Clark⁴ believes that, when adhesions have

formed, the increase in the thickening of the pleura in the lower chest and around the heart, as compared to that occurring in the upper chest, is due in part to the relatively more powerful movements of structures such as the domes of the diaphragm, the flaring of the ribs, and the pulsation of the heart. The visceral pleura is reflected over the mediastinal structures and does not cast a recognizable shadow. In disease, however, it may produce changes which can be observed in the roentgenogram.

The Mediastinum. — In roentgenograms of the chest, we prefer an exposure that will show the shadow of the trachea down to, and including, its bifurcation. In the posteroanterior or anteroposterior roentgenograms, with the patient placed correctly, the shadow of the trachea in the neck will be seen in the midline. Deviation to the right or left of the midline above the level of the clavicles may result from rotation of the head, and must be differentiated from displacements of other origin. Below the clavicles, the trachea continues in its midline position until it reaches the level of the aorta, where it deviates slightly to the right in roentgenograms exposed during the inspiratory phase of respiration. In adults, in roentgenograms made in the expiratory phase, the trachea is displaced slightly to the right throughout its thoracic position. In all instances, the shadow of the lumen of the trachea is fairly uniform, although its lumen decreases slightly from above downward. Just above the trachea, the laryngeal portion of the upper respiratory pathway is narrowed and produces a funnel-shaped shadow, with the apex of the funnel cephalically placed.

The tracheal cartilages, especially when ossified, aid greatly in the demarcation of the trachea.⁵ Careful inspection of the shadow of its contour will not infrequently show evidences of compression or deviation that may prove significant. As a rule, the lumen of the trachea is larger in men than in

women. Its caliber is not uniform throughout, becoming progressively narrower as it approaches the bifurcation.

The level of the bifurcation of the trachea varies with age. In adults, it usually divides at the level of the intervertebral disc between the fourth and fifth, or as low as that between the fifth and sixth, vertebral bodies. It is well to remember, however, that the relationship in the cadaver may vary considerably from that in the living. Likewise, the level of the roentgen tube may cause discrepancies in the level of the bifurcation and the carina.

The angles made by the bifurcating bronchi with the trachea vary with age and from one another. In adults, the angle of the right bronchus is approximately 20 degrees and the left, 40 degrees. The eparterial bronchus sometimes branches directly from the trachea.

The shadows of the trachea and the right and left bronchus can readily be seen in the oblique and lateral projections of the chest. In the lateral view, the trachea may be followed from the suprasternal region down to its bifurcation. During inspiration, the entire trachea moves downward and slightly forward, unless fixed by mediastinitis.

The soft tissues flanking and surrounding the trachea have been discussed by Dann.⁶ His investigators have helped substantially in identifying the structures producing these shadows. The right border of the supracardiac portion of the mediastinum is formed by the superior vena cava and innominate artery, while the most lateral margin of the mediastinal shadow is almost invariably formed by the superior vena cava. (Figure 2) The more medial density is a combination of the vena cava and the ascending aorta, which lies slightly anteriorly and to the left.⁷ The space between the vena cava and aorta, as described by Dann⁶ in his studies in young cadavers, is rarely seen roentgenographically. However, a markedly distended aorta may approach the right border of



FIGURES 2 AND 3. Healthy adult chest. *a.* superior vena cava ; *b.* trachea ; *c.* hilum or root zone ; *d.* pre-aortic space ; *e.* a composite shadow, made up of the inferior vena cava, the phrenopericardial ligaments, and possibly also fluid in the cul-de-sac.

the superior vena cava and, in some instances, actually forms the right border of the superior mediastinum. When enlarged shadows of this general configuration are seen, one should consider a substernal tumor, aneurysm of the innominate artery, and diverticulum of the esophagus. Roentgenoscopic observations of the changes in the shadows during the grunt of forced expiration and a study of the esophagus may assist in differentiating between substernal thyroids, innominate aneurysms and esophageal diverticula.

The left border of the supra-aortic portion of the superior mediastinum is formed by the soft tissue shadows of the left common carotid and subclavian arteries. The recurrent laryngeal nerve lies to the left of the trachea. The shadow of the aortic arch is usually seen extending beyond the left border of the spine. With increasing age, its knob-like contour becomes more prominent, until it is most pronounced during the arteriosclerotic period of life. The left posteroanterior oblique view of the chest permits excellent visualization of the aorta and, when used in conjunction with roentgenoscopy, gives valuable information concerning aortic dimensions and elasticity. The aorta follows a more sagittal course in narrow-chested individuals than in flat-chested persons. Increases in prominence in the outline of the shadow of the aortic knob may result from syphilis or hypertension, while an absence or decrease in the outline of the knob suggests either coarctation or right aortic arch.

The pulmonary artery is seen best with the patient in the anteroposterior or right antero-oblique positions. It lies just to the left of the base of the aorta, nestled within the confines of the aortic arch. Sometimes it is seen better after a cough, which serves to separate it from the aorta.⁸

In recent communications, O'Kane, Andrew and Warren⁸ and Margolies⁹ reemphasize the importance of the roentgenographic and roentgenoscopic examinations in determining

the size and shape of the heart. In addition to these estimations, the roentgenologist must concern himself with the pulsations and the mobility of the heart and any abnormal densities. There are many conditions, such as congenital deformities of the chest and acquired affections, such as pneumothorax and hydrothorax, which may exert profound influences upon the heart. Examinations must be made in the anteroposterior, posteroanterior, oblique, and lateral views, in order to assure adequate study of the heart. The most important view is the left antero-oblique view, but the degree of rotation and the optimum position for a roentgenographic demonstration must always be guided by the preceding roentgenoscopic impressions.

Under normal conditions, the heart in thin individuals lies in the vertical position, whereas in the more stocky, broad-chested person, it assumes a more transverse position. Furthermore, patients with round chests more frequently have a rounded heart than patients with flat chests.¹⁰ In the latter type of patient, the heart has a more oval circumference.

Normally, with the patient facing in an anteroposterior direction, the right border of the heart is formed, from above downward, by the right auricle, the inferior vena cava and the cardiophrenic ligament.⁹ (Figure 3) In some patients, paracardial fat pads form part of the cardiac border. Roentgenographically, however, the shadow of the outline of the heart may sometimes be seen as a density within the less dense fat pad. The left cardiac border is formed by the pulmonary artery, the left auricle, and the left ventricle.⁹ With the patient in the lateral position, the anterior border of the cardiac silhouette is formed, from above downward, by the base of the aorta, the pulmonary conus and the right ventricle. The posterior border is formed, from above downward, by the arch of the aorta, the pulmonary artery, the left auricle, the left ventricle and the inferior vena cava. The cardiac silhouette,

with the patient in the right anterior oblique position, is formed by the right auricle and the inferior vena cava on the right side, with the aorta, the pulmonary artery, the pulmonary conus, and the right ventricle, mentioned in order, from above downward, forming the left border. With the patient in the left anterior oblique position, the right border is formed by the superior vena cava, the right auricle and the right ventricle, whereas the left border is made up of the silhouette of the aorta, the pulmonary artery, the left auricle and the left ventricle, mentioned in order, from above downward. These cardiac contours bear a definite relationship both in size and degree of expansion with one another.

Normally, the average anteroposterior diameter of the heart is about two-fifths of the transverse diameter of the chest.¹⁰ While empirical judgment of the size of the heart is used constantly to decide increases in cardiac size, it is by no means satisfactory. The cardiothoracic ratio, diagonal measurements and cardiac area determinations are much more satisfactory. As a rule, however, the average anteroposterior diameter of the heart is less than three-fourths of the average transverse diameter of the heart. Realizing that the average anteroposterior diameter of the heart in females is 8.7 cm. and in males, 7.8 cm., one can obtain a fair impression as to size.¹⁰ The oblique diameter of the heart is about one-third larger than the average anteroposterior diameter.

In the lateral view, the attachment of the pericardium may be seen extending down from the posterior edge of the heart shadow. (Figure 3) It is seen as a faint shadow with its posterior border straight, or slightly concave, extending to the superimposed diaphragmatic shadows. This shadow is a vantage point for observing slight pericardial effusions,¹¹ which tend to distend the cul-de-sac and distort its normal concave appearance.

During deep inspiration, especially in tall, thin persons,

the right and left phrenopericardial ligaments exert a pull upon the domes of the diaphragm, by virtue of their attachments to the diaphragm, which is synchronous with cardiac pulsation. As this frequently occurs in healthy individuals during deep inspiration, it must not be confused with the abnormal diaphragmatic tug produced by adhesions between the domes of the diaphragm and the pericardium, which may be seen during the expiratory phase of respiration also. Chances for error are minimized by roentgenoscoping patients in the horizontal posture, the patient lying on the right side and the rays projected anteroposteriorly. This allows the mediastinum to drop toward the right, permitting the examiner to see more of the left dome of the diaphragm and its connections with the heart. Phrenopericardial tugging observed in this position may be evidence of adhesions. With the patient lying on one side, the entire mediastinum shifts to the lower side. This evidence can be recorded when the rays are projected in a horizontal plane.¹²

The horizontal position may also be used to advantage in identifying pericardial effusions. One must be well acquainted with the normal appearances of the heart and aorta in this position before using it for refinements in diagnosis. In this position, the transverse diameter of the heart is larger than in the erect position and the width of the shadow of the aorta is greater. This may be due partly to the elevation of the diaphragm. Roesler¹³ thinks that there is also an actual increase in the cardiac volume in the horizontal position. Occasionally, a swallow of barium, with the patient in the horizontal position, will outline a cardiac or aortic aneurysmal dilatation, which might otherwise be overlooked.

Under normal conditions, the space between the pericardium and epicardium is very small. It was our good fortune to examine a healthy student with air in the pericardium, as a result of an accidentally inflicted wound. The space between

the pericardium and the epicardium, as outlined by air, was almost negligible when compared with the pneumopericardio-gram of tuberculous pericarditis.

While practically every portion of the heart may become calcified in disease, the calcification associated with old age must be considered physiological. In the past, roentgenologists constantly overlooked calcifications within the heart.¹⁴ Calcified valves may be seen in the anteroposterior view near the middle or lower half of the heart. The aortic valves are usually seen overlying, or just to the left of, the spine and are, therefore, more adequately seen on slight rotation. Mitral calcifications, however, lie well to the left of the midline, and when seen in the lateral projection, are situated more posterior than the aortic calcifications. Roentgenoscopically, these shadows have to and fro movements, going toward the apex during systole and returning during diastole.

The Diaphragm. — The right dome of the diaphragm is about 1.5 cm. above the left dome, in most instances. When transposition of the viscera occurs, the opposite obtains. In certain other instances, the shadow of the left dome of the diaphragm is either on the same level or above that of the right dome. We presume that, in the absence of eventration or herniation, such instances may be regarded as normal variants. The splenic flexure of the colon is usually highly placed in such individuals, or there may be a large collection of air in the fundus of a cascade type of stomach, which may be responsible, to some degree, for the position of the diaphragm.

In the anteroposterior and posteroanterior projections, the right and left halves of the diaphragm are dome-shaped. The attachment of the mesial portions of the domes is on a higher level than its attachment to the ribs. This results in a costophrenic sulcus of varying depths. The domes of the diaphragm are regular in this plane and, during inspiration, any peaking may be the result of either pleural adhesions or

localized areas of inelasticity of the lung structures. The domes of the diaphragm may be wavy at their outer aspect or produce a shadow of multiple arcs over its surface, due to variation in attachment or hypertrophy of the constituent muscle bundles. This has been described as individualization of the costal components. Occasionally, in the posteroanterior and lateral projections, one finds a rounded hump of the mesial aspect of the right dome of the diaphragm. This observation has been reemphasized¹⁵ as being due in the healthy individual to a weakness of the anteromedial portion of the diaphragm as compared to that of the posterolateral portion. In addition, the inferior vena cava may play a part in preventing downward excursion of the diaphragm. Tumors of the liver and subphrenic lesions may produce an appearance that simulates such a condition.

An anomalous enlargement of the right lobe of the liver will produce a shadow in the lateral view that may simulate that of an interlobar collection.¹⁶ In the lateral view, the domes are higher anteriorly and the posterior costophrenic sulcus is much lower than the anterior, rendering it possible for a relatively large localized pleural collection to escape detection in the dorsoventral roentgenogram. The upper surface of the shadows of the right and left domes runs from before backward. These shadows may be parallel or may cross each other, either anterior or posterior to a point that corresponds to the middle of the domes. During the inspiratory phase of respiration, the shadows of the domes lose their dome shape and become relatively straight.

The movement of the domes of the diaphragm is extremely variable. The most correct impression of its movement is obtained during ordinary respiration. Frequently, efforts to study diaphragmatic excursion during forced respiration are confusing, due to the influence of costal and abdominal muscles. The movement of the domes of the diaphragm is usu-

ally more pronounced in men than in women. The domes are higher and the excursion is greater in the horizontal posture than in the erect. Therefore, anyone interested in detecting slight restriction in movement should examine the patient in the erect posture. Lateral roentgenoscopy of the diaphragmatic domes, with the patient lying on the back and the rays projected from the opposite side, is difficult because of the increased dome shape of the diaphragm and the encroachment upon the costophrenic sulci. If the patient is studied roentgenoscopically in the horizontal plane while lying on one side, and the rays projected ventrodorsally, the dependent diaphragm will become more cephalically placed and will have a greater excursion. These observations have also been noted by Pierson and Newell.¹² Such positions or modifications thereof allow a study of the costophrenic sulci and aid in differentiating between thickened pleura and pleural collections.

The Hilum Shadows. — The structures in the hila include the bronchi, the pulmonary arteries and veins, the tracheobronchial and aortic lymph nodes, nerves, and connective tissue. The lymph nodes are classified as follows; (a) bifurcation nodes, located between the right and left main bronchi; (b) tracheobronchial nodes, situated above the bronchi and along the trachea; (c) the aortic nodes; and (d) the paravertebral nodes. Sukiennikow¹⁷ states that there is a greater number of lymph nodes on the right side. Miller⁵ says that there is a greater number of nodes associated with the eparterial bronchus than with the bronchi supplying the middle and lower lobes.

The hilum shadow is irregular in outline with ill-defined borders. The density of these structures, as seen in the roentgenogram, varies considerably with the type of roentgenographic exposure. In the arbitrary properly exposed roentgenogram, the shadow has varying densities and is not homo-

geneous. It would seem that if roentgenologists could agree upon what a properly exposed film consists of, varying densities could be regarded as significant observations. We have placed considerable reliance upon individual ability to see varying densities in the hilum shadow, i.e., the bronchial shadow is different from that of the vessels. When the density of the shadow is more or less homogeneous, it is our impression that one can be fairly certain that abnormal changes have become manifest.

The National Tuberculosis Committee ¹¹ has described the limits of the hila as follows :

'On the right side, the normal shadow may be said to extend, from above downward, from the top of the right main bronchus to approximately a point where the right lower-lobe bronchus, below the origin of the middle-lobe trunk, begins to subdivide into its lower-lobe branches (or trunks). This includes the anatomical hilum, the greater portions of the larger bronchi, and some lung tissue in front and behind. The extent of this hilum shadow from above downward is approximately two interspaces and a rib.

'On the left side, the pulmonary artery passes above the left bronchus, and from above downward, the left hilum shadow may be said to extend from the upper border of the pulmonary artery down to approximately a point where the left lower-lobe bronchus begins to subdivide into its lower-lobe branches (or trunks). Such a hilum shadow on this side is slightly shorter than the one on the right and is slightly less than two interspaces and one rib in length.

'While the top of the right bronchus is higher than the left, if the tops of the hilum shadows be limited in the manner just described, they are on a plane approximately level.

'The thickness of the hilum shadow in the anteroposterior direction is a matter that requires further study, and probably largely from the lateral view. Measurements from lateral

views are very uncertain and subject to considerable personal equation. One should learn to acquire the idea of depth as well as length and width when interpreting hilum shadows.'

In the lateral view, the shadows of the hila are superimposed and it is impossible to define their limits, even within wide margins, because of so many variable factors, such as target film distance, length of time of exposure, and disturbed shadow outline due to transmitted pulsation from adjacent vessels. We suggest, however, that one can develop an individual impression as to their normal extent by studying roentgenograms of the chest of healthy individuals, made with a Potter-Bucky diaphragm in an almost lateral projection, with the patient so placed as to prevent the superimposition of the hila. Such films allow one to see shadows of the varying densities of the hila, and, if a thorough understanding is obtained, it becomes possible to determine deviations from the normal with a high degree of precision.

In the dorsoventral and ventrodorsal roentgenograms, the outer boundary is regarded as normal when it lies within the inner zone of the lung field. Everyone familiar with the interpretation of variations of hilum shadows realizes how difficult it may be. Oblique and lateral views often assist in detecting glandular enlargements that might otherwise escape notice.

Calcified lymph nodes occur frequently in the hila of healthy individuals. They represent, as a rule, evidence of a pre-existing active tuberculous process. Calcification of a lymph node does not necessarily mean that the node may not contain viable tubercle bacilli which, under certain conditions, are capable of multiplying and setting up an active disease. In adults, we have felt that nodes that are calcified in a relatively homogeneous manner are probably healed, whereas those in which the calcification is punctate and appears as a mottled shadow have a much greater potentiality



FIGURE 4

Calcified tracheobronchial lymph nodes. Note the irregular calcification of the nodes. They are somewhat punctate in type. These are examples of nodes in which infection may become reactivated.

for reactivation. (Figure 4) Chests of children with calcified nodes of this type should probably not be regarded as healthy. While it is true that calcified nodes are usually regarded as evidence of past tuberculous infection, it is entirely possible for them to be due to some other process.

It is important to evaluate the dense, circular shadows, due to vascular trunks obtained in an axial plane, and appearing in the roentgenogram as densities similar to those cast by calcified nodes. These shadows are readily seen roentgenoscopically and, should any doubt arise as to their nature, slight rotation of the patient will permit identification of their true nature. Calcified shadow densities do not disappear on rotation, whereas vascular shadows do.

The Zones. — The chest has been variously divided into different zones for convenience of description of the single film. The classification in most frequent use is that which divides the one side of the thoracic cavity into 3 zones; the inner or hilum, the middle or truncal (or vascular markings), and the peripheral, which contains the finer subdivisions of the vascular tree in which the only shadows visible consist of fine linear markings.

In stereoscopic films, there is very little occasion to use such a classification, except so far as the various zones have different capacities for expansion.¹⁸ The hilum zone expands and contracts less than the truncal or vascular zone, and the peripheral zone has more expansile and contractile power than the truncal or vascular zone.

The Lobes. — Ordinarily, the right lung is divided into 3 lobes and the left, into 2. Not infrequently, there is an accessory lobe on the right side called the azygos lobe.¹⁹ This lobe is recognized by the fine pleural line which extends from the apex of the lung downward, to end as a dense oval shadow near the upper aspect of the right border of the superior mediastinum. The dense oval shadow is due to the

azygos vein, which maintains its foetal position and carries with it a reflection of the parietal pleura, down to the point of entrance into the superior vena cava. The size of the azygos lobe is quite variable, depending upon the position of the vein.

More recently, other accessory lobes have been described in roentgenologic literature.²⁰ An accessory lobe may occasionally be found at the base of either lung, either partially or completely formed. These lobes are variable in size and represent a division of the lower lobe. They can be recognized by the presence of interlobar pleural shadows, extending upward from the mesial portion of the domes of the diaphragm toward the hila. This lobe may occasionally be the site of a pathological process and, therefore, readily recognizable. On one occasion, we observed two lobes only, on the right side. The observation was later confirmed by post-mortem examination.

The position of the various lobes is readily observed in those instances where either interlobar shadows are visible or after introduction of an opaque medium into the bronchi. It should be emphasized, however, that the size and extent of the lobes are quite variable. On occasions, the lower lobes may extend very high posteriorly, so high that physical signs, and even shadows in the roentgenogram, may be misinterpreted as representing lesions in the lower portion of the upper lobe.

The Vascular Markings.— Extending from the hilum on either side are heavy shadows passing into each lobe. These shadows are more pronounced in the lower lobes and, in roentgenologic literature, are described as truncal shadows. It has seemed to us, however, that such a terminology conveys an impression that a bronchus is responsible for, or at least associated with, these visible shadows. From the work of Miller,²¹ Miller^{22, 23, 24, 25, 5} and Greineder,²⁶ such an inter-

pretation may not be true. For years, roentgenologists attempted to localize foreign bodies and pathological lesions according to the distribution of these shadows as they passed into each lobe. It was recognized that only very large bronchi were visible, and these were restricted, for the most part, to the hilum zone. On the other hand, the roentgenologist thought that the truncal shadow, as seen and described in the roentgenogram, consisted of bronchus, artery, vein, nerves, lymphatics, and connective tissue. Wherever one of the large branch shadows was seen, there was thought to be a bronchus. We know now that such is not the case. Miller²⁵ has shown that 'the pulmonary artery follows, in all of its subdivisions, the subdivisions of the bronchial tree. As each main branch of the pulmonary artery arches over its corresponding stem bronchus, it comes to occupy a position posterior (dorsal) and slightly lateral to the bronchus. The relation of the main trunks of the pulmonary vein to the bronchi is quite different; they are situated anterior (ventral) and mesial to their stem bronchi and in their ultimate distribution are situated as far removed from bronchi as possible.'

Miller,²⁵ interested in another phase of the same subject, made similar observations, as did Greineder,²⁶ with even more striking demonstrations by tomography. If one can be certain that veins, as a rule, have a larger diameter than the arteries, it is our belief that the shadows of the larger veins can be differentiated from the larger arteries in roentgenograms of excellent detail made during the diastolic phase of the cardiac cycle.

We appreciate that the above discussion may be considered academic and of no practical importance so far as the distribution of the bronchi is concerned, because of the improved methods for delineating bronchi after the instillation of opaque media and the increased use of the bronchoscope. Nevertheless, it is our firm belief that too many roentgenolo-



FIGURE 5

Illustration of a roentgenogram showing the vascular markings in the right upper lung field. The pulmonary artery and vein were ligated in this instance. In the left upper lung field, the blood has been allowed to drain out. Note the difference in size of the vascular markings. (Reproduced by permission from Miller.²¹)

gists are misinterpreting normal variations of the vascular markings in terms of pathology. (Figure 5) Miller,²¹ in 1919, recognized and warned us of such a possibility. Now, it has become necessary for industry to have pre-employment as well as follow-up examinations and, unfortunately, many roentgenologists, upon whom the burden of the roentgen examination has fallen, are, *all too frequently, misinterpreting vascular shadows as fibrosis, and thereby imposing an unnecessary hardship on both the employee and the employer.*

Vascular shadows are otherwise confusing in interpretation, particularly with regard to the dense, circular shadows, due to blood vessels recorded in an axial plane, which may simu-

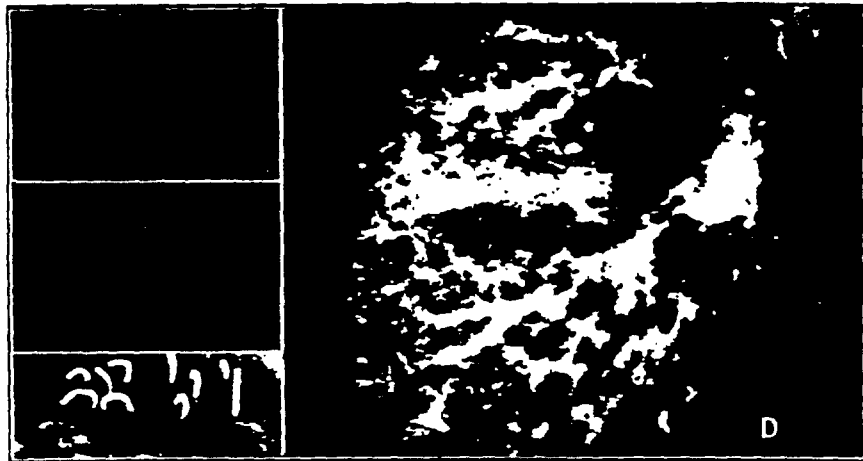


FIGURE 6

Curved paraffin cylinders in *c.* were examined to demonstrate differences in appearance when rays were directed in one plane, *a.* and in another plane, *b.* Note the differences in density of the cylinders in *a.* and *b.* *d.* vessel shadows in roentgenogram of inflated normal lung. (Reproduced by permission from Miller.²¹)

late an appearance of thoracic calcium²¹ and, in the more peripheral portions of this zone, may produce an appearance of beading or early nodulation, when the roentgenogram is made during the systolic phase of the cardiac cycle. The appearance of beading may simulate that of a pathological process, such as that seen in silicosis, baritosis, or a mycotic infection. Stereoscopic dorsoventral films may not be sufficient to identify the exact nature of the shadows, but films exposed in an oblique view and rotation of the patient during the roentgenoscopic examination will usually be of aid. If the dense shadow is due to a blood vessel, slight rotation will cause the shadow to disappear; if due to a calcified lesion, the shadow will remain. Furthermore, the blood vessel shadows are circular and homogeneous, whereas a calcified lesion

may be irregular and less homogeneous, or even mottled. (Figure 6)

Miller⁵ called attention to another structure, the ring-shaped cartilage which occurs where bronchi leave the main stem bronchi. This cartilage may produce a dense, circular shadow in the roentgenogram.

McPhedran and Weyl^{27, 28} have emphasized the importance of synchronizing the roentgen exposures of the chest during the late diastolic phase of the cardiac cycle. They have performed important experiments in an effort to evaluate this phase of the general problem, but further investigation is necessary. This work, thus far, serves to show that vascular displacement and vibration cause considerable blurring of roentgenographic detail. Likewise, kymograms illustrate clearly what a marked influence the cardiac pulsation exerts on the lung structures close to the heart.

The Bronchi. — The first subdivision of the right bronchus is the *right upper lobe bronchus* (eparterial), which comes off at an angle of almost 90 degrees and passes above the pulmonary artery. This bronchus gives off a branch, the vertebral, which passes upward to the apex of the lung parallel to the spine. This bronchus gives off branches which extend anteriorly and posteriorly. The upper lobe bronchus gives off another important branch which is designated as the second interspace bronchus, because it comes off behind the level of the second interspace in front. Likewise, this bronchus gives off anterior and posterior branches.

The portion of the right bronchus between points from which the upper and middle lobe bronchi come off is termed the *stem bronchus*. Foreign bodies frequently lodge in this region.

The *middle lobe bronchus* comes off the stem bronchus anteriorly. It divides into many branches, but there are 2 main divisions, as a rule; (a) the branch which passes down-

ward and anteriorly, and (*b*) the branch that passes upward and laterally.

The *lower lobe bronchus* bifurcates into several main branches which have been variously described as the anterior, middle, posterior division branches in the lower portion of the lower lobe, and the apical branch in the upper portion. All of the main subdivisions give off many branches. Disease of the mesial and posterior division bronchi produce shadows that border upon the shadow of the heart in dorsoventral roentgenograms. Further identification of the bronchi can be obtained in the oblique and lateral projections.

The *left upper lobe bronchus* (hyparterial) passes below the pulmonary artery. Its subdivisions are similar to those of the right side, except that there is a branch which goes to the lingual tip of the upper lobe which is that portion in front of the heart.

The *left lower lobe bronchus* has a longer course than that of the right side before it gives off any subdivisions, otherwise it and its subdivisions are similar to those of the right side.

The Lung Fields. — Medicine owed a debt of gratitude to William Snow Miller whose life has been dedicated to the study of the anatomy of the lung. No discussion of the healthy lung can possibly be complete without frequent reference to Miller's work, and much of that which we shall discuss has been gleaned from his reprints and recent book.⁵

In the discussion of the healthy lung, we have approached its consideration from the standpoint of the smallest functioning unit in the lung, the primary lobule. It is obvious, therefore, that the following discussion is of more importance histologically than roentgenologically.

As the bronchial tree is followed to its ultimate divisions, there occurs a lack of the regular bronchial outline and the gradual appearance of an air passage, whose walls are lined

with variable, irregularly placed air spaces. These terminal air channels are called the respiratory bronchioles, which, in turn, divide into smaller branches known as the alveolar ducts. These alveolar ducts are smaller than the respiratory bronchiole from which they spring and are lined by more alveoli than the aforementioned bronchiole.^{22, 24} The alveolar ducts, in turn, lead into from 2 to 5 spheroidal cavities called atria. The atria, in turn, open into numerous air sacs around the periphery of which are situated the alveoli.

The bronchial musculature forms sphincters about the channels leading into the atria and around the alveoli which line the smaller bronchioles.²⁵ These sphincters probably play an important role in the muscular spasm known to occur in asthma. Normally, air passes unimpeded by these sphincters. With muscle contraction, the air channels reduce in size preventing the egress of air, which produces an increase in residual air with the formation of emphysematous lobules.

The structures described above comprise the anatomical unit or primary lobule of the lung, which may be defined as a ductulus alveolaris, the air spaces connected with it, and all their associated vessels, nerves, and lymphatics. The primary lobule has the form of a truncated pyramid, with its base directed toward the periphery of the lung.^{22, 24} Its air passage (ductulus alveolaris) usually enters the primary lobule at the side rather than at its apex. Primary lobules vary in size from 0.45 mm. to 0.845 mm., the larger units situated in the peripheral and basilar portions of the lung. It is obvious that these lobules are not seen in the roentgenogram. It is only barely possible that one sees, roentgenographically, the larger, secondary lobules, which are made up of 50 to 250 of these primary units.

The primary lobules receive relatively little blood from the bronchial blood vessels.^{22, 24} The pulmonary artery, on the other hand, follows the course of the respiratory bron-

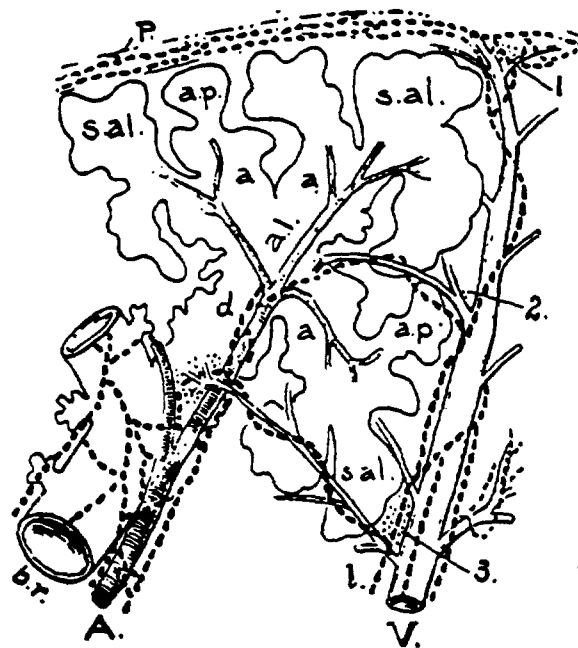


FIGURE 7

Composite diagram (modified from Miller) of the primary lobule lymphatic system, indicating the primary distributions or accumulation points for dust which will lead to predominant phases of pneumoconiosis, b.r., respiratory bronchiolo; d.al., alveolar ducts; a., atria; s.al., sacculi alveolare; a.p., alveoli; a.p., aveoli opening into respiratory bronchioles and alveolar ducts in close relation with the origins of peribronchial and perivascular lymphatics; A, branch of pulmonary artery, accompanying the air passages; V., branch of pulmonary vein in interlobular septum; P., pleura; P.B., peribronchial lymphatics; P.V., perivascular lymphatics; 1, 2, 3 and other dotted areas, lymphoid deposits. (By previous permission of Dr.W.S.Miller.)

chiole and the alveolar duct into the center of the primary lobule.⁵ Each atrium is supplied by a branch of the pulmonary artery, which then divides into enough smaller tributaries to supply the alveolar sacs. The pulmonary vein, in contra-

distinction to the pulmonary artery, courses along the periphery of the primary lobule at a distance from the air channels.⁵ (Figure 7) Its branches arise from the pleura, the end of the alveolar duct, the walls of the alveolar sacs and the points of division of the bronchi and bronchioles.

The lymphatics of the lung may be divided into the deep lymphatics and the superficial lymphatics.²⁴ The deep lymphatics include all of the channels which follow the bronchial tree and its associated blood vessels toward the hilum. The superficial lymphatics lie within the pleura.

The bronchial lymphatics in the primary lobule are 3 in number, with no lymphatics present in the air spaces as vessels. The lymphatics of the pulmonary artery lie between the artery and the bronchiole, anastomosing with 1 of the 3 bronchial lymphatics. These, in turn, unite with others to form a rich network running toward the hilum. The lymphatics of the pulmonary veins follow the course of the veins from the various points of origin described previously, with one exception. Since no lymphatics lie in the walls of the air sacs, there are no lymphatics along the veins arising from their walls.

The pleural lymphatics form a rich network which drains into the lymph nodes in the hilum. There is some evidence supporting the belief that the superficial pleural lymphatics, covering the diaphragmatic surface of the lower lobes, drain into the abdominal pre-aortic nodes by way of the ligamentum pulmonale.²⁴ The numerous valves in the superficial lymphatics point in no particular direction. The valves are less numerous in the deep lymphatics and lie close to the hilum in the large vessels, allowing flow in the direction of the hilum only. Similar valves lie in some of the deep lymphatics close to the pleura. These valves point toward the pleura, allowing lymph flow into the superficial pleural lymphatics without permitting a reflux of superficial lymph into the deep set of lymphatics.

In addition to the lymph vessels, the primary lobule contains small masses of lymphoid tissues, which may be found in the walls of the primary lobule.²⁹ Besides these distinct lymphoid masses, one can identify a sheath-like dispersion of lymphoid tissue along the pulmonary artery and along the air channels. This faintly defined sheath of lymphoid tissue is rarely found in the lungs of humans under thirty years of age. With the advent of middle life, however, these lymphoid deposits become more conspicuous until they are quite prominent at approximately sixty years of age. The lungs in healthy individuals over sixty frequently show the artery and bronchus completely surrounded by this lymphoid tissue.²⁹ Perhaps this accounts for the prominent linear markings frequently associated with advancing age, markings which are usually lacking in children and young adults. It is important to know the distribution of these lymphatic masses and lymphoid networks as they are the centers about which disease processes often commence.

Considerable evidence has been accumulated substantiating the belief that massage, passive motion, capillary wall injury, increased venous and capillary pressure, and decreased blood proteins accelerate lymphatic flow.^{30, 31} The importance of these findings may be appreciated when one realizes that the rate of lymph flow and the progress of phagocytes within lymph vessels are negligible when the part being studied is at rest. Without the former observations, one would be at loss to explain the rapid appearance in the hilum glands of foreign particles ducted into the lung. In one set of experiments, ink particles were demonstrated within the hilum nodes several minutes after their injection into the lung.³⁰ Drinker and Field³⁰ demonstrated that phagocytosis was not entirely responsible for the transfer of foreign particles into the perialveolar spaces. Perhaps alveolar pores really do exist, allowing free interchange of such material.^{32, 33} This, in part,

helps explain the rapid appearance of foreign bodies in the hilum nodes, as the finding obviates complete dependency upon phagocytosis in the transfer of foreign material from the alveolus into the perialveolar tissue.

Dolley and Wiese³⁴ recently found that unilateral pneumothorax produced a decrease of about 45 per cent in the lymph flow from the lungs. Such data, correlated with those previously referred to, would seem to indicate that the respiratory excursions of the lungs and the massaging action of the pleura play an important role in pulmonary lymph flow. It also seems reasonable to conclude that the rate of lymph flow might vary in different portions of the lung, depending upon differences in respiratory excursions in various portions of the lung. That such differences in the amplitude of pulmonary expansion do occur seems well substantiated by the work of Keith, Miller, Scott, et al.

While there is good evidence indicating that lymph constantly courses through the lung toward the hila, no absolute proof concerning the magnitude of this lymph flow is available. Drinker and Field³⁰ were unable to demonstrate any appreciable flow in their animal experiments. If little is known concerning this particular factor, certainly less is known of the true function of the lymphatics.

Effect of the Phases of Respiration on the Roentgen Appearances in the Chest. — During inspiration, the first five ribs rotate up and out, carrying the sternum forward and upward. These movements increase the anteroposterior dimensions of the chest, thereby expanding the upper lobe in an anteroposterior direction, allowing an influx of air.¹⁸ The ribs maintain a constant relationship with the upper lobe in its anteroposterior excursions, with the result that the impressions of the first five ribs may frequently be seen in post-mortem specimens on the anterior surface of the upper lobe. Such markings, however, are unusual in the lower lobes. During inspira-

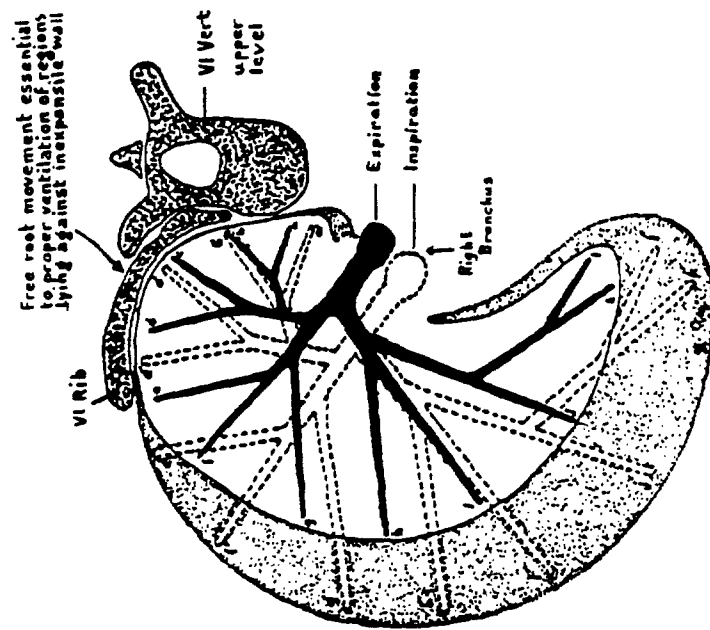
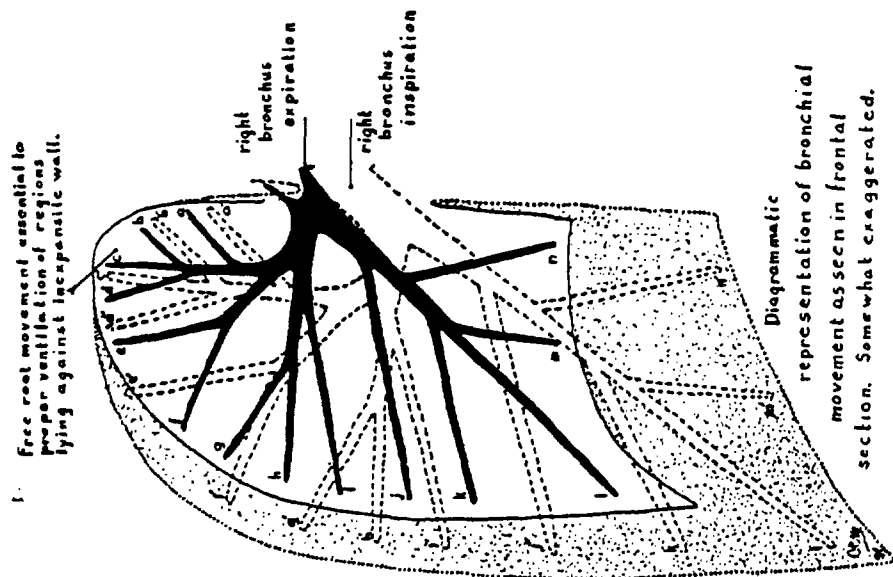


FIGURE 8. See caption on page 99.

tion, the lower lobes are expanded by the descent of the powerful diaphragmatic muscles, which increase the depth of the chest, and by the action of the lower intercostal muscles, which increases the transverse diameter of the chest.¹⁸ These forces are much greater than those influencing the upper lobes. Because of this, the lower lobes are drawn downward during inspiration, thereby massaging its pleural surfaces against the long axis of the ribs, which explains the absence of the rib markings previously referred to as being commonly found in the upper lobes. The evidence concerning the formation of rib impressions in the upper lobe is indirect. Clark⁴ has called our attention to the relative lack of pigmentation of the lung surface where rib impressions are found, whereas more pigmentation is found in the lung tissue adjacent to the intercostal muscles. Such evidence is in favor of Keith's¹⁸ ideas concerning the mechanism of respiration. Clark⁴ also believes that these observations support the premise that the massage effect of the ribs is an important factor in lymphatic flow. This seems reasonable in view of the fact that other portions of the lung, such as the lung around the heart, the tip of the lung extending into the costophrenic sulcus (where it is massaged by the diaphragm), and the posterior surfaces of the lower lobes, are less pigmented than the inside of the lung and the lung surfaces where massaging action is minimal. The summation of these inspiratory forces produces expansion in a forward, downward and outward direction, with considerably more expansion taking place in the lower lobes than in

FIGURE 8

Diagrams showing the differences in expansion in the various portions of the lung. Note decreased expansion in upper lobe and in peravertebral gutter. (Reproduced by permission from Macklin.³⁸)

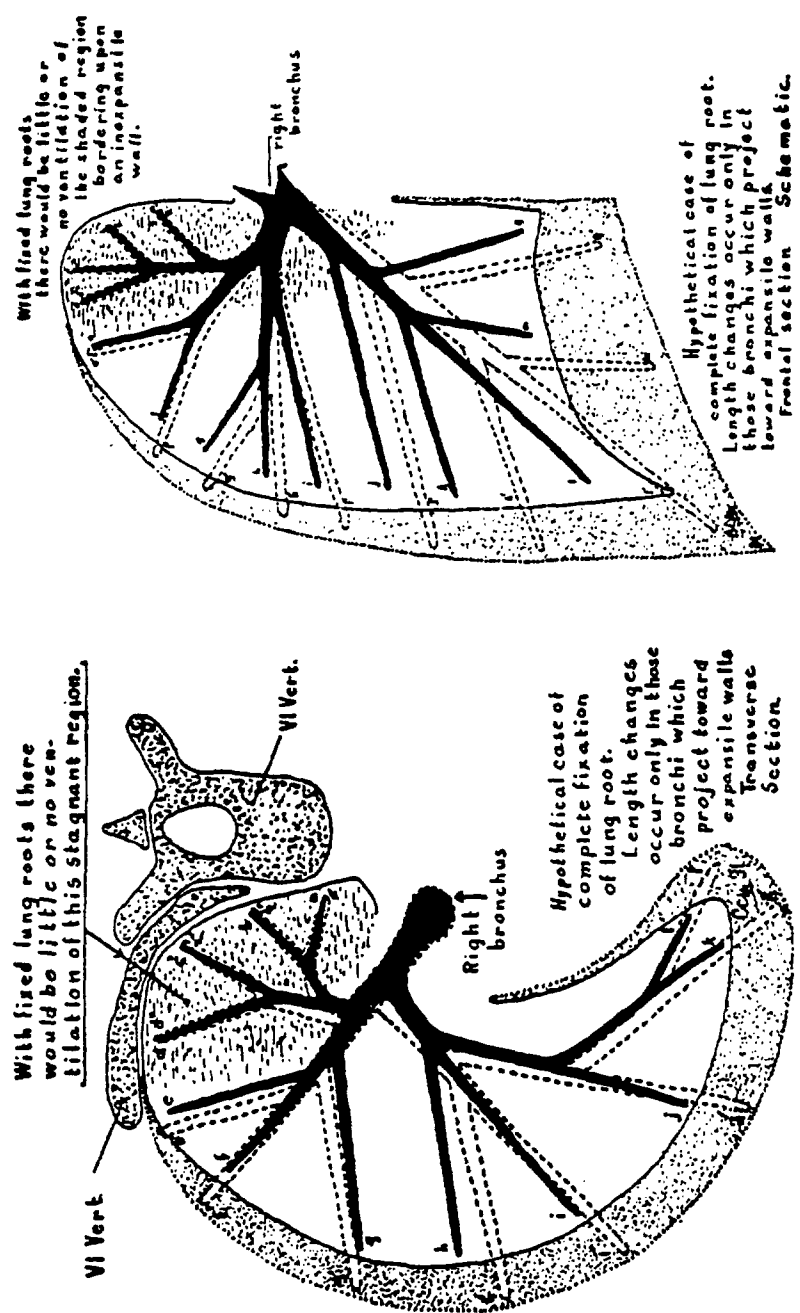


FIGURE 9. See caption on page 101.

the upper.¹⁸ The trachea and its ramifications also feel the effects of these inspiratory forces as the trachea and hila move downward and forward, while the bronchial tree and the vascular structures become elongated.*^{35, 36} Keith¹⁸ has stated that, when more is learned about normal respiration, the lobation of the lung and the great fissures will be found to be of functional significance.

Working against these inspiratory mechanisms are the heart, the vertebral column and vertebral portions of the ribs. These portions of the chest are antagonists to pulmonary expansion by virtue of their anatomical characteristics.^{18, 35, 36} Being relatively fixed structures, they cannot move to allow the nearby lung to expand. (Figures 8 and 9)

In like fashion, the apex of the upper lobe does not expand as fully as the lower portion of the upper lobe, which is not inhibited by the peculiar conical shape of the upper thoracic cage. One can conclude, therefore, that the amount of air entering the lower lung field and periphery of the lung is greater than that which enters the upper lobes or hila.

Attention should again be directed to the importance of respiratory movements upon the lymphatic flow and its possible influence upon the distribution of a pathological process. While the evidence is purely deductive, it seems reasonable

* Keith and Miller believe that the bronchial tree spreads and elongates during inspiration. Macklin's work, while not conclusive, does not support the spreading of the bronchi, although it does show the elongation very well.

FIGURE 9

Diagrams showing differences in expansion in the various portions of the lung in a case with infection. The shaded portion of the diagram in the upper, mesial and posterior portion of the lung field represents the area of infection. Note the marked decrease in the expansion of the lung. Compare with Fig. 12. (Reproduced by permission from Macklin.³⁰)

that adequacy of lymphatic flow and degree of respiratory mobility go hand in hand. Perhaps this might account for the frequency of upper lobe tuberculosis and for the mid-lung field changes of silicosis. Perhaps it might also account for the clear peripheral and paracardiac zones in the lung fields in patients with pronounced changes due to silicosis with a quiescent infection.

Roentgen Technic. — Considerable data have been accumulated showing that the diagnostic value of chest roentgenograms depends upon certain film characteristics which are determined by the technical factors used for the examination.^{37, 38} For this reason, certain fundamental technical requirements have been defined which, when adhered to, have produced a high quality of chest examination. Briefly, these factors may be summarized as follows :

1. *Time.* Exposures made in from $\frac{1}{30}$ to $\frac{1}{10}$ second are essential to overcome lack of detail resulting from vascular motion. Synchronization of the roentgenographic exposures with the late diastolic phase of the cardiac cycle further reduces vascular blurring. This method, if coupled with some means of determining full inspiration, would be ideal, as one could then be reasonably sure that slight vascular changes in different films of the same patient were, or were not, due to physiological variations.^{37, 38}

2. *Distance.* Focal-spot film distances between 4 and 6 feet are most satisfactory, as distances under 4 feet produce considerable distortion, while exposure made at distances over 6 feet are not sufficiently superior to warrant the expense of bulky high tension apparatus.

3. *Screens.* Screens should be chosen for excellence of detail, although high-speed intensifying screens are of considerable assistance in avoiding blurred images. Screens must be kept clean and care taken that contact is maintained throughout their surfaces.

4. *Voltage.* The roentgen tube voltage should be so chosen that the densities in the diagnostic areas of the film will lie between the limits of 0.4 and 0.6. This depends upon the other physical factors employed, but, as a rule, will vary from 50 to 80 kv. (peak).

5. *Current.* The choice of the tube current also depends upon each of the factors previously considered. Bearing these factors in mind, that tube current should be chosen which will produce proper roentgenographic densities.

6. *The Roentgen Tube.* The line or band focus tubes have been recommended as they combine the detail produced by a small effective focal-spot with the durability of the broad focal-spot tube.

7. *Film Processing.* Unless a time-temperature method of development is employed, in which all factors are carefully calibrated and controlled, it is useless to follow the technic described. Warm solutions, impure chemicals, over or under developing will ruin any film, no matter how perfect its exposure may have been.

In addition to these factors, it is necessary to use accurate methods for measuring patients. One must also be careful to make all measurements through the same anatomical points. An impulse timer, a cathode current stabilizer, suitable indicating meters, adequate power supply, full wave rectification for the high tension equipment, and 1 kv. per step auto-transformer control, are all essential for obtaining roentgenographic results which can be repeated on different occasions.

Stereoscopic examinations made in the anteroposterior or posteroanterior directions are desirable in chest roentgenography. The gross exaggeration of vascular markings and other abnormal densities, seen in single flat films of the chest, is avoided and properly evaluated by stereoscopy, which places these questionable areas at their right depth. Lateral,



FIGURE 10. See caption on page 105.

oblique, recumbent, or any other position, determined roentgenoscopically, will add valuable information.

Most chest cassette changers are equipped with some type of fixation device, such as a windlass and binder, which encircles the patient's back for immobilization. After immobilization has been accomplished, care must be exercised to determine whether or not the patient is rotated. Even slight rotation will cause a difference in density of the two sides of the chest. Such differences in density have been regarded, by some, as evidence of pleural thickening or hypoventilation. (Figure 10)

ROENTGENOLOGICAL STAGES OF PROGRESS⁴⁹

Roentgen appearances represent pathological changes. At first, in the study of pneumoconiosis, it seemed logical that the stages of progressive changes should be based upon a sequence of advancement which could be shown by such a simple means as the roentgenogram. Clinical groupings must necessarily be based upon such phenomena as increasing dyspnea and incapacity for work, together with certain clinical signs. Even these, when the first classifications of pneumoconiosis were based upon similar industries, rather closely followed the roentgenographic demonstration of the progression of pathological changes.

It is unfortunate that the roentgenological groupings of the various countries have differed. In the United States, the roentgenological classification of Lanza and Childs,³⁹ who

FIGURE 10

a. healthy chest; *b.* same patient. Note the increased density over the right hemithorax which is due to slight rotation. This was produced by the supporting strap when the patient was placed against the film changer.

divided the condition, as shown by the roentgenogram, into 3 well-known stages, designated numerically, was followed until comparatively recent years, when it no longer seemed feasible. In South Africa, where the disease received probably the greatest amount of study in the early years of our more extensive knowledge of silicosis, three pathological stages were first recognized — early, intermediate and advanced silicosis.⁴⁰ The roentgenographic appearances ascribed to these were quite similar to those of our own three stages. Later on, the condition was *legally* divided into three *clinical* stages — ante primary, primary, and secondary. Roentgenographic appearances were grouped to conform to these in showing progression or extent. Compensation laws elsewhere, as in Ontario, have been based, to a considerable degree, upon the highly successful legislation in force in South Africa. As a result, the roentgenological classification of silicosis has followed the same designations of stages as that of the legal classification, which is quite natural. With no federal laws in this country directly governing the condition, and no clinical classification of the disease, the roentgenological classification first adopted has held priority.

In order to make comparisons with the groupings used elsewhere, it is necessary to give a brief résumé of the roentgenological stages of silicosis as described in this country.

First Stage. This stage has been characterized roentgenographically by a definite increase in the prominence and extent of the hilum shadows, an increased prominence and thickening of the trunk shadows, and a greater prominence of the linear markings of the peripheral zone. Enlarged glands may be interpreted in the hilum regions. This stage must be subject to certain modifications. Frequently, appearances are detected in the central portions of the lungs, usually more or entirely on the right side, consisting of a slight haze, with or without fine lines. Within these areas,

there occasionally become manifest a few small, faint, hazy spots, which are probably the beginning of lymphoid deposit enlargement. In many cases, the definite appearances of the first stage, as just described, are not apparent. In a flat roentgenogram, these hazy areas often have the appearance presented by a very small female breast or a pectoral muscle in a male, but in the wrong locality. This manifestation is probably due to the beginning of the interstitial fibrosis and of the visibility of a few of the individual nodules. One who has observed the effects of dusts in many occupations will find that individuals who follow certain dusty trades do not present progressive appearances that run true to form or that can be made to conform to the usual general stages, for the reason that there are types of the condition peculiar to certain industries, and, possibly, to certain individuals.

The general manifestations of this first stage are not characteristic of pneumoconiosis alone, but may be simulated by many other conditions, especially, passive congestion, acute or chronic respiratory infections, chronic bronchial catarrh and bronchiectasis. Moreover, the hilum and trunk shadows are subject to considerable normal variations in appearance in the adult.

As a numerical stage, this one implies, theoretically at least, an early period in progression, yet individuals may remain in this phase for years, or indefinitely. As the manifestations of a typical first stage, with prominent hilum and trunk shadows and linear markings, are not characteristic of pneumoconiosis alone, I am inclined to doubt the fairness of giving this stage a definite medicolegal status, or of using it as a basis for compensation.

Second Stage. This most typical stage has been characterized by the distinctive distribution of small, rounded densities, varying in size from a pinhead to a pea, throughout both lungs. (Figure 11) The nodules appear first on

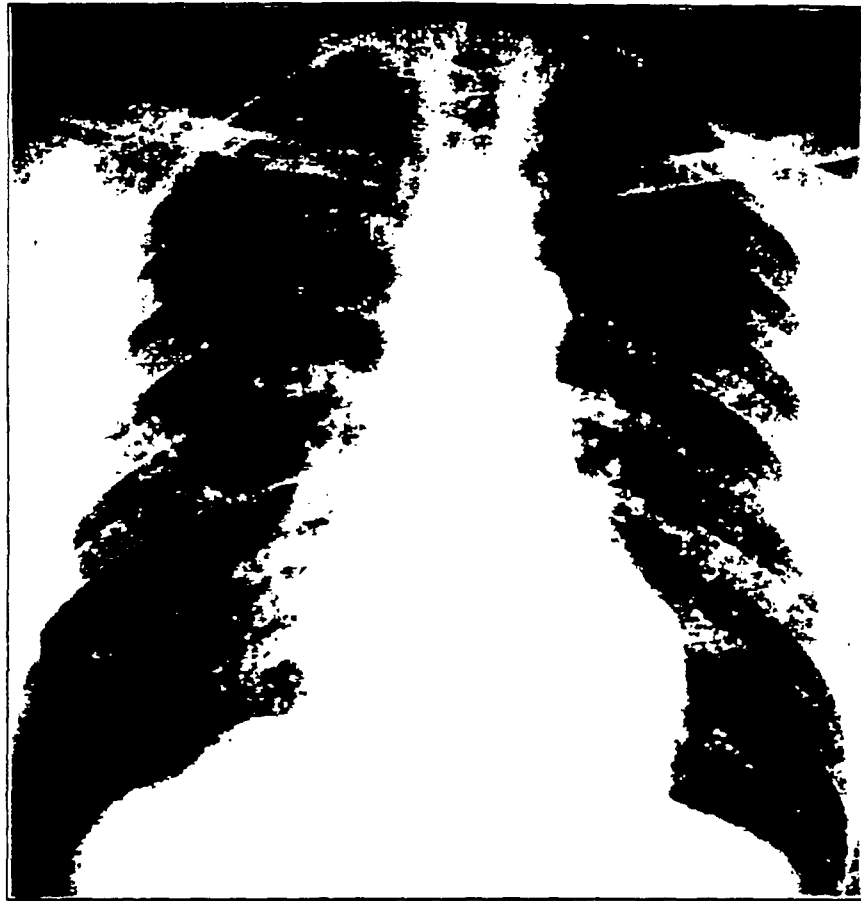


FIGURE 11

Simple silicosis showing the uniform distribution of small, round densities throughout both lungs, in a hard coal miner.

the right side, around the root of the lung, and usually become quite perceptible here before they are seen on the left side. This predominance is apparent until the distribution becomes quite general throughout both lungs, and then there

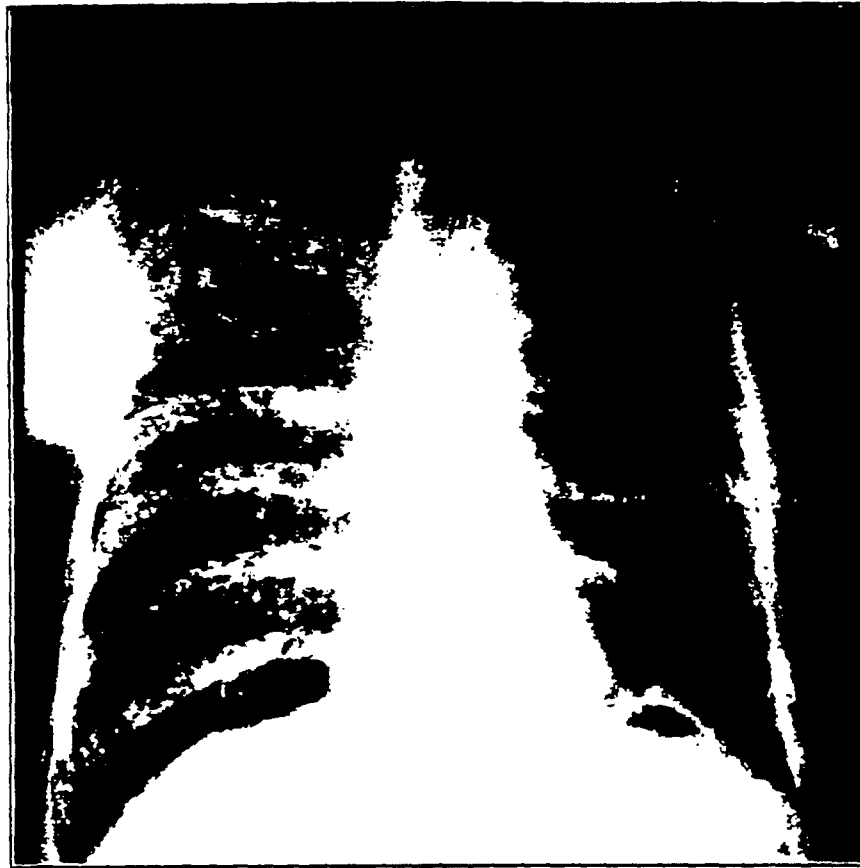


FIGURE 12

Silicosis with infection and beginning coalescence of the nodules in both lung fields, especially in both upper lobes, in a hard coal miner with an active tuberculous infection. There is a small pneumothorax in the right side.

is no appreciable difference. The spots are always more numerous around the hila and less in the basilar portion of the lungs. The densest and most sharply defined nodules are usually found in association with the more rapidly fibrosing

dusts, such as those to which many abrasive tool grinders are exposed. There is always more or less evidence of diffuse fibrosis present, as a continuation of that often visibly started in the first stage, and as a forerunner of the essential process of the third. It may not be apparent because of emphysema. When not apparent, its presence may be suggested by lack of lung expansion and diaphragmatic excursion, or sharp peaking of the domes. Theoretically, one would expect this second stage nodulation to be superimposed upon first stage appearances, but very often, the latter are not apparent. They may be obscured by emphysema, but this is not the sole cause.

Third Stage. This stage has been characterized by manifestations due to a predominance of diffuse fibrosis, which may present three fairly definite and distinct appearances, although all of them may be observed in the same case:

(1) The larger nodules of the second stage, where most prevalent, either may coalesce into very much larger and irregular masses, or be found close together with more or less haze between them. It may be difficult in such cases to differentiate between second and third stages, except by a lack of expansion and diaphragmatic excursion, and the presence or absence of diaphragmatic irregularities. (Figure 12)

(2) A more or less diffuse fibrosis, somewhat similar in appearance to the fibrotic process representing the late result of an extensive bilateral chronic pulmonary tuberculosis. Usually, definite nodules are still present, although not always.

(3) The presence of massive fibrotic areas presenting the appearance of extensive pulmonary consolidations. In the late period of the stage, there is usually one such area on each side, rather symmetrically located subapically, but sometimes unilateral, or more extensive on one side than the other. (Figures 13 and 14) In an earlier period of the stage,



FIGURE 13

Anthracosilicosis with infection. Some of the lesions are healed. Other lesions, such as the one in the right lower lobe, extend to the periphery of the anterior chest wall. Note that the massive lesions in the upper lung fields have a clear zone of lung around them. Another interesting feature is the absence of any evidence of nodulation.



FIGURE 14

Close-up of a massive or conglomerate lesion in the right upper lobe. The lesion is centrally placed and does not extend to the periphery at any point. From the roentgen standpoint, this lesion is regarded as silicosis with a quiescent infection.

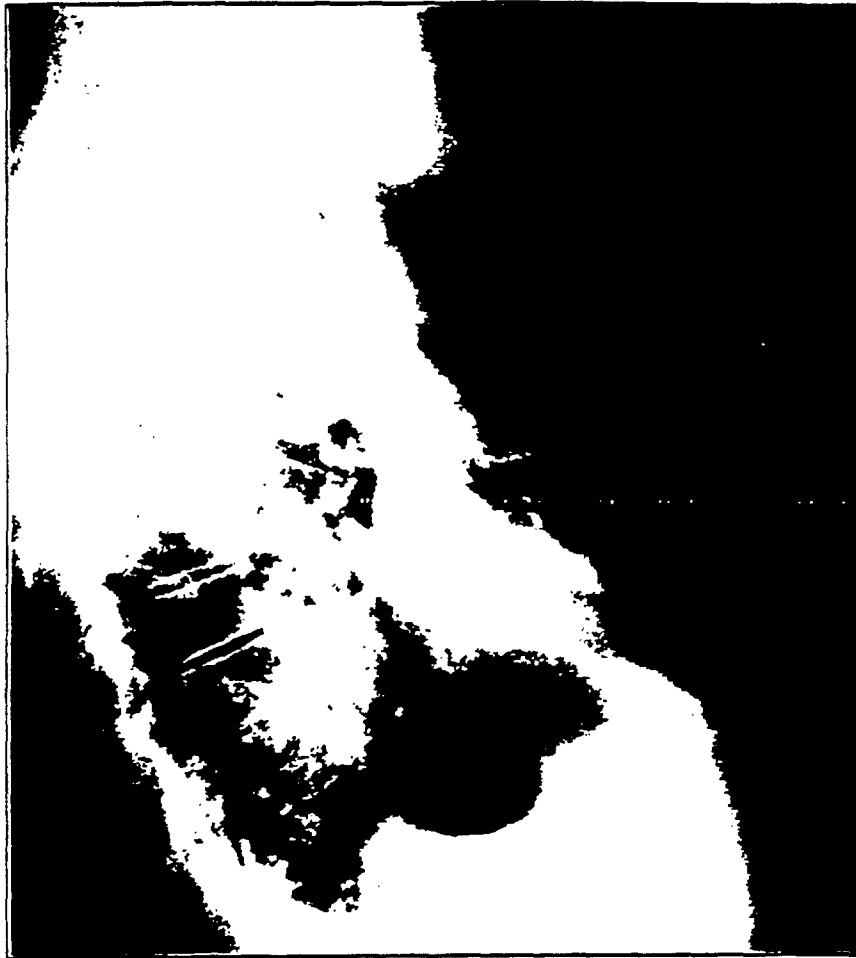


FIGURE 15

Lateral view of the chest of an individual having anthracosis. Note that the major portion of the lesion is in the upper half of the lower lobe and the lower half of the upper lobe.

when small, they may appear quite asymmetrical. Lateral roentgenograms have shown us that many or most of these



FIGURE 16

Massive lesions in a hard coal miner. Note the symmetrical position of the masses.

subapical masses are in the apices of the lower lobes, and numerous autopsy reports in the literature have confirmed this location. (Figure 15) Often, associated with these

manifestations, dense, fibrous bands are found extending in various directions, but prominently downward, and frequently causing marked diaphragmatic deformities. They often resemble thickened trunks. The heart and vessels are quite likely to be displaced when these bands are present. Diaphragmatic excursion is very much restricted or entirely absent. (Figure 16)

Other Classifications. The present general classification of South Africa is as follows :

- | | |
|---|----------------|
| 1. Rather more fibrosis than usual | |
| 2. More fibrosis than usual | |
| 3. More fibrosis full | |
| 4. More fibrosis to commencing fibrosis | } Ante primary |
| 5. Commencing fibrosis | |
| 6. Commencing fibrosis plus | |
| 7. Commencing to early | } Primary |
| 8. Early commencing | |
| 9. Early to medium | |
| 10. Medium fibrosis | } Secondary |
| 11. Medium to advanced fibrosis | |
| 12. Advanced fibrosis | |

Complicated classifications are more apt to become popular in the examinations of individuals in a single industry than when several industries are studied, as seems to have been the case in connection with the South African gold miners, and as was the case with Jarvis ^{41, 42, 43} in his studies of the Barre granite cutters. They are, no doubt, admirably adapted for use when practically the entire work of an in-

dividual or board is restricted to the examination of silicotic persons, especially in one industry, but they are hardly practicable for roentgenologists in general, who are more favorable to simple classifications.

In the formulation of the most excellent Workmen's Compensation Act of Ontario, especially in respect to silicosis, the clinical classification of the South African Board has been embodied. This section of the Ontario Act reads as follows :

(g) — (a) 'Silicosis' shall mean silicosis of the lungs (a fibroid condition of the lungs caused by the inhalation of silica dust).

(b) A person shall for the purpose of this Act be deemed to have or to have had silicosis, —

- (I) In the ante-primary stage when it is found by the Board that the earliest detectable specific physical signs of silicosis are or have been present, whether or not capacity for work is or has been impaired by such silicosis ;
- (II) In the primary stage, when it is found by the Board that definite and specific physical signs of silicosis are or have been present, and that capacity for work is or has been impaired by that disease, though not seriously and permanently ;
- (III) In the secondary stage, when it is found by the Board that definite and specific physical signs of silicosis are or have been present, and that capacity for work is or has been seriously and permanently impaired by that disease, or when it is found by the Board that tuberculosis with silicosis is or has been present.

The 1925 South African law defines three legal stages of silicosis practically identical with the above Ontario specifications. In view of the greatly altered aspect of the disease in South Africa, it has become necessary to recognize addi-

tional *clinical* groups of cases, which Watkins-Pitchford⁴¹ summarizes somewhat as follows:

(a) *Simple Silicosis*. The condition in an individual who remains free of overt tuberculosis. It is non-progressive and produces but little effect upon the health. From the pathological standpoint, these subjects present numerous small, inert nodules of dense fibrous tissue, symmetrically distributed. Each nodule is sharply defined from the surrounding tissue, and is so well encapsulated as to be easily shelled out. They vary in size from 3 to 4 millimeters and are rarely over 6 millimeters. If closely aggregated, they may produce a certain amount of disability, mainly shortness of breath. The roentgenological examination is more reliable than the clinical one.

(b) *Tuberculosis with Silicosis*. This is a progressive and serious disease associated with larger nodules unsymmetrically distributed and composed of young connective tissue elements among which tubercle bacilli are found. Adjacent nodules are frequently aggregated into masses. There are 2 classes of cases in this group: (1) The individual may have had simple silicosis at first, followed by the development of unsymmetrical tuberculous lesions (*tuberculosilicosis*). This change may occur after many years. (2) The tuberculous lesions may be manifest first, followed by the silicotic ones in close association (*silicotuberculosis*). Here again, the roentgenographic findings are important. In tuberculosilicosis, the hitherto uniformly mottled areas are now invaded by one or more unsymmetrical areas of shadow with mottling around them. The Miners' Phthisis Act now provides separately for these two general groups of cases, as has been the case in Ontario.

At the meeting of the First International Conference on Silicosis⁴² at Johannesburg, in August, 1930, the three clinical stages recognized locally were accepted by the Conference.

They were as follows: *First Stage* — Respiratory symptoms slight, few or no physical signs and capacity for work little impaired. Roentgenograms show the linear shadows increased and present discrete shadows of nodulation. *Second Stage* — All physical signs are increased. The nodular shadows are increased in number and size and show a tendency toward confluence. *Third Stage* — All signs and symptoms are greatly accentuated, and there is a total loss of working capacity. When tuberculosis is present, the stage classification must be based more or less upon a loss of working capacity than upon physical signs and roentgenographic appearances. It was recommended that an internationally comparable roentgenographic technic and terminology be adopted, and that a further study be made of the correlation of roentgen appearances, pathology and symptomatology of silicosis with or without tuberculosis.

In 1931, Dr. Pancoast and I decided that it was more rational to dispense with any attempts to employ general stages of progression in any of the ways so far discussed and to substitute a pathologic-roentgenological classification, which could be combined with the clinical aspect peculiar to any one industry, or with all of them. Those who have studied a single phase of pneumoconiosis in one occupational group, such as asbestos workers, coal and hard rock miners, granite cutters, and sand pulverizers, for example, have found it most difficult to apply any definite numerical order of progression by stages such as that used for other occupations. Likewise, those who study a large number of occupations and industries find it difficult to adhere to a general division into stages, such as that, for example, as had its origin in the study of hard rock miners, without making exceptions or creating certain variants. This becomes more complicated with each new occupation studied. Pneumoconiosis implies a condition of fibrosis, occurring, so far as we

know, in only one general way, but with certain variations dependent upon uncertain or unknown influences.

Now, the clinical phenomena must, of necessity, depend upon obvious forms of pathological changes. Furthermore, roentgenographic appearances portray these pathological changes as soon as they become macroscopic, or reveal their influences while the actual changes are still microscopic. Therefore, it seemed wiser to substitute, for a numerical progression of pathological changes typical for a few of the occupations, a general pathological classification which would include the typical and the variants, and, at the same time, be applicable to both clinical and roentgenological studies for each industry or occupation.

A study of the diagrammatic representation of the lymphatic system of the lungs, such as that of Miller,^{46, 47} and an application of the pathological features of the condition to this serves to indicate that there are practically three simultaneous primary distributions or accumulation points for dust, which correspond to later possible manifestations of fibrosis. (Figure 7) These have been summarized as follows: (1) Blockage of the peribronchial and perivascular lymphatic systems, with later intensification of trunk shadows, linear markings, and pulmonary lymph nodes; (2) Lodgment in lymphoid deposits, with subsequent development of nodular fibrosis; (3) Thoroughly interstitial fibrosis following a rapid block of lymphatic channels. To these are added the pleural side-track, which is more or less common to all cases and needs no special classification.

From a study of various industries, it seemed evident that fibrosis might exhibit *predominance* in any of these distributions in individual occupations. For example, in coal mining, the driller may show predominance of the nodular type, whereas the man in the breaker may exhibit only the first stage for many years; in hard rock mining, the nodular type

predominates until diffuse fibrosis supervenes ; in the granite cutter, the interstitial type predominates, although the nodular may, exceptionally, do so ; in the asbestos worker, the interstitial type is characteristically predominant. Interstitial predominance, rapidly progressing, will develop into diffuse fibrosis without nodular predominance. And so we might run through all the fibrosis producing dusty occupations.

The following pathologic-roentgenological classification was offered as a suggestion to overcome the existing confusion :

- | | |
|--|---|
| 1. Peribronchial-perivascular-lymph node predominance | rapid
slow |
| 2. Early interstitial predominance
(interferes with diaphragmatic movement) | with nodular appearance
without nodular appearance
rapid or slow |
| 3. Late or advanced interstitial predominance | |
| 4. Nodular predominance | non-progressive
progressive |
| 5. Advanced diffuse or terminal fibrosis | conglomerate nodular type
interstitial type
massive fibrosis type |

It was thought that many individuals might show, or occupations be characterized by, various combinations of these types ; and that, in one industry or occupation, there would be a sequence of predominant appearances which would correspond closely to the clinical aspect and progression of

the disease. This has not been true in my experience, however, even in studies of individuals exposed to pure silica without contaminating dusts.

We found it difficult to understand, or to explain, all of the underlying factors in the rapidly developing interstitial fibrosis. Gardner,⁴⁸ however, offered an explanation. He stated that the obstruction in the deep pulmonary lymphatics diverts the lymph flow and dust cells toward the pleura, where counter-resistance is met. Consequently, dust cells pass through the walls of the lymph vessels and accumulate in the surrounding connective tissue at a point where the two lymphatic systems communicate. In support of the theory first advanced,⁴⁰ of the choking of lymphatic channels by dust cells and the contraction of their lumen, which has been open to criticism, Gardner believed that the point of actual obstruction could be in the pulmonary lymph nodes, which would produce stasis in both deep and superficial lymphatics. He further suggested that the previous condition of these lymph nodes, as to changes induced by childhood tuberculosis and other agencies, might have some influence on the rapidity of onset and progression of silicosis. It is my feeling that active infection may exert an important influence upon the roentgen manifestations.

These roentgenological phases of the condition, which we have described previously,^{49, 50, 101} may be briefly summarized as follows :

1. The perivascular-peribronchial-lymph node aspect due to the relaying of phagocytosed dust to the pulmonary lymph nodes and their subsequent enlargement and ultimate partial fibrosis to the gradual enlargement of lymphoid deposits along the course of lymph vessels and the subsequent thickening of these vessels and stasis of contents. This is characterized roentgenographically by increased prominence of the hilum and trunk shadows and linear markings. This appear-

ance is by no means characteristic of pneumoconiosis alone, and even if it does indicate the condition, the phase is absolutely not incapacitating. It, together with a barely perceptible appearance of macroscopic nodules, corresponds to the so-called first stage.

2. The nodular aspect is due to the gradual enlargement of lymphoid deposits and their coalescence into quite apparent macroscopic nodules symmetrically scattered throughout both lungs. This corresponds to the so-called second stage. It is conspicuously absent in many instances, especially when the silica intake is rapid.

3. The interstitial type of the condition results from a hilumward and pleuralward block in the lymphatics and the escape of dust phagocytes in large numbers into the interstitial interalveolar tissue and subsequent fibrosis. It appears as a faint homogeneous haze, first on the right side, then on the left. If the silica intake is comparatively slow, it may accompany the perivascular-peribronchial-lymph node aspect, but if more rapid, it may be associated with the nodular type, or may progress without the latter directly into the terminal stage of the condition without any evidence of nodulation. The unprotected or inadequately protected sand blaster, sand pulverizer, and sandstone abrasive worker have been among those especially prone to present this rapid interstitial aspect.

4. Terminal and incapacitating silicosis is characterized by two general appearances, — a terminal diffuse fibrosis of a conglomerate nodular type, one which is quite similar in appearance to a generalized chronic fibroid tuberculosis, and the terminal stage characterized by large consolidated areas.

In 1935, another classification or silicosis terminology was suggested because it was felt that, if objective terms, descriptive of the type of pathological change, could be generally adopted, material progress would result. The committee's

report,⁵¹ given below, will certainly be revised from time to time as experience directs. Abstracts from the report are as follows :

It should be distinctly understood that the tabulation which follows applies only to *silicosis*, that form of pneumoconiosis resulting from the inhalation of dust with a high silica (SiO_2) content *. Other forms, like asbestosis, are excluded from this consideration because their pathology is essentially different from that of silicosis.

The tabulation contains two columns ; on the left, the roentgenological appearances ; and on the right, the corresponding pathological lesions. There is further subdivision to describe the appearance of ; (1) the healthy lung, (2) the uncomplicated silicotic lung, and (3) the lung of silicosis with infection. The changes described under the first division are those compatible with a state of good health ; and, while they *may* be produced by the inhalation of relatively small amounts of silica dust, they are not sufficiently characteristic or advanced to substantiate a diagnosis of silicosis. Similar or identical appearances may also result from the inhalation or nonsiliceous dusts, from certain infections, from cardio-vascular disease, and from certain other rare conditions. The changes involved are, for the most part, confined to the lymphatics and perilymphatic connective tissues and do not affect the parenchyma of the lung. Since, by definition, silicosis is a disease characterized by nodular fibrosis in the parenchyma of the lung, these alterations, even when they may have been caused by inhaled silica, do not constitute a basis for a diagnosis of silicosis. The second group covers the discrete and conglomerate nodular fibrotic reactions of sim-

* Some of the nonsiliceous components of certain industrial dusts seem to modify the pathological reaction, but the character of shadows cast by these modified lesions is not sufficiently defined at the present time to include them in the tabulation. Later, when more information has accumulated, certain other terms may have to be included.

ple silicosis. The last group deals with silicosis complicated by infection. In the majority of instances, the infecting organism is the tubercle bacillus, but the classification is sufficiently broad to include other types of infection. Certain criteria by which one attempts to differentiate various forms of infection will be discussed.

Roentgenological Appearances

Histological Appearances

HEALTHY LUNGS AND ADNEXA

- | | |
|--|--|
| 1. <i>Healthy lungs.</i> — As defined by the N.T.A. Committee report. | 1. Essentially the normal tissues of the vascular tree, the mediastinum, the bronchi, and trachea. |
| 2. Irregular exaggeration of the linear markings, with possibly some beading confined to the trunks. | 2. Cellular connective tissue proliferation about lymphatic trunks in the walls of vessels and bronchi. Beading may be due to various causes, as blood vessels seen end on, arteriosclerosis, minute areas of fibrosis in lymphoid tissues along the trunks. |
| 3. Increased root shadow. | 3. Cellular reaction in the tracheo-bronchial lymph nodes with extensions along afferent lymphatic trunks. |

These changes come within normal variations when not accompanied by recognized organic disease. *Irregular exaggeration of the linear markings, with possibly some beading,* belongs in the healthy chest group even when found in persons with a history of considerable exposure to silica, for such changes are nonspecific in character and they do not involve the parenchyma of the lung. Silicosis as a clinical

disease begins only when the lung proper is affected. Likewise, under 3. *Increased root shadow* may be of nonspecific origin and hence is not diagnostic. In the *early* stages of silicosis, the mediastinal shadow may be widened, owing to the enlargement of the tracheobronchial lymph nodes from accumulated dust and cellular reaction to it; later, when specific fibrosis develops, the tissues generally contract and the nodes decrease in size. The changes described under 2 and 3 may be caused by many forms of irritation; if they are due to silica, they are identifiable only by microscopic examination. They do not, apparently, interfere with respiratory function, and they are not of diagnostic significance.

Roentgenological Appearances *Histological Appearances*

SIMPLE SILICOSIS

1. *Nodulation*. — Discrete shadows not exceeding 6 mm. in diameter, tending to uniformity in size, density, and bilateral distribution, with well-defined borders surrounded by apparently normal lung shadow. The outer and lower lung fields characteristically show fewer nodules.
 2. Conglomerate shadows that appear to result from a combination or consolidation of nodulation usually with associated emphysema manifested by —
 - a. Localized increased transparency of the lung with loss of fine detail.
 3. Circumscribed nodules of hyaline fibrosis located in the parenchyma of the lung. Occasionally, some of these nodules may show microscopic foci of central necrosis.
 4. The result of coalescence of discrete nodules; an area in which the nodules are closely packed and most of the intervening lung is replaced by more or less hyaline fibrous tissue. The lung architecture is partially obscured. No demon-
-

Roentgenological Appearances

Histological Appearances

SIMPLE SILICOSIS — continued

- | | |
|---|---|
| <p>b. Intensification of the trunk shadows by contrast.</p> <p>c. Depression of the domes with possible tendency toward individualization of the costal components of the diaphragm.</p> <p>d. Lateral view: Increase in the preaortic and retrocardiac space with exaggerated backward bowing of the spine. Widening of the spaces between the ribs may or may not be present.</p> | <p>strable evidence of infection. Emphysema is a compensatory dilatation of the air spaces with or without thickening of the septa.</p> |
|---|---|

The second group of changes is limited to *simple silicosis* uncomplicated by demonstrable signs of infection. This condition is characterized by the presence of small, discrete nodules of fibrous tissue disseminated throughout the functional parts of both lungs. The lesions and the shadows cast by them tend to be spherical, hard, sharply defined, and vary in size from 2 to 6 mm. While the distribution is usually uniform throughout both lungs, the extreme apices and the outer portion of the bases are frequently uninvolved. In less advanced cases, the nodules remain discrete and separated by air-containing tissue.

Number 5 deals with the *conglomerate shadows* of simple silicosis, which appear to develop from a combination or consolidation of discrete nodules. The resultant lesion and the shadow that it casts are often difficult to distinguish from the *massive shadows* of silicosis with infection, 9. It is gen-

erally assumed that conglomeration results from accidental overlapping and fusion of discrete nodules when they become very numerous ; but since conglomeration is usually a localized affair and does not occur in the same position of the lung of every individual, it is logical to enquire why the nodules happen to fuse in one portion of the lung and not in others. Microscopic examination of the tissues from such areas reveals no evidence of active infection. The nodules seem to be much closer together than in other portions of the lung, they are less uniform in size, and they are usually embedded in a matrix of diffuse fibrous tissue having the same characteristic hyaline appearance as that forming the nodules themselves. It seems probable that conglomeration may have occurred because the portion of the lung in question was previously damaged by a localized inflammatory process occurring before or during the early period of dust exposure. Because the tissue was injured, more dust would tend to accumulate in the area, the nodules would develop irregularly and would frequently be very close together. The silica lodging in pre-existing granulation or scar tissue would exert its characteristic effect, and a diffuse hyalinization would result. This explanation for conglomerate reaction is, at present, hypothetical ; proof will come from long continued serial roentgenographic studies of groups of persons exposed to silica dust and from the chance autopsy that may be obtainable. To differentiate *conglomerate shadows* from the *massive shadows* of infection, reliance must be placed upon the absence of change in size and character of the shadows in serial films taken over an extended period of time and upon the clinical findings in the case.

Emphysema is usually associated with far advanced silicosis and it is particularly liable to complicate conglomerate nodulation. It occurs in the immediate vicinity of the conglomeration as a result of the distortion produced by con-

tracting scar tissue ; there is, also, a generalized 'compensatory' emphysema found along the borders of the lung, particularly at the bases. The latter type is also common in far-advanced generalized nodulation.

AUTHOR'S NOTE : I am not at all certain that conglomerate shadows and even nodulation should be included under a classification of simple or uncomplicated silicosis. My experience in an industry of pure silica workers rather points to some complicating factor. I am almost convinced that shadows, such as described above, can be explained by a complicating healed infection. This will be discussed later.

Röntgenological Appearances Histological Appearances

SILICOSIS WITH INFECTION

The characteristic appearances described under simple silicosis are modified by infection as follows :

- | | |
|--|---|
| <p>6. Localized discrete densities and/or string-like shadows accompanying those of simple silicosis described above.</p> | <p>6. Strands of fibrous tissue, often along trunks and septa, with or without areas of calcification ; indicative of 'healed' infection.</p> |
| <p>7. <i>Mottling</i>. — Shadows varying in size with ill-defined borders and lacking uniformity in density and distribution, accompanying simple silicosis.</p> | <p>7. (a) Areas of bronchopneumonia with or without caseation, i.e., acute infection.
(b) Lobular areas of proliferative reaction with or without caseation, i.e., chronic infection.</p> |
| <p>8. <i>Soft nodulation</i>. — The nodular shadows described under simple silicosis, 4, have now assumed fuzzy borders and/or</p> | <p>8. Perinodular cellular reaction either exudative or proliferative in character.</p> |

Roentgenological Appearances Histological Appearances

SILICOSIS WITH INFECTION — continued

irregularities in distribution.

This change may or may not accompany the simple mottling of 7.

- | | |
|---|--|
| 9. <i>Massive shadows</i> of homogeneous density not of pleural origin symmetrically or asymmetrically distributed. | 9. Extensive areas of fibrosis probably due to organized pneumonia of tuberculous or nontuberculous origin superimposed upon a coexisting silicotic process. Outlines of normal structures may be partially destroyed. |
|---|--|

In the last group, *silicosis with infection*, are included all cases with detectable evidence of infection whether active or inactive. In this respect, we depart from the South African procedure, which includes here only active infection. The difficulty of determining activity, particularly in the silicotic subject, is our chief reason for this arrangement.

Number 6 covers foci of healed infection. Identification of such changes depends upon the same criteria that are generally employed in otherwise normal individuals. In the silicotic subject, the shadows usually occur upon a background of generalized nodulation, although in some cases, there may be a distinct tendency toward excessive nodulation in the immediate vicinity of the scars left by the infection. Where the exposure to dust has been limited, the major evidence of nodulation may occur about the foci of healed infection with much less reaction in the remainder of the lung. The string-like shadows of healed fibroid tuberculosis

are not difficult to interpret if they occur in the classical location, i.e., in the upper third of the lung. In the lower lung, they present a problem whose solution depends largely upon the experience of the roentgenologist.

The term, *mottling*, 7, we have reserved to describe the shadows of infectious lesions in contradistinction to *nodulation*, which is restricted to those of the silicotic dust nodule. It is essential that this distinction be appreciated and recorded in the terminology. In tuberculosis, mottling is due to bronchogenic or aspiration foci of disease which exhibit a characteristic clustered arrangement. The lesions may be exudative (acute) or productive (chronic) in type; the difference will be registered in the roentgenogram by a mottling which is fluffy and ill-defined, or hard and sharply defined, as the case may be. The distribution of the mottled foci, together with the presence of large foci of older disease interpreted as tuberculosis, and clinical and laboratory findings establish the character of the infection. Mottling due to chronic infection that has developed previous to or simultaneous with the relatively early periods of dust exposure may exhibit little or no effect from the inhaled silica for many years. In nontuberculous broncho-pneumonias, the large chronic foci are absent, and the disseminate mottling may involve different parts of one lung or of both lungs. In many instances, the nature of the infection must be established by serial examinations over considerable periods of time and by careful correlation with clinical and bacteriological findings.

Soft nodulation, 8, is a term that has been coined to describe a rather uncommon combination of silicosis with infection, usually tuberculous. The ordinary hard, sharply defined nodular shadows of simple silicosis, under these circumstances, appear to have enlarged and lost definition. Their borders are now fuzzy and blend imperceptibly with the sur-

rounding lung structure. Such lesions generally occur in association with localized conglomerate shadows in the apex or other portions of the lung. Histologically, the infection appears to have localized in and about pre-existing silicotic nodules, so that each is surrounded by a zone of exudative or productive cellular reaction.

Massive shadows of homogeneous density, g, are cast by the areas of combined silicosis and infection, usually chronic in nature. The two processes appear to have developed simultaneously and unusual amounts of dust accumulate in the diseased area. Generalized nodulation usually occurs throughout the remainder of the lungs. Pleural densities can be differentiated in stereoroentgenograms, and by overexposure, it often becomes possible to penetrate the extremely dense intrapulmonary areas and analyze their internal structure. Not infrequently, cavities may be visualized that were completely overlooked with the usual technic. When due to tuberculosis, such lesions are often bilaterally symmetrical. If the process extends to the pleural surface, a tuberculous etiology is postulated, while other infections are more often deep-seated.

Histological examination of such lesions shows conglomerations of simple nodules embedded in masses of more or less perfectly organized granulation tissue. Often the fibrous tissue has undergone the same peculiar hyalinization that characterizes the interior of the silicotic nodule. Usually, the outlines of the original lung architecture are completely destroyed. Manifestations of infection depend upon the nature of the process. If tuberculous, there will be foci of caseation and possibly small cavities. Calcification is not infrequent. If the process is inactive, the presence of fibrous tubercles, which do not exhibit the hyalinization of silicosis, may be present. The occurrence of giant cells is helpful. A partially organized nontuberculous pneumonia usually contains foci

of acute exudation of variable size. Clinically, such unresolved pneumonias frequently exhibit periods of exacerbation followed by regression. They may be due to a great variety of organisms, including the Friedlander group and oral anaerobes. Where all manifestations of activity have disappeared, the lesion is probably best classified as a conglomerate shadow of simple silicosis, 5.

ROENTGENOLOGIC CONSIDERATIONS OF SILICOSIS AND SILICOSIS WITH INFECTION ¹⁰²

Many of the dusty industries with a silica hazard present certain unusual or interesting features, which have been largely attributed to the contaminating dusts. Since this consideration is concerned chiefly with silicosis, the individual industries will not be discussed separately. It is my belief, however, that various types of lesions found in different industries can be explained as being due to the modifying influences of infection. Possibly infection and contaminating dusts play a major role.

Silicosis: Nodulation, slow in progression, is one of the most characteristic of all appearances occurring in pneumoconiosis (when present). It results from exposure to dust in many industries, such as, mining hard coal, foundries, sand blasting, and sand pulverizing. The nodular aspect is due to the gradual enlargement of lymphoid deposits and their coalescence into quite apparent macroscopic nodules, in many instances, symmetrically scattered throughout both lungs. This corresponds to the beginning so-called second stage. It is conspicuously absent in some instances.

In early nodulation, where the progression has, presumably, been slow, the roentgen findings are as follows: The hila may be enlarged. The trunk shadows and linear markings are increased in prominence. The nodules are small, discrete, slightly dense, fairly uniform in size, and evenly

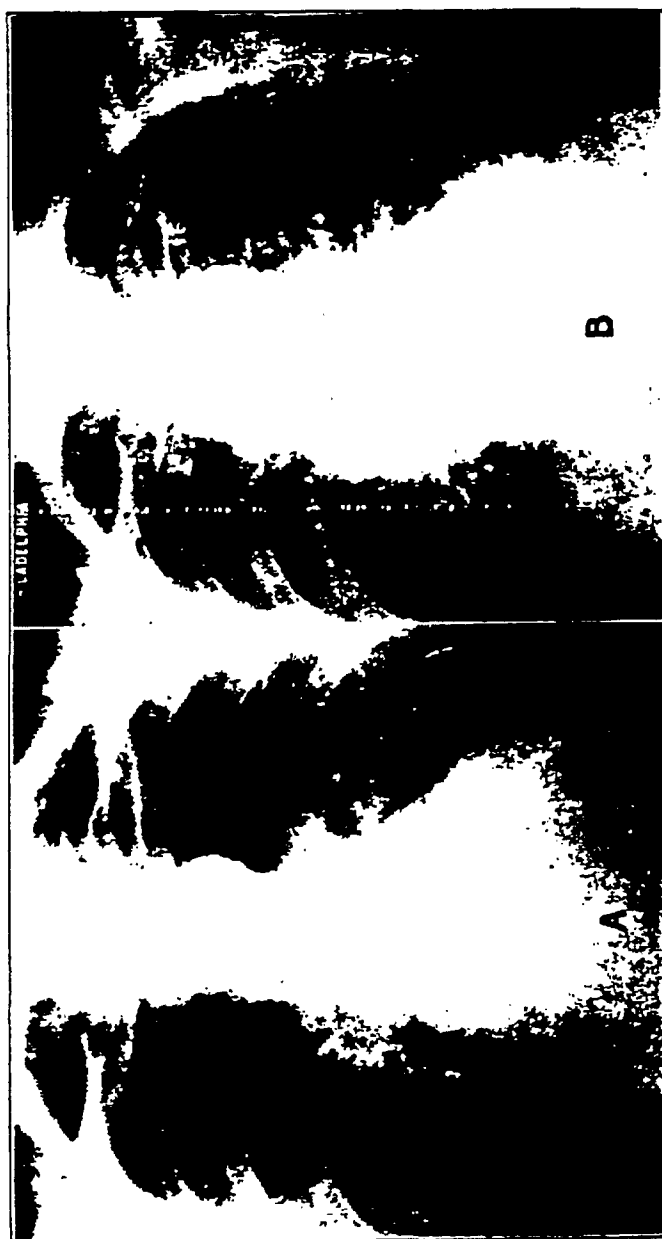


FIGURE 17

a. Metastatic hypernephroma involving the glands of the mediastinum. *b.* Six months later. There is a fine, soft nodulation throughout both lung fields which tends to coalesce in the region of the hila. This roentgen appearance may simulate that of soft nodulation in silicosis with infection.

distributed throughout the lung, more on the right at first. There is a slight homogeneous haze, beginning in mid-lung fields. At this time, there is no interference in diaphragmatic movement. There is, occasionally, thickening of the interlobar pleura. (Figure 11)

The above observations are included under simple silicosis only because of the fact that those most interested in this subject support such statements. I have always accepted early nodulation as evidence of simple silicosis, until it was possible to study a group of workers in a pure silica industry in which some of the workers were exposed to large quantities of finely divided silica over a period of years. In some instances, the exposure period was over forty years. In spite of such exposures, a large percentage of the workers showed no changes in their lungs. Individual lack of susceptibility has not seemed an adequate explanation to me for such observations.

Differential Diagnosis. — There are many conditions that will cause lung changes which simulate the roentgen findings of silicosis. Some of these affections have been definitely identified, others have not.

Metastatic Malignant Conditions of the Lungs. — Most cases of this kind are readily differentiated from nodular silicosis, but occasionally, one encounters a case in which appearances are very similar, because the metastatic nodules are large and widely separated. (Figure 17)

Tuberculosis. — These nodules are small, soft and fluffy, unlike the smaller, dense ones in silicosis, yet in some cases, it is impossible, either with clinical or roentgenographic evidence to differentiate the two conditions. The history will greatly aid in reaching a definite conclusion. Roentgenographically, the main variation may be the difference in size and density of the hila, although this is not always the same. (Figure 18)

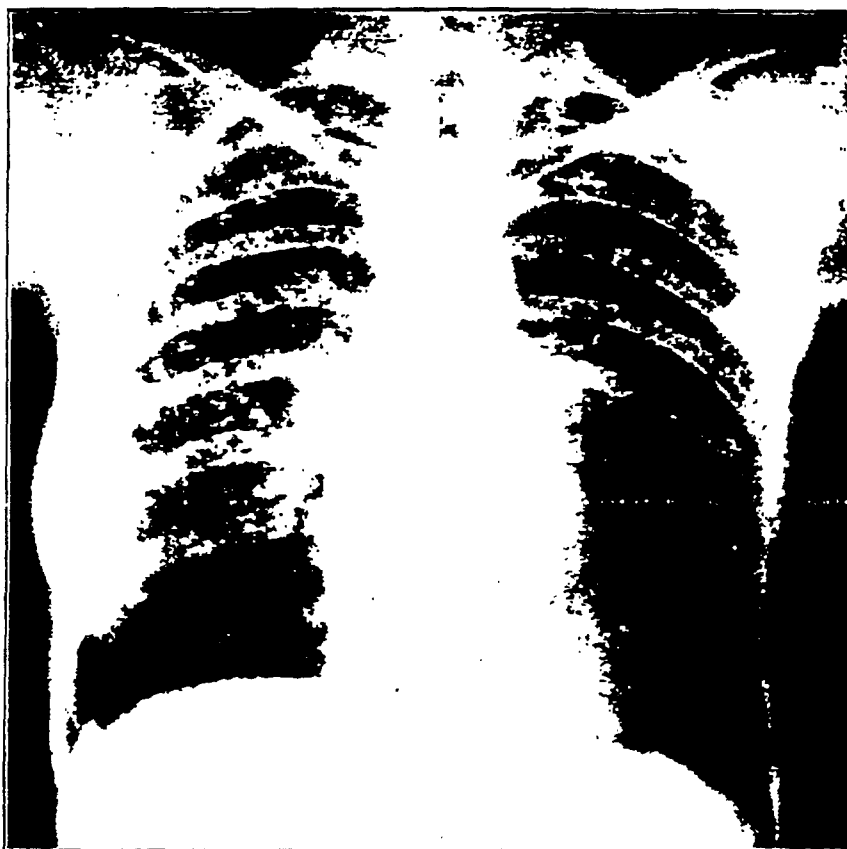


FIGURE 18

Miliary tuberculosis. This appearance could readily be mistaken for that of a nodular type of silicosis.

Mycotic Infections. — Actinomycosis, sporotrichosis, leprothrix, and other fungoid infestations may cause nodulation in the lung and simulate silicosis. Such nodules, however, are usually larger, softer, and of considerable variation in size. In proven mycotic infections, *dyspnea* has been an outstand-

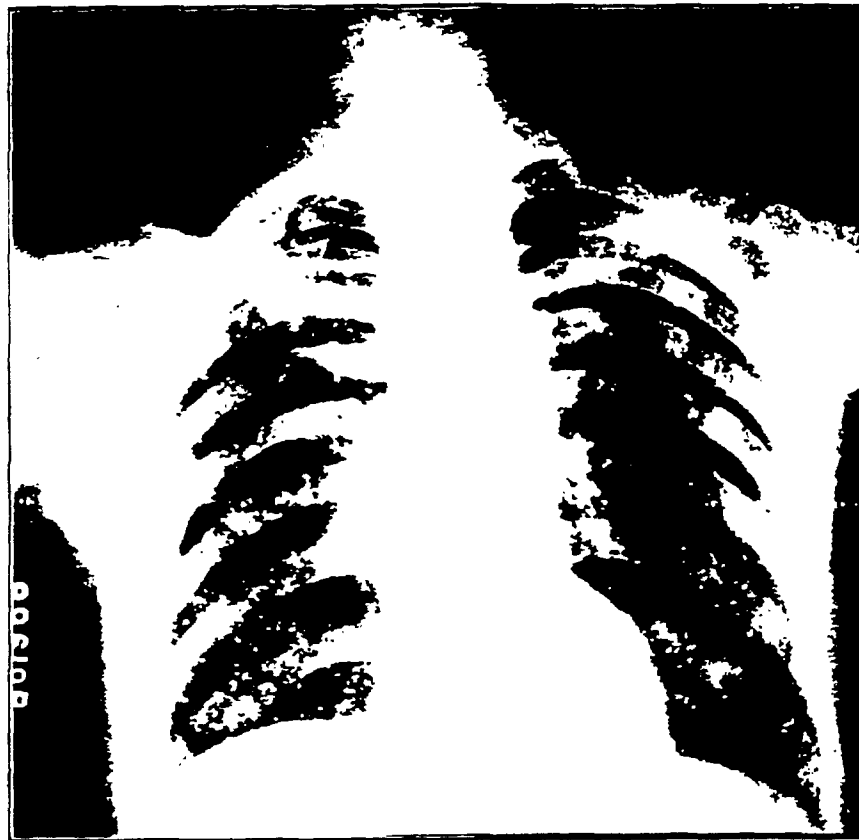


FIGURE 19

Miliary calcification of unknown origin. This patient had a carcinoma of the stomach and the miliary calcification in the lungs was a coincidental finding. He had never been exposed to dust and had never worked in the wheat fields. Dr. LeRoy U. Gardner studied pathological sections obtained at post-mortem examination and was unable to determine the cause of the calcification.

ing symptom. Fawcitt⁵² believes that these conditions are more prevalent than is commonly thought.

Miliary Calcifications. — Most roentgenologists have had



FIGURE 20

Case of baritosis. This individual worked in barium rock and had worked in this industry for a number of years. The findings in his chest roentgenograms were entirely coincidental and were not causing any symptoms. The individual was not incapacitated. In fact he had few, if any, symptoms. A study of the roentgenograms made approximately seven years before the one illustrated above showed a fine, discrete nodulation at that time, but not nearly so marked as is evident at present.

the experience of seeing one or more cases of multiple symmetrical lung calcifications, and most of us have regarded them as evidences of healed miliary tuberculosis, with or

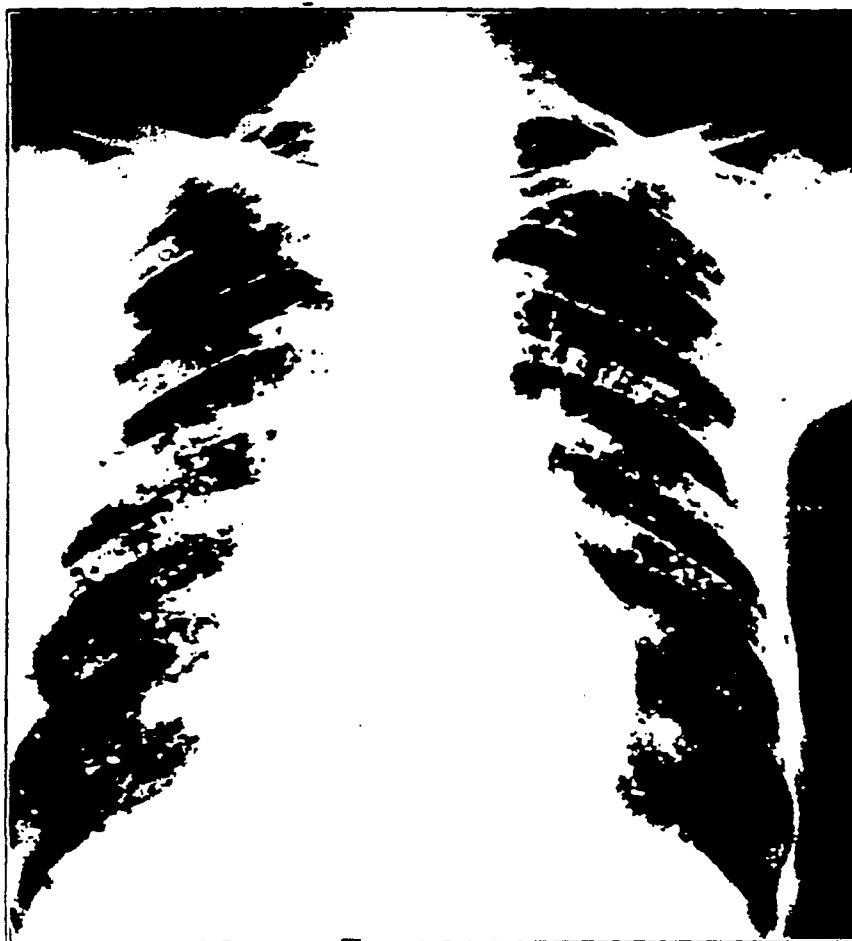


FIGURE 21

Soft nodulation in a hard coal miner, diagnosed as silicosis with infection. Note the tendency toward coalescence in the middle third of the right lung.

without good reason to so diagnose the condition. The appearance is not likely to be confused with silicosis. Sayers and Meriwether had the unusual experience of finding 125

such cases in an examination of 18,285 men at the Pilcher, Oklahoma, Mining Clinic. Many, but not all, of these men had been miners, and some of those, who had been, showed real evidences of pneumoconiosis in addition. (Figure 19) The lungs showed a diffuse distribution of dense, discrete, apparently calcified, shot-like spots throughout the lungs. They were of all sizes up to 1 cm. in diameter, and were variously ascribed to fungus infections, pneumomycosis, and healed miliary tuberculosis.

Baritosis. — I have seen several patients with widespread dense nodulation, typical of that seen in so-called simple silicosis, and similar to the cases seen in Italy.⁵³ These individuals have been exposed to barium dust, which may be responsible for the changes observed in their lungs. (Figure 20)

Silicosis with Infection (nodular predominance type). — Pulmonary tuberculosis is a frequent complication of pneumoconiosis. The latter condition may be a predisposing factor in the frequent incidence and serious consequences of the infection, especially when the progress of the pneumoconiosis is rapid. The two conditions often must be differentiated when only one exists, or pneumoconiosis is present in only a minor degree and presumably not sufficient to exert any predisposition. Differentiation is necessary because the two conditions may produce quite similar roentgenologic appearances. (Figure 21)

Tuberculous infection may be present in what appears to be a purely nodular type of silicosis. (Figure 22) It is far more likely to be a superimposed process, and presumably exogenous in the absence of any typical adult lesion. When the nodules tend to become conglomerate, or there is the appearance of considerable interstitial change between them, we may suspect a superimposed tuberculosis, although it cannot be absolutely proven. In the typical nodular type, the shadows vary in size from those barely visible to that of a

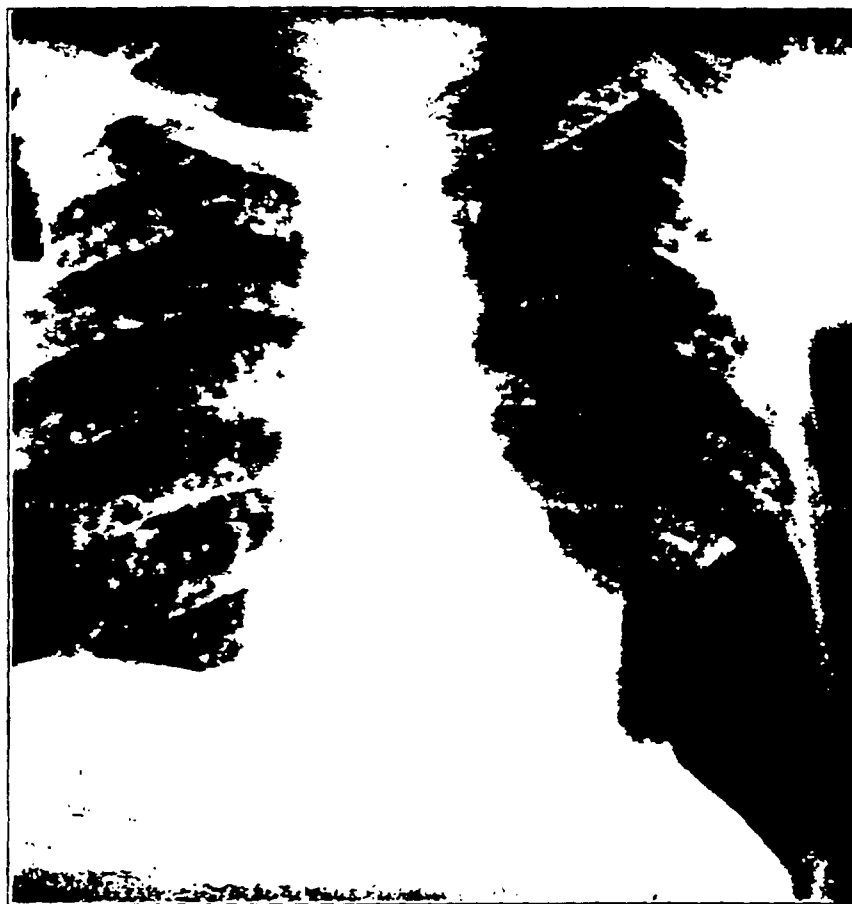
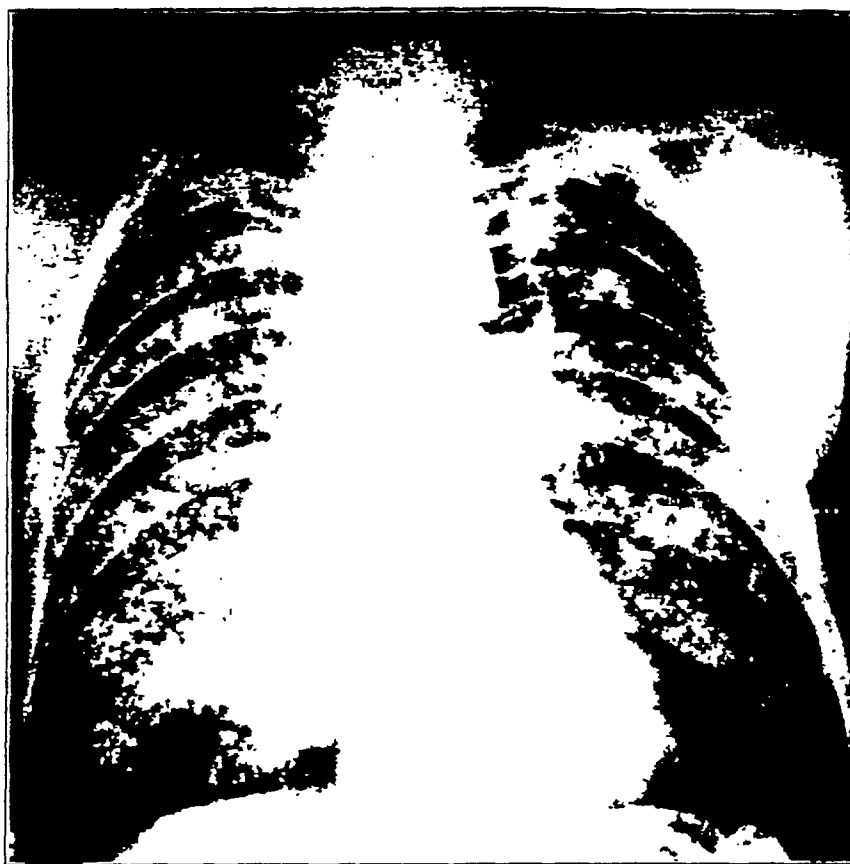


FIGURE 22

Perinodular or soft nodulation in a granite worker. Compare with Fig. 21. The nodules in Fig. 22 are much larger than those seen in Fig. 21.

small pea; they are more or less regular in outline and rather sharply circumscribed. The density depends considerably upon the size. The question of superimposed tuberculous infection may also arise when the nodular shadows



• FIGURE 23

Silicosis with infection. The infection is active and there seems to be a general coalescence of most of the nodules. One would expect the infection in such an individual to be more active than that illustrated in either Fig. 21 or Fig. 22.

are larger, and have a hazy, or fuzzy and irregular, and much less dense periphery, especially toward the apices. If the less dense periphery is spreading rapidly under serial studies, one may suspect superimposed tuberculosis of the

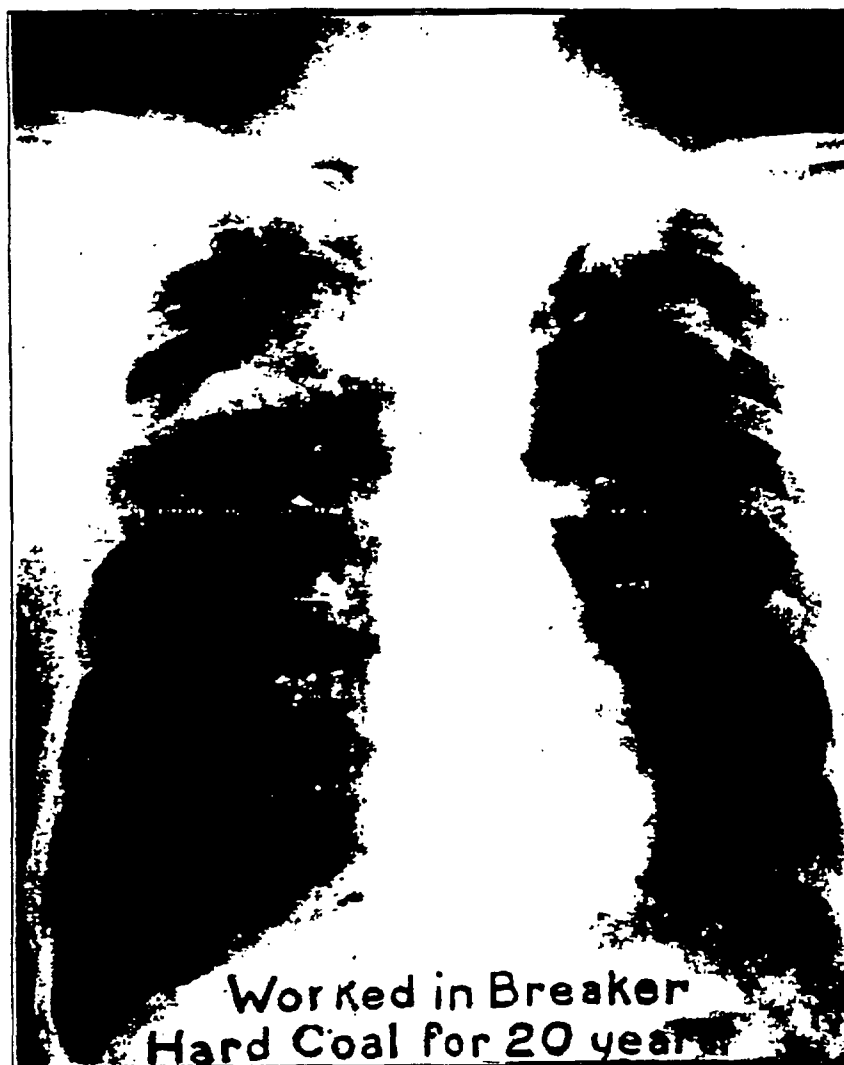


FIGURE 24A

A rather typical lesion seen in an individual having silicosis with a healed infection. These lesions are often bilateral and often found in the upper third of the lung fields. Note the aerated lung surrounding these areas.



FIGURE 24B

Close-up of the lesion in the right upper lobe.

perinodular type (Figure 22) especially if this is occurring in some nodules and not in others. To make this distinction by a single examination is likely to be guess work. Even serial observations must be made by the use of exactly identical technic.

It is entirely possible that other infections may complicate an already established silicotic process in the lungs, and, in the absence of other clinical or laboratory observations, it may be impossible to identify the nature of the contaminating

influence. Unless the infection be active, as indicated by changing roentgen appearances, determined by serial roentgenograms or by clinical observations, one may justifiably regard the lesion as silicosis with healed infection. (Figure 23)

Silicosis with Healed Infection (conglomerate nodular type). — The roentgen findings are as follows: The hila may be increased in prominence, but usually are not noticeable. The trunk and other lung markings may be increased. There are conglomerate masses and beginning massive fibrotic masses which do not extend to the periphery of the lung and are usually found in the upper two-thirds of the lung fields, involving either the upper lobe bases or apices of the lower lobe. (Figure 24) The lower third of the lung is emphysematous, as a rule. The trachea is not displaced, unless there has been a pre-existing tuberculous process. There is moderate interference with diaphragmatic movement, with possible peaking, humping or waviness of the domes. Not infrequently, there is thickening of the interlobar pleura.

In previous communications on this subject, I have regarded the above findings as indicative of a simple silicotic process. Increasing experience with pure silica workers has lead me to believe that such roentgen appearances represent silicosis with a healed infection, or at least a quiescent period in the infective process. During the active period of the infection, the lesion extends to the periphery of the lung. As the infection subsides, the conglomerate mass is found more mesially, with a clear lung area peripheral to it. Such observations as these have taught me to feel more optimistic as to the future in the treatment of silicosis with active infection. (Figure 25)

I have considered, under the healthy chest, a possible explanation for the location of the conglomerate lesions. It is in such lesions as these that the massage action of the thoracic cage, heart, and diaphragm upon the lymphatic flow may be

of major importance. With the subsidence of the active infection and its accompanying fixation, the lung structures can again begin to move relatively freely. In my experience, it has seemed that the extent of the lesion has progressed more readily in those areas of the lung (upper lung fields) that are more susceptible to fixation or interference with respiratory movements. (Figures 9 and 25)

Silicosis with Active Infection (conglomerate nodular type). — Pulmonary tuberculosis may be present before a silicotic process becomes predominant, or it may occur afterward. If the infection is active, the lesion extends to the periphery, usually. *It should be borne in mind that oblique or lateral views may be essential to diagnose or exclude peripheral extension of a lesion situated in certain portions of the lung.*

If the silicotic process was present before the tuberculous infection, the trachea usually remains in the midline, whereas, if the tuberculous process was present first, the trachea is more likely to be displaced, either to the side or anteriorly or posteriorly. Usually, pulmonary tuberculosis, with the possible exception of Friedlander's pneumonia, can, for practical purposes, be regarded as the cause of tracheal displacement.

In the larger lesions, heavily exposed roentgenograms, or those made with the aid of the Potter-Bucky diaphragm, may assist in demonstrating a cavity or calcium nodules, as a means of identifying the nature of the infective process.

I have seen only a few individuals in whom silicosis with active infection was complicated by a spontaneous pneumothorax. All of these patients did poorly. One wonders, therefore, if such observations may not be regarded as collateral evidence against the use of collapse therapy in the treatment of silico-tuberculosis.

*Differential Diagnosis.*¹⁰⁰ — *Infiltrating or Permeating Malignant Metastases.* — Quite frequently, metastatic malignant

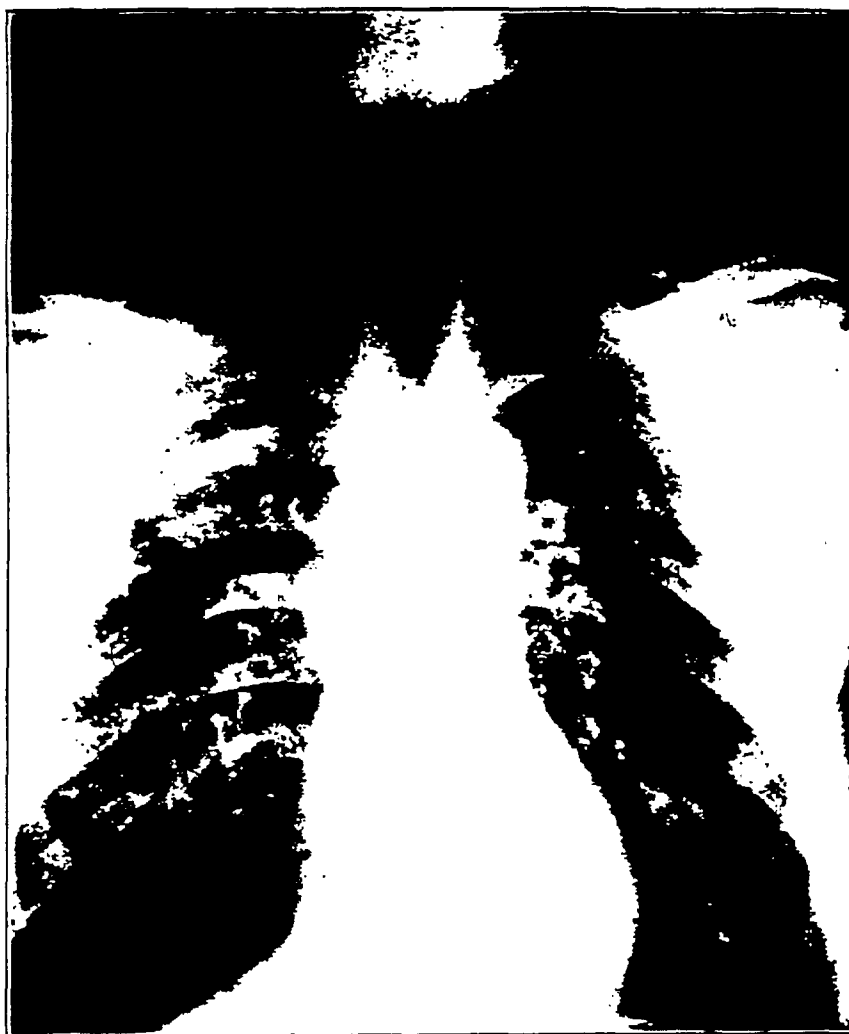


FIGURE 25A

Silicosis with infection. There are a few small, discrete nodules distributed throughout both lung fields. In the right upper lung field, there is a massive lesion which extends to the periphery.



FIGURE 25B

Five years later. The lesion in the right upper lung field has contracted down to a relatively much smaller mass, and there is now aerated lung around it. Elsewhere, the nodules in the lungs have not changed. This individual is working in pure silica dust, which contained high concentrations of finely divided silica up until a few years ago. This individual, at one time, had a silicosis with active infection. He now has silicosis with an inactive infection.

processes in the lungs are of such a character as to take on an appearance closely simulating that of pneumoconiosis of a conglomerate type. Naturally, the individual is incapacitated, and often in a manner similar to a silicotic patient.

Polycythemia or Erythremia. — This condition may be regarded from two points of view. As an independent disease, it usually has an associated increased prominence of the hilar and trunk shadows, due to the engorged pulmonary vessels. On the other hand, high red cell counts, even sufficient to suggest polycythemia, are very frequently associated with pneumoconiosis. There seems to be reason for believing that lung fibrosis and pulmonary arteriosclerosis can produce a condition akin, perhaps, to polycythemia and due, presumably, to impaired interchange of oxygen and carbon dioxide. Yater and Constam⁵⁴ reported two cases of primary pulmonary arteriosclerosis, one an anthracotic patient with emphysema, and the other with a mitral stenosis. They stated that secondary pulmonary arteriosclerosis involves the larger vessels and may be due to mitral stenosis, emphysema, congenital heart disease, and conditions in which the lung volume is reduced, as tuberculosis and tumor. Polycythemia is one of the clinical phenomena. Rosenthal⁵⁵ reported three cases of workers in dusty atmospheres or with irritating gases in whom there developed cyanosis and dyspnea, and true polycythemia in one. Moschowitz⁵⁶ believes that secondary pulmonary arteriosclerosis is very common. He found it in 6.5 per cent of autopsies and places the causes in the order of frequency as, mitral stenosis, emphysema, pulmonary fibrosis, extensive pleural adhesions, kyphoscoliosis, and congenital heart lesions. We have been interested in determining any relations between pneumoconiosis and polycythemia, and, in our search for any true associations, we have been inclined to accept the views of Wood,⁵⁷ that there are really two conditions to be considered. One is a compensatory mild polycythemia

from lung fibrosis, and the other, the true erythremia originating in bone marrow changes and capable of producing alterations in the roentgenographic appearances of the chest referred to at the beginning of this paragraph.

Silicosis with Infection and Massive Lesions. — Terminal and incapacitating silicosis with infection is characterized by three general appearances — a terminal diffuse condition of a conglomerate nodular type, one which is quite similar in appearance to a generalized chronic fibroid tuberculosis, and the terminal stage characterized by large consolidated areas, which quite frequently closely resemble tuberculous consolidations. Some of the lesions seem to be quiescent, with the roentgen findings as follows: The trachea or hilum is not displaced. The hila may be prominent, but often is not noticeable. The lung markings are increased, but usually obscured by emphysema. The lesions are usually in the upper two-thirds of the lung fields and do not extend to the periphery. The nodules are not particularly prominent. There is marked emphysema of the lower one-third of the lung field. As a rule, there are no cavities; if present, they may or may not be due to vascular injuries. The Potter-Bucky film shows trabeculation of massive areas without calcification or cavity. Frequently, there is marked interference with diaphragmatic movement. Peaking of the domes of the diaphragm occurs often, and sometimes, there is thickening of the interlobar pleura.

If the tuberculous infection was present before the silicosis became manifest and the infection is active, the roentgen findings are as follows: The trachea and the hila may be displaced, if pneumoconiosis was not the predominant lesion originally. Usually, lung markings are largely obscured by emphysema. The massive lesions are usually in the upper two-thirds of the lung fields and extend to the periphery. Nodular formation, if present, is generally best seen in the

lower lobes. Emphysema is marked. A Potter-Bucky film shows a homogeneous density of the lesion, with occasional cavity formation. At times, there are evidences of calcification. Almost invariably, there is interference with diaphragmatic movement, with peaking of the domes of the diaphragm.

One frequently observes a case in which there is a rather extensive consolidated lesion in the upper chest, more marked on one side, or limited to one side. In addition to this, there are nodular lesions, usually lower down, on one or both sides. This type of case represents a most difficult one for differential diagnosis. If the case is essentially one of tuberculosis, the massive lesion is most likely a rapidly progressive one and the nodular appearance may be due, in part at least, to a bronchopneumonic spread to one or both sides.

The differentiation really lies between massive consolidations of the terminal stage of silicosis with a healed infection and massive tuberculous lesions which are made up of a large amount of fibrosis as a result of the accompanying pneumoconiosis. Such lesions have the contracting characteristics of any large fibrosing tuberculous areas. This may be bilateral, but the chances are that one side started before the other, or progressed further and fibrosed more than the other. One must regard these lesions as of tuberculous, or partly tuberculous, origin when they are *peripherally* located. The unilateral or older bilateral lesion of this nature will cause homolateral displacement of the trachea and possibly of the vessels, and because it must retract upward and outward, the hilum shadow is apt to be similarly displaced. These displacements were brought to our attention in the first American paper on the roentgenological aspect of silicosis by Lanza and Childs³⁹ in 1917. Silicotic consolidations with infection will not displace the mediastinal structures unless the tuberculous infection preceded the silicotic process.

When there are bilateral consolidations, one of which is obviously tuberculous because of the effects of retraction, the lesion on the opposite side may not extend to the periphery, and yet be tuberculous, as indicated by upward displacement of its corresponding hilum shadow. This lack of peripheral location is explained by the fact that it cannot retract upon and displace the central movable structures, like the trachea, because they have already been fixed by the contralateral lesion.

There is still further differentiation to be made by roentgenological studies. If the individual, consolidated lesions are intentionally over-exposed, especially with the use of the Potter-Bucky diaphragm, considerable difference will be noted in the structures. The silico-tuberculous consolidation shadow will appear generally homogeneous, and any stringy appearance due to tuberculous fibrosis will be coarse and far apart, and cavities, or calcifications, or both, may be found. The purely silicotic consolidation with healed infection will not be nearly so homogeneous. In addition, there is apt to be a close trabeculation.

FURTHER CONSIDERATIONS OF TUBERCULOSIS AND SILICOSIS ⁹⁹

Throughout the following paragraphs and to some extent throughout the chapter, the terms 'silico-tuberculosis' and 'tuberculo-silicosis' are used. These terms are rather confusing and possibly the terms should be dropped entirely.

On the other hand, these terms are frequently referred to in compensation legislation and in the literature on this subject. The terms 'silico-tuberculosis' and 'tuberculo-silicosis' have gained a great deal of notoriety in the radiologic literature particularly and in those instances, this author at least, implies one of the following ideas.

a. silico-tuberculosis

1. The patient has had a well developed silicosis before tuberculosis became superimposed. In such instances, the trachea remains in the midline.

2. Silico-tuberculosis may mean that the tuberculous lesion although predominant, has had its clinical and radiologic manifestations modified to such an extent that the roentgen and clinical signs are not those of tuberculosis alone, even though the tuberculous process is out of proportion to that of silicosis.

b. tuberculo-silicosis

1. The term tuberculo-silicosis is employed in those instances in which tuberculosis was the primary lesion and silicosis was superimposed. In these individuals, one does not refer to childhood tuberculosis, but only to the adult type and in such instances, the trachea is usually displaced toward the tuberculous process.

It has seemed advantageous to discuss the relations of tuberculosis and silicosis under certain definite groups of cases, which will be dealt with in numerical order as follows :

- 1 : The first group to be discussed comprises those cases of pulmonary tuberculosis in which there can be found no roentgenological evidences of silicosis after several years of service in a dust-laden atmosphere, and the condition present is obviously only tuberculosis. We must bear in mind that dust exposure does not preclude the possibility of anyone contracting tuberculosis. A consideration of this group must include the differentiation of the evidences of the infection from all of the phases of pneumoconiosis. We have seen all phases of silicosis interpreted from roentgenograms when tuberculosis was obviously the only condition present. In some of these cases, compensation had been granted and in others, claimed on the basis of silicosis simply through lack of diagnostic experience, and because the claimants worked

where the production of a silica-laden atmosphere was assumed as a possibility. The differential diagnosis in this group resolves itself into the ability to recognize evidences of any of the above-mentioned phases of silicosis, on the one hand, and those of pulmonary tuberculosis on the other. There seem to be many roentgenologists who are not able to do this, although they make the attempt, and especially in connection with compensation cases. A further discussion of this group would entail a prolonged dissertation on both diseases. It should be borne in mind that this group does not include cases which may have both conditions, but that tuberculosis is the only one present.

2: The second group of individuals to be discussed includes those cases of obvious pulmonary tuberculosis in which there are evidences of a very mild degree of silicosis or pneumoconiosis after several years of service in an industry, but in which it seems reasonable to believe that the latter condition is not a predisposing factor. By the term, 'mild degree,' is implied a very moderate nodular appearance, which may have been produced or have been found after ten to fifty years' occupation in the same industry. In many instances, it is easy to draw the line between predisposition and lack of it, but in some cases, this is a very difficult question to settle, and the value of the decision must be governed to a considerable extent by the diagnostician's ability and experience. The law in some countries gives every worker the benefit of the doubt when both conditions are present, justly so in many instances and, no doubt, unjustly in others. Unfortunately, animal experimentation is of little or no value in helping us to decide whether a very slowly progressing pneumoconiosis, requiring many years to become evident in even a minor degree, is in any way a predisposing cause of the infection, because it is impossible to obtain proof of the action of silica or other dusts when inhaled in moderate quantities

upon animals over a period of very many years. Ordinarily, one recognizes two distinct types of human pulmonary tuberculosis, childhood and adult, which are, in many respects, almost different diseases. Anyone familiar with silicosis must recognize a third type, namely, silicotuberculosis, which presents many points of difference from the other two, and which will be discussed later.

In the group of cases now under discussion, we would not use the term *silicotuberculosis*. In these individuals, one would expect to find the usual appearances of adult tuberculosis, throughout its course. In many instances, we have noted evidences of a tendency to heal with only the usual amounts of fibrosis. Complete clinical and roentgenological healing of such lesions would almost always imply lack of predisposition by reason of dust. Lack of complete healing may leave one in doubt as to future predisposition and change in type, whether the individual changed his occupation or not, and subsequent serial examinations would have to be the deciding factor. In this connection, it should be borne in mind that silicosis with an active infection may be a progressive condition for many years after cessation of the dusty occupation, and predisposition may become manifest a long time after the dust exposure.

Our greatest experience with the group of cases under discussion has been in connection with coal-miners. The frequently alleged protection against tuberculosis afforded by coal-dust is well known to everyone interested in pneumoconiosis. In discussing this problem, we must not lose sight of the fact that, in the coal-miner, we are dealing with two different kinds of dust, and, therefore, two entirely different conditions; one, anthracosis, due to coal-dust alone and relatively harmless; and the other, best designated by the term *anthraco-silicosis*, or *silico-anthracosis*, recently used by Cooke,³⁸ and due to a mixture of coal-dust and silica. Pure coal-dust by itself, does not produce more than a mild degree

of pneumoconiosis and probably does not, in itself, or through its own effects alone, predispose to tuberculosis. If tuberculosis occurs in the pure anthracotic, it is likely to take the usual form of adult tuberculosis. On the other hand, there seems to be conclusive evidence presented by Cummins,⁵⁹ Middleton,⁶⁰ and others, that coal-dust aggravates the action of silica when the latter is inhaled in large amounts, but Mavrogordato,^{61, 62} Heffernan,⁶³ and Heffernan and Green,⁶⁴ and others, believe that, when the intake of silica is slight, the action of coal-dust may be a retarding one on the silica. Certainly the excessive inhalation of both dusts is productive of a predisposition to tuberculosis. Cooke⁵⁸ quotes from Cummins⁵⁹ that retention of coal-dust to any serious degree does not occur unless the lymphatics of the lung have been previously damaged by the action of silica, in which case the coal-dust is capable of accumulating and leading to serious, and even fatal, disease.

3 : The third group of cases for discussion comprises a very important one in which the individuals present obvious tuberculosis associated with slight evidences of silicosis, after a comparatively short period of occupation, and yet, it is most likely that the silicosis is a definite predisposing factor in the infection and its progress. There may be another factor of the silica-saturated lung to be considered, which will be discussed later in connection with group 4. This present group has been a very interesting one to us and one which confused us considerably until we became oriented, because it was a new experience. We refer particularly to sand pulverizers and workers in some other industries, who are exposed to unusually large amounts of dust containing a very high percentage of silica. In the incapacitated individuals, tuberculosis was the striking feature. In most cases of this group, the evidences of silicosis were slight. The tuberculous manifestations were almost invariably those of a diffuse pneumonic process, unlike

the cases of more chronic silicotuberculosis. Moreover, the clinical aspects differed. There was a rapid onset of dyspnea, cough, hemoptysis, night-sweats, asthenia, and extreme weakness, in contradistinction to the characteristic comparative freedom of the more chronic cases from serious symptoms until near the end. We have no way of determining whether any of those cases had tuberculous lesions before starting work, or whether some or all contracted the disease during their comparatively short time at their occupation. All of these workers were cases in which a complete knowledge and understanding of the physical factors incident to the industry were absolutely essential in the correct roentgenological interpretations.

4: The fourth group, which merits separate considerations, embraces cases of obviously advanced tuberculosis in which there are no demonstrable evidences of silicosis, or, if present, they are entirely masked in individuals working under conditions in which the physical factors in the occupation would lead one to suspect the possibility of a rapidly developing silicosis, as in group 3. The essential theoretical point is that the intake of silica has been unusually rapid, but there has not been sufficient time for demonstrable fibrosis, or even demonstrable prefibrotic stages to become established. The question arises as to whether a silica-saturated lung, with the preliminary stages of silicosis in progress, can act as a predisposing factor in the incidence of tuberculosis, or can reactivate old but unhealed lesions. We have frequently come in contact with cases in which these questions have arisen and it is an issue that we can no longer evade.

These two aspects of the situation must be considered separately, and the second will be discussed first. It would seem that the question of reactivation would have to be answered largely by the results of animal experimentation. Gardner⁶⁵ states that it has been generally assumed by clinical investi-

gators that the terminal tuberculosis, which causes the deaths of at least 25 per cent of silicotic subjects, is the result of infection *acquired* during industrial life. Hence, he carried out a series of animal experiments to determine whether or not certain dusts might not *reactivate* old, partly healed foci, and from this source produce endogenous reinfection. Guinea pigs were infected through the air passages by the author's usual attenuated strain of tubercle bacilli, which produced the typical 'primary complex' with definite tendencies to limitation and healing, and the usual primary involvement of regional lymph nodes. Groups of infected animals were dusted with quartz, granite, and carborundum dusts respectively. The dusting occurred at intervals when the healing process was started, well under way, or nearly completed. We are interested particularly in the effects of quartz dust, containing 99.34 per cent SiO_2 . In the first group, when dusting began fifty-four to eighty-seven days after infection and was continued eight hours per day, except Sundays, for variable periods, there was a spread of the tuberculous process in all the animals. In those exposed to dust one hundred fifty-one and one hundred seventy-seven days after infection, there was evidence of a spread in 60 per cent. In the third group, in which dusting began two hundred six days after infection, 33 per cent showed spread. This was usually most marked in those animals dusted for the longest period before killing. The spread developed by direct extension from the primary foci with or without subsequent widespread bronchogenic dissemination.

We are particularly interested in the question as to whether reactivation of tuberculous lesions can occur before it is possible for any demonstrable evidences of silicosis to become apparent. Gardner's experiments would seem to prove *that it can*, but we must bear in mind that his quartz-dusted animals were exposed to an atmosphere containing 859 million par-

ticles per cubic foot under 10 micra, of which 188 million were under 1.5 micra in size, which are very high silica dust counts for human industrial exposures. On the other hand, we must remember that the micro-organisms in the human subject are of much greater virulence than those used by Gardner.

Let us approach the problem from the standpoint of reactivation during a prefibrotic stage of silicosis. Gardner and many other investigators have shown us, and many of us have seen for ourselves, that dust cells will phagocyte very variable numbers of particles of different dusts, and that, in the case of silica, only as few as one to four particles will be incorporated, compared to enormous numbers of coal or carborundum particles. Probably, or at least, mainly for this reason, according to Gardner, quartz gives no limitations to locomotion and rapid removal to lymphoid tissue, nor does it interfere with physiological action in the sense of phagocytosis for the tubercle bacillus. It is also known that a reactivated tubercle has included in its structure great numbers of dust cells. Gardner states that it is uncertain just how the dust reactivates the quiescent tuberculous focus. It has been proved that inflammatory reactions, specific in the case of the frequent incidence of Friedländer pneumonia, and nonspecific through the applications of irritants, are not the factors *per se*.

Gardner⁶⁵ concedes that, in view of our present knowledge, or lack of it, the action must be chemical, either by silica or some other substance. He regards two general views as tenable: (1) That silica may injure the tissues and produce an environment particularly suitable for the growth of the bacillus through the elaboration of some substance by the action of silica on the body cells and better suited to the micro-organism than its environment in the tubercle protected from body fields by a wall of hyaline fibrous tissue; and (2) that in dilute solution, silica stimulates proliferation of the bacil-

lus, although we have as yet no accurate information as to such solubility in body fluids.

The second aspect of the situation, in regard to the newly silica-saturated lung acting as a predisposing factor in the *incidence* and rapid progression of a tuberculous infection, is not so easily disposed of. The foregoing remarks on animal experimentation would certainly apply to the rapid progression of the infectious process when once it had been initiated. Predisposition under the circumstances imposed has not been proved, but it is an issue which must be met. We are inclined to regard it as a strong possibility as yet unproved. The type of case discussed under group 3 would lead one to suspect the possibility from both clinical and roentgenological standpoints.

As to the roentgenological appearances in such cases, whether the infection was reactivated or incidental through chance or predisposition, we might expect the usual rapid forms of adult tuberculosis or a rapidly progressing and spreading pneumonic process, such as were encountered in many instances in group three cases. The important deciding factor in predisposition for the incidence of infection or in the silica saturation's being the cause of reactivation and progression cannot be the roentgenological examination, because the latter does not show evidences of silicosis. The decision, for the present at least, must rest almost solely upon the actual and *proved* physical factors present in the individual's occupation, which must be beyond all shadow of doubt before condemning a man in this group or placing the responsibility.

5 : The fifth group comprises those cases of obvious tuberculosis in which there is evidence of silicosis which could readily be a predisposing factor in the incidence of the infection. This group embraces a large number of cases, and has received wide recognition. The peculiar feature of this

group is that the roentgenological evidences of the infection are usually out of all proportion to the clinical aspect of the cases. These cases are well recognized as one of the group of silicotuberculosis. The rapidity of progress of the infection is often, if not usually, commensurate with that of the silicosis. In the more slowly developing cases of silicosis, the infection behaves differently from ordinary tuberculosis. As Gardner ⁶⁶ states, the clinical study of human pneumoconiosis complicated by tuberculosis has shown that the disease tends to run a chronic course, and the symptoms of intoxication are not so pronounced as in undusted individuals. The slow progress is due to the fibrosis, which is usually intensified by the combination of the two conditions, as is generally recognized. Gardner regards the lymphatic obstruction of the silicosis as sufficient explanation for the lack of toxic symptoms. Obviously the more rapid the progression of the silicosis, the more likely is the infectious process to be influenced like the cases of groups three and four.

6: The last group does not so much concern a definite group of silicotuberculous individuals as it does a pathological problem. Frequently, when examining silicotic suspects, we encounter cases whose roentgenograms show very obvious calcifications in the pulmonary lymph nodes, with or without a calcified primary nodule, and the question arises as to the possible effects of childhood tuberculosis upon the progression of silicosis. In 1925, Gardner ⁶⁶ stated that the lungs of individuals whose pulmonary lymph nodes were involved with healing or healed tuberculosis would more readily react to smaller amounts of dust, and that an obstructive fibrosis would occur in a shorter time. He intimated that this might account for the great irregularity in the time required for the development of demonstrable silicosis in industry. We believe this still remains a theoretical problem, but we feel that an individual with a homogeneous calcification of the

lymph nodes is a safe subject for employment in a dusty atmosphere, whereas the individual having calcified nodes that are punctate or mottled may not be.

Tuberculosis is a very frequent complication of silicosis, and, as such, is almost certain to be the eventual cause of disability and is quite likely to be the essential cause of death. It is to the interest of the employer, therefore, to make certain that hygienic surroundings under living, as well as under working, conditions are kept healthful. It is highly important that the worker in hazardous dusts does not contract tuberculosis from an outside source if it can be prevented. Naturally, the individual with active tuberculosis should not be employed, and, with past evidence of an infection, employment should be considered with the utmost caution.

COMPLICATIONS IN SILICOSIS¹⁰⁰

Primary Bronchogenic Carcinoma. — Primary bronchogenic carcinoma is arousing much interest in connection with pneumoconiosis because of the possibility of the latter condition's acting as a predisposing cause. (Figure 26) The publicity of the historic and very obvious predisposition of the Schneeberg miners to lung cancer and the more recent knowledge of a similar state of affairs among the Joachimstal underground workers has led to a belief that pneumoconiosis may play a part in the etiology of bronchial carcinoma.

We have studied a few such patients, yet we are not convinced that there is ample justification at the present time for a belief that pneumoconiosis is a predisposing factor in the incidence of primary bronchogenic carcinoma; and for the following reasons:

1. We have had the opportunity to study a large number of cases of pneumoconiosis from various industries and, in addition, have been so situated, in a medical center and in an institution well equipped with an active bronchoscopic clinic,

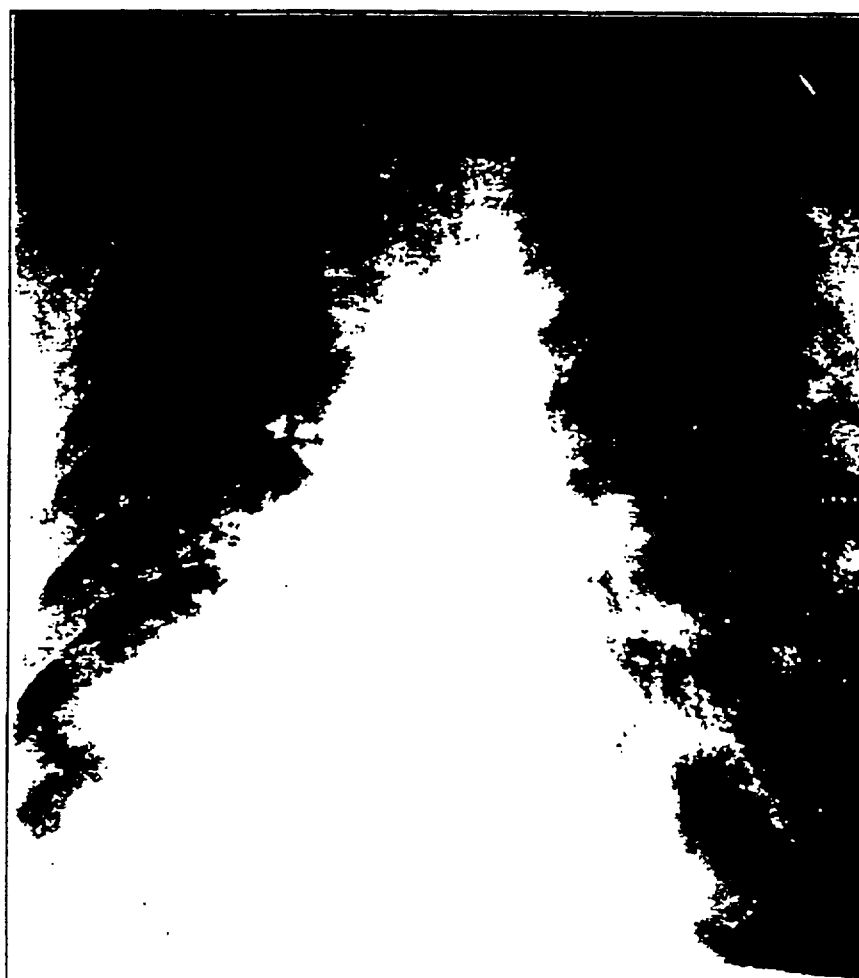


FIGURE 26

Primary carcinoma of the right lower lobe in a worker. There is very little, if any, roentgen evidence of silicosis. The silicotic changes, however, were demonstrable in the post-mortem examination.

as to have referred to us for examination or consultation many cases of primary lung cancer. Notwithstanding these facts, we have seen only three proved cases of bronchogenic carcinoma in association with pneumoconiosis. While Weller⁶⁷ states that other authors have reported cases among coal miners, sandstone workers, cigar makers, and in many other occupations, we cannot accept isolated reports as conclusive evidence, especially as every male patient with cancer of the lung has, presumably, had a job of some kind.

2. The Schneeberg and Joachimstal cases were obviously due to some factor different from the usual causes of pneumoconiosis, and radium emanation or arsenic would seem to have been the most likely of the unproved predisposing causes.

3. While animal experimentation, as carried out by Willis,⁶⁸ Willis and Brutsaert,⁶⁹ Murphy and Sturm,⁷⁰ and others, has suggested that certain cell hyperplasias, producible through the action of dusts, tar, and other irritants, may be precancerous conditions, there is no absolute proof that they are. Moreover, similar hyperplasias are noted bronchoscopically quite often in the human subject and are invariably reported histologically as inflammatory in origin. The Jacksons⁷¹ report that, on many occasions, they have removed tumor-like bodies obstructing a bronchus and the histologic examinations have shown nothing except chronic inflammatory tissue. These growths have been regarded as serious because of the resulting secondary effects of atelectasis, drowned lungs, suppuration, abscess, and bronchiectasis. The etiologic factors have been regarded as stagnation of secretions, especially of a purulent character, and the specific granulomas — tuberculosis, syphilis, and fungus infections. Any inflammatory process in the bronchi may be associated with a nodule that assumes tumor-like form, and mechanical contraction through elongation and shortening of the bronchi during respiration

might mold protuberant inflammatory new formations into tumor-like form.

Fried,⁷² like many others, is inclined to ascribe bronchogenic carcinoma to infections and inflammatory conditions. Of the three types of cells found in the bronchial mucosa—ciliated, goblet and 'basal' epithelial cells—the last is concerned in the regeneration of respiratory mucosa. Metaplasia of these cells occurs in numerous bronchopulmonary diseases, as influenzal pneumonia, measles, diphtheria, and whooping cough, and Fried regards this metaplasia as, in a sense, a pre-cancerous state. He believes that only the basal cells produce cancer. This opinion is not shared by others. Fox,⁷³ for example, found 3 cancers with mucous producing or goblet cells among 37 positive biopsies in 85 referred bronchoscopic tissues.

A brief review of the Schneeberg and Joachimstal situations seems worth while for a better understanding of the relations between lung cancer and mining operations. Weller⁶⁷ states that cancer among the Schneeberg miners 'is probably the most extraordinary and at the same time the least understood of all associations which have been discovered to exist between occupation and the incidence of neoplasm.' Literature on the subject of the high mortality among the underground workers of the Schneeberg cobalt mines in Saxony began as early as 1500. Weller refers to the report of Thiele, Rostoski, Saupe, and Schmorl⁷⁴ of official investigations of 154 miners during a period of nearly four years, during which time 21 died, and 13 of these with a diagnosis of primary lung cancer made at autopsy. Two of these men had not worked in the mines for many years, which, we believe, makes the theory of radium emanation as the etiologic factor a debatable point, although it may not exclude it. Weller also refers to the investigation of the mines by Rostoski, Saupe, and Schmorl.⁷⁵ There was much hard rock

drilling and a great amount of dust. The ore contained iron, bismuth, tin, zinc, lead, manganese, uranium, cobalt, and nickel, chiefly in combination with sulphur and arsenic. Because of the uranium content, and therefore radium also, the ore was radioactive, as was, also, the mine atmosphere up to as high as 50 mache units. The possible etiologic factors considered were silica, chemically active dusts, especially arsenic, and, in volatile form, radium emanation and the flora of the damp mines. Many of the men had demonstrable silicosis. The exact factor still remains uncertain. In regard to the radium emanation, Martland⁷⁶ states that the active deposit present after death of the individual would be entirely too small to be measured.

At Joachimstal, in Bohemia, and 30 kilometers from Schneeberg, are the mines which have been famous as a source of radium, although originally they were worked for silver, cobalt, nickel, bismuth, and arsenic. The mining for uranium preceded the discovery of radium. In 1929 and 1930, Pirchan and Siki⁷⁷ investigated these miners, who also had been known to have a high mortality rate for pulmonary diseases. During that period, 19 miners died, 10 recently active and 9 pensioners. 13 of these were examined post-mortem, and primary lung cancer was found in 9. Tuberculosis, pneumonia, and trauma were the other causes of death. Unfortunately, careful studies were not made from the standpoint of the etiology of the cancer. The atmosphere of the mines had approximately the same radioactivity as at Schneeberg. Unlike Schneeberg miners, these men presented no marked degree of silicosis. We cannot help but feel that radium emanation and arsenic must be strongly suspected as etiologic factors in both of these mines, although it would be most difficult to obtain positive proof for the implication of the former.

- *Cardiac Lesions.* — A few articles in the literature call at-

tention to silicotic fibrosis as an etiologic factor in cardiac enlargement and break-down. While this may be true, it is very difficult to prove, and one raises the question as to whether there may not be some other etiologic factor operative coincidentally. If such were true, silicosis might be regarded as a major factor without sufficient evidence.

THE ERYTHROCYTE SEDIMENTATION REACTION IN SILICOSIS

There is some evidence⁷⁸ to show that the sedimentation rate in silicosis is increased. I have not had sufficient experience to allow me to state whether it is increased in individuals without active infection. Preliminary observations would seem to justify the following statement, however. In the absence of clinical or roentgen evidence of active infection, in workers showing roentgen evidence of silicosis or of silicosis with healed infection, I have rarely found an increased sedimentation rate. If this observation is subsequently substantiated, the sedimentation rate will become an excellent test to use in conjunction with other clinical studies to evaluate the activity or quiescence of a given lung lesion.

ASBESTOSIS

*The Asbestos Industry.*⁴⁹ — This industry has grown extensively during the past twenty years. This has caused attention to be centered around it as a factor in industrial disease, mainly, however, because of some very unusual features found in the lungs of those exposed to the inhalation of the dust. The first important one is the fact that pneumoconiosis is produced in the workers apparently by silicates instead of directly by silica. This fact will, no doubt, lead to further interesting studies of industries in which silicates predominate in the dusts. There is some confusion in regard to the action of silicates in the production of pneumoconiosis, es-

pecially as certain of them, like clay and kaolin, are supposed by some authorities to retard the action of silica. In addition to these, talc, French chalk, soap stone, pumice, and many other silicate substances are extensively used in various industries. Another unusual feature of pneumoconiosis in asbestos workers is, first, the ante-mortem and autopsy finding in the lungs of some very interesting, unusual, and characteristic mineral deposits, and still another is a progression of fibrosis and roentgenographic appearances differing from those of most injurious dust producing industries. Numerous articles have appeared on the subject during the past four years. The most comprehensive one has been that of Merewether and Price,⁷⁹ supplemented by Merewether,⁸⁰ in 1930, and again in 1934,⁸¹ in a general review of all phases of the asbestos industry in Great Britain and a resume of the results of their examinations of 363 asbestos workers. Prior to 1928, there were available records in medical literature of only 2 deaths among employees in the factories, but since then, a considerable number have been reported. The importance of the condition and its differences from other forms of pneumoconiosis seem to warrant a general acceptance of the term, 'asbestosis.'

Lynch and Smith,^{82, 83} in their reports, have been the first in this country, at least in medical literature, to deal specifically with the subject. One of their cases, a negro, died as a result of a gunshot injury after working twenty-eight months in a mill during a period of three years. His lungs showed definite interlobular, perivascular, and peribronchial fibrosis, and also the various asbestosis bodies. The second case, also a negro, died of lobar pneumonia after four and a half years of continuous work in a mill. His lungs showed, everywhere, a definite increase of young fibrous tissue, in addition to the various asbestos bodies.

In view of the meagre number of complete autopsy re-

ports in this country, the following report of Lynch and Smith⁸³ is given in its entirety :

Autopsy

The body had been embalmed prior to the autopsy. There was marked subcutaneous edema, especially of the feet and legs to above the knees, and some of the hands and forearms. The toes and fingers were stubby, but there was no cyanosis, perhaps interfered with by the embalming. The sternum was flattened above and definitely depressed over the epigastrium.

The pericardium and heart were definitely displaced to the left, the apex well outside the nipple line at about the fifth interspace. The heart weighed 343 gm. empty, was definitely broadened but not lengthened, the right heart furnishing the breadth, the left, not enlarged. The right cavities were well enlarged and open, the left ventricle closed. The right myocardium measured 8 to 10 mm. in thickness well up from the apex toward the pulmonary orifice. Microscopically the muscle fibres were not of uniform size, and there was generally a distinct enlargement of those of the right. They were of indistinct outlines and striations, and those of the right ventricle were frequently vacuolated. There was an increase of stroma, and the veins, particularly of the right, were definitely distended with blood.

The aorta was the seat of a minor atheroma, and the whole vascular system, outside the heart, was in good condition.

Both pleural sacs were completely obliterated by old adhesions, usually very dense, especially over the whole right lung and the upper and lower left. The diaphragm was up to about the fourth rib on the left but was low on the right. The left lung was retracted well over to the outer wall and high, occupying about one-half to two-thirds of the usual space. The pleura generally was thick and cartilage-like, especially over the apex and base. The lung was coarse,

leathery, nodular and lumpy over the upper half and the base, the middle being more spongy and air-bearing. The vessels and bronchi were very prominent emphysematous bullae in the parenchyma especially in the apex where there was a group or chain of honey-combed sacs. The interlobar pleura was obliterated. The bronchi appeared congested. The hilum lymph nodes were inconspicuous, did not appear enlarged, and were smoky black. There was no evidence of tuberculosis.

The right lung was large, filling the whole right chest and encroaching some toward the left. Its whole pleura was very thick and cartilage-like. The interlobar pleura was sealed outwardly, but was open, a clean membrane presenting, between the adjacent lobes. The lung was very much like its fellow generally, the upper lobe densely fibrous and lumpy, vessels and bronchi prominent, and a mass of emphysematous bullae in the apex. The middle lobe was not so fibrous, nodular or lumpy, and had prominent bronchi and air bullae. Both lungs, especially the right, bore much frothy fluid, which, expressed and examined, revealed numerous asbestosis bodies, free, in sheaves or clumps, and ingested by giant cells. The two ends were often engulfed by different giant cells. There were also a few dust cells, with fine black granular pigment, and masses of a yellowish amorphous substance, of the color of the asbestosis bodies, in cellular debris.

Microscopically there was an extreme grade of hyalinizing fibrosis of the lungs, universally but irregularly distributed. The pleura was thick and fibrous, and there was marked interlobular fibrosis. Scattered here and there were irregularly rounded areas of hyaline fibrous tissue, in somewhat laminated form, within which were masses of greenish-black granular substance. Here were also areas of liquefaction and calcification in the centre of these hyaline nodules. A large part of the alveoli were obliterated or virtually so. Some

lobules remained open, the sacs having thick fibrous walls. In these open alveoli, the epithelium was sometimes cuboidal and there were fairly numerous large round phagocytes, some with a group of nuclei, some mononuclear. These macrophages contained black or greenish-black or brownish granular pigment and an occasional asbestosis body. Where the lung was less fibrous, there were young connective-tissue cells and lymphocytic accumulations. The bronchioles were dilated and their walls thick and fibrous. In some areas, the lobules of alveoli showed marked emphysema, large empty sacs with thin walls. Asbestosis bodies in typical forms, with a variety of architectural figures, yellowish-brown, clubbed, dumb-bell and rod forms, were to be found widespread, singly or in groups within giant cells in the alveoli of less fibrous areas, singly in the alveolar walls and interlobular tissues showing the younger fibrosis. Associated with them was much granular substance of the same color, as if from disintegrated asbestosis bodies. Besides these pigments, there was much ordinary black anthracotic material around the vessels of the interlobular tissues. The interlobar pleura was especially thick. The large bronchi were practically normal. The peribronchial lymph nodes were the seat of marked fibrosis, edema, atrophy of follicles, and accumulation of masses of black and yellowish-brown granular pigment, of the same order as that in the lung.

The liver was grossly and microscopically in a state of extreme chronic passive congestion, which state, in lesser degree, was conspicuous in spleen, kidneys, and other viscera.

More and more data of this type are becoming available as recorded in the more recent contributions of Merewether,⁸¹ Lanza, et al.,⁸⁴ Lanza,⁸⁵ and Shull.⁸⁶

Symptomatology. — The symptom-complex of pulmonary asbestosis may be of interest to roentgenologists, especially because the condition is a rather new one in the group of

pneumoconioses, or, rather, an old one come to light and extensively studied. We have made a composite grouping of symptoms assembled from the reports of Wood,⁸⁷ of his 15 cases, Haddow,⁸⁸ Stewart and Haddow,⁸⁹ Soper,⁹⁰ and Wood and Gloyne,⁹¹ including 22 cases additional to those reported by Wood, as follows :

(1) *Onset*. This is variable and depends upon the exact occupation and the quantity of dust inhaled. Both are important, for the individual may not be engaged in a very dusty duty, but may be working in a room made very dusty by another procedure in the manufacturing process. Wood states that symptoms may appear comparatively early and gives, as the time limits, one and fourteen years. Haddow places the average period at about five years, and states that the condition is usually recognized first during or after an attack of influenza or winter cold. Cases are usually better in summer.

(2) *Cough*. This is a very variable symptom, sometimes nearly or entirely absent and seldom excessive.

(3) *Expectoration*. This is moderate when present, but is usually lacking except during bronchitic attacks.

(4) *Anorexia*. This is a rather constant late symptom. Haddow regards it as an indication to stop work. Individuals may then live for several years, but become progressively weaker, more emaciated, and exhausted, until pneumonia or bronchitis brings death.

(5) *Dyspnea*. This is the most striking symptom and practically the most important one. It is progressive as in other forms of pneumoconiosis, whether the individual stops work or not. Wood and Gloyne speak of it as 'a terrible tightness of the chest,' which is very expressive. While dyspnea is due primarily to inelasticity of the lungs, which is always beautifully demonstrated by roentgenoscopic observations of the diaphragmatic excursion, it is also, in a measure,

a result of interference with blood supply. Therefore, it is slow and insidious in its development, until, finally, the lungs are able to accomplish no more than just sufficient oxygenation of the blood to sustain life.

(6) *Cyanosis*. This is a very frequent late manifestation.

(7) *Weight Loss and Emaciation*. These manifestations are very striking in the late stages. They are out of all proportion to the physical signs, and thus differ from tuberculosis alone.

All of these symptoms are more or less those found in other forms of pneumoconiosis, and a symptomatology largely applicable to all forms is placed here for emphasis on this particular one. The last three symptoms are to be emphasized in connection with the same pulverizing industry.

There is an interesting skin lesion found in connection with the asbestos industry known as the asbestos corn. It has been mentioned particularly by Gloyne and by Soper, among the writers on pulmonary asbestosis. The fine asbestos fibers penetrate the superficial layers of the epiderm like thistle thorns and produce small corns or hypertrophies of the epiderm around them. No asbestosis bodies have ever been found in these lesions.

*Roentgenologic Considerations.*⁴⁹ — Comparatively few of the report on pulmonary asbestosis contain roentgenographic findings, and in many of those which do, the descriptions are not so scientifically made as to permit of easy comprehension or interpretation. Most writers who describe the appearances or mention them casually seem to regard them as most unusual or characteristic of the occupation, notably Wood,⁸⁷ Wood and Gloyne,⁹¹ and Merewether.⁸⁰ It is true that they are unusual, but we would hardly regard them as characteristic for the reason that other conditions induce quite similar roentgenographic appearances. In all industrial pneumoconioses in which the roentgenographic appearances deviate

from the stereotyped description first given to those of the hard rock miners, one must become familiar with those of the industry and correlate them with diaphragmatic excursion, clinical findings and degree of disability represented before being able to interpret the findings for that particular industry. For example, from the roentgenograms of 17 asbestos factory employees, examined by Pancoast in 1917 and reported by us in 1926,⁹² the appearances were not regarded as of any particular moment, but simply as minor changes such as are frequently found in connection with slowly fibrosing dusts. On reviewing these roentgenograms with Dr. J.V.Sparks,⁹³ who made the roentgenological studies of Wood's cases, referring to our records of the diaphragmatic measurements, and with a knowledge of the peculiarities of the asbestos industry, particularly from the standpoint of roentgenographic appearances, we at once realized that our first interpretations were based upon an entirely erroneous conception. It is questionable whether anyone viewing a number of roentgenograms of atypical pneumoconiosis representing several industries, could pick out the ones of asbestosis subjects alone. All of the cases represented in the series just mentioned were only moderately advanced. In some roentgenograms of asbestos factory workers seen with Dr.Lanza,⁹⁴ we noted that there was a direct progression into a terminal diffuse fibrosis without any nodular predominance. This terminal state seemed to have been reached in about twenty years, on an average. Nevertheless, in some instances, the nodular fibrosis was quite evident.

Wood⁸⁷ has given a very excellent description of the roentgenographic findings in the 15 cases examined, and this has been corroborated by Soper⁹⁰ in 1 case, by Oliver⁹⁵ and by Wood and Gloyne.⁹¹ Our interpretation of these findings was that there had been a rather predominant early interstitial fibrosis, with, later on, an associated soft nodular fibrosis.

The transition to the third stage, if we should adhere to that term, or at least to the terminal stage, would seem to have been mainly through a continuance of the interstitial predominance with very little prominence of nodular fibrosis. The early interstitial predominance was described as resembling a ground glass appearance.

On reviewing our own roentgenograms with Dr. Sparks and comparing them with the excellent roentgenograms of some of Wood's cases, we found a great similarity in the appearances. The early evidence of the condition was a faint homogeneous haze in the lower part of the mid-lung field which seemed to correspond to the appearance we have previously suggested as an early predominance of interstitial fibrosis. This haze spreads through the lower one-half or two-thirds of the lung field and may almost obscure the domes of the diaphragm later on, with very little evidence of nodular fibrosis. With it, there is a very decided restriction in movement of the domes of the diaphragm without the appearance of pleural adhesions, which seems to be proof of the general fibrotic nature of the process. Thickened pleura does exist, of course, as in any fairly well advanced case of pneumoconiosis. The most unusual feature of the condition seemed to us to be the fact that, in both sets of roentgenograms, the appearance seemed to begin more often on the *left* side than the right, and progressed more on that side. This is the reverse of all other occupations that we have studied. Merewether³⁰ states that it begins on the right side, as one would expect, and the cases we observed may have been examples of the exceptions which are occasionally observed in any industrial pneumoconiosis. We noted the same tendency to obliteration of the left cardiac border as mentioned by Wood, because of the diffuse fibrosis and the prominent trunk shadows. Wood states that the late stage may or may not show consolidations. Wood and Gloyne³¹ state that

the ground glass appearance in late cases, on careful analysis, resolves itself into a fine mottling and linear shadows which, toward the base, may look like cobwebs.

It is evident that the nature of these changes in the lungs requires the best type of roentgenograms for their portrayal. Also, the seriousness of what may seem to be minor changes to the uninitiated requires a roentgenoscopic study of diaphragmatic movements to make one realize the actual state of disability present. It is evident also, as Merewether states,⁸⁰ that an opinion as to the degree and intensity of an asbestosis fibrosis, based upon a comparison of roentgenographic changes with those shown in standard silicosis films, will be an underestimate.

More recently, Merewether⁸¹ and Shull⁸⁶ have not emphasized on which side the process is most evident in the beginning or late in the disease. This coincides with my more recent experience. The disease process may be diffuse, bilateral, or unilateral, but is practically always limited to the lower half or lower two-thirds of the lung field.

Of the 363 cases examined by Merewether and Price, 133 had roentgenographic studies. Of these, 52 were said to have presented appearances of diffuse fibrosis and 22 were suspicious.

Merewether⁸¹ says that approximately seven years must elapse between the commencement of exposure and the production of a serious degree of asbestosis. This space of time includes not only the trapping period of the fibrosis-producing dose of dust, but also the maturation period of the fibrosis, which periods, of course, overlap. The existence of this fibrosis-producing period accounts for the fact that no appreciable number of asbestosis cases are discovered until the second five years of employment. Relatively few cases mature in this minimum period of seven years; many, even in the more dusty processes, require eleven years. When

fibrosis of serious import has matured, the worker is unduly short of breath on any extra exertion, has a slight cyanosis of the lips, and a little dry cough, mostly in the mornings. He still, however, is quite able to work and, usually, is not concerned about his state of health. Many workers, in fact, will not admit symptoms of any kind at this stage.

Discussion of the Nature of the Roentgenologic Appearances seen in Asbestosis.—From my experience, the roentgenologic findings in asbestosis are largely limited to the lower half or lower two-thirds of the lung fields, whereas in silicosis, the opposite obtains. In asbestosis, the lesion may be bilateral or largely unilateral. Emphysema, blebs, or bullae always accompany well established changes in asbestosis and occur in the upper portions of the lung fields; whereas, in silicosis, the lower lung fields, to a large extent, are the site of these changes.

I have found it extremely difficult to determine the early manifestations of asbestosis. I believe an explanation for this would include the lack of an adequate pre-employment examination, in which a careful fluoroscopic study of the respiratory and cardiac movements is made. It seems to me that fluoroscopy, directing special attention to costal and diaphragmatic expansion, is essential in the determination of early changes in asbestosis.

If one can assume that the pathological process is due to changes secondary to the lodgment of asbestos fibers in the smaller bronchioles, the roentgen findings might be explained as follows. Disturbance in the roentgen appearance would not be seen except following the lodgment of the fiber in the smaller bronchioles. Sufficient time would have to elapse until a definite collar of fibrosis had become manifest so extensively that it would limit the ingress and egress of air into the lung areas distal to the point of obstruction.



FIGURE 27

Asbestosis, early manifestations indicated by the beginning obliteration of the costophrenic sulcus. Moderate hypoventilation of the lower lung fields due to thickening of the parietal pleura and slight thickening of the interlobar pleura.

In such damaged bronchioles, it is justifiable to anticipate that the lack of ventilation and drainage, distal to the point of obstruction, might become an ideal site for the development of infection or atelectasis. The infection, in turn, would increase the amount of serum protein in the tissues and assist, or stimulate, a pre-existing tendency for fibrosis. The fibrosis, itself, might limit the amount of normal lymph flow and, in succession, the infection would extend to and involve neighboring areas. When a sufficient amount of tissue had become involved, one might be able to demonstrate beginning changes in the roentgenogram, such as beginning obliteration of the costophrenic sulcus and fluoroscopically, some limitation of costal and diaphragmatic expansion. (Figure 27) I should assume, when such an appearance is observed in the roentgenogram, that the process might be regarded as moderately advanced.

The next step in the pathological process would seem to include an extension of the affection to the periphery. With involvement of the periphery, there is a definite limitation in the respiratory movements, which would be extremely slight in the early case of asbestosis but marked in the more advanced degrees of the disease. (Figures 28 and 29) The ribs are somewhat fixed and in thin individuals, they are dependent. In fat persons, they change very little. The interspaces may become quite narrow.

In the consideration of the healthy chest above, I called attention to the importance of the respiratory movements in lymph flow, and also to the fact that any restriction of these movements would tend to limit the flow of lymph. Any limitation of the flow of lymph increases the development of pre-existing pathologic changes. It was, likewise, emphasized above, that thickening of the pleura occurs to a greater extent in the lower thoracic region than it does in the upper, the explanation for this being the powerful costal and dia-

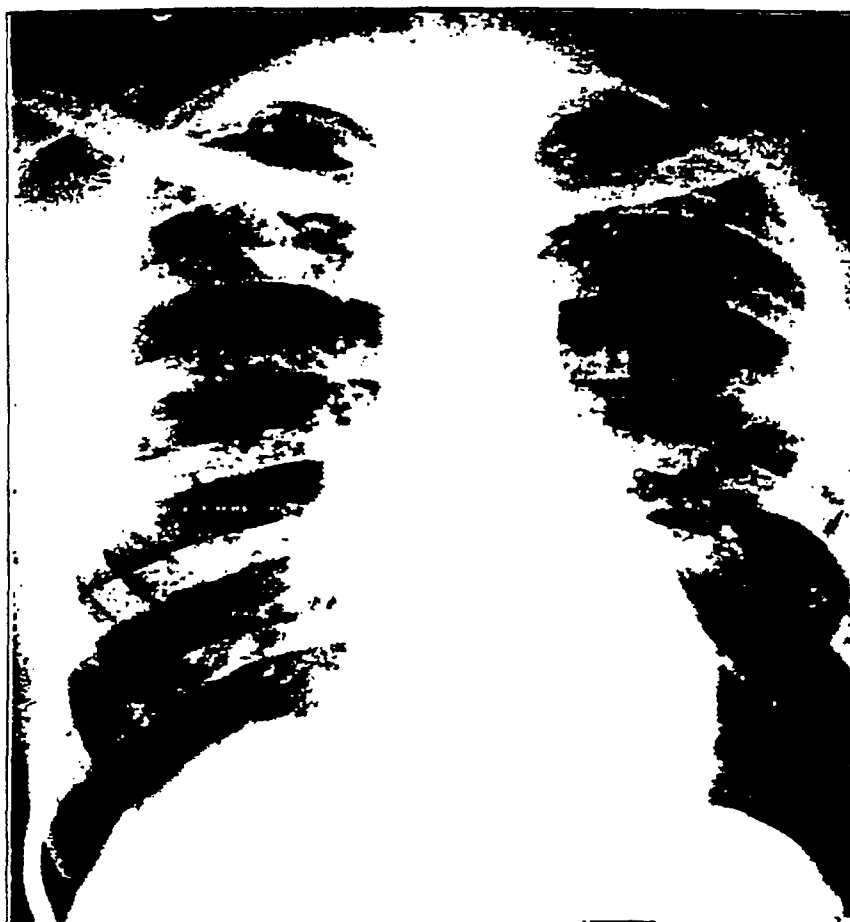


FIGURE 28

Man aged 41; silk weaver for twenty years, asbestos weaver for five years. The roentgenographic findings are within the limits of a healthy chest.

phragmatic movements in the lower chest and the relative fixation of the upper chest.

Almost every autopsy protocol has called attention to the

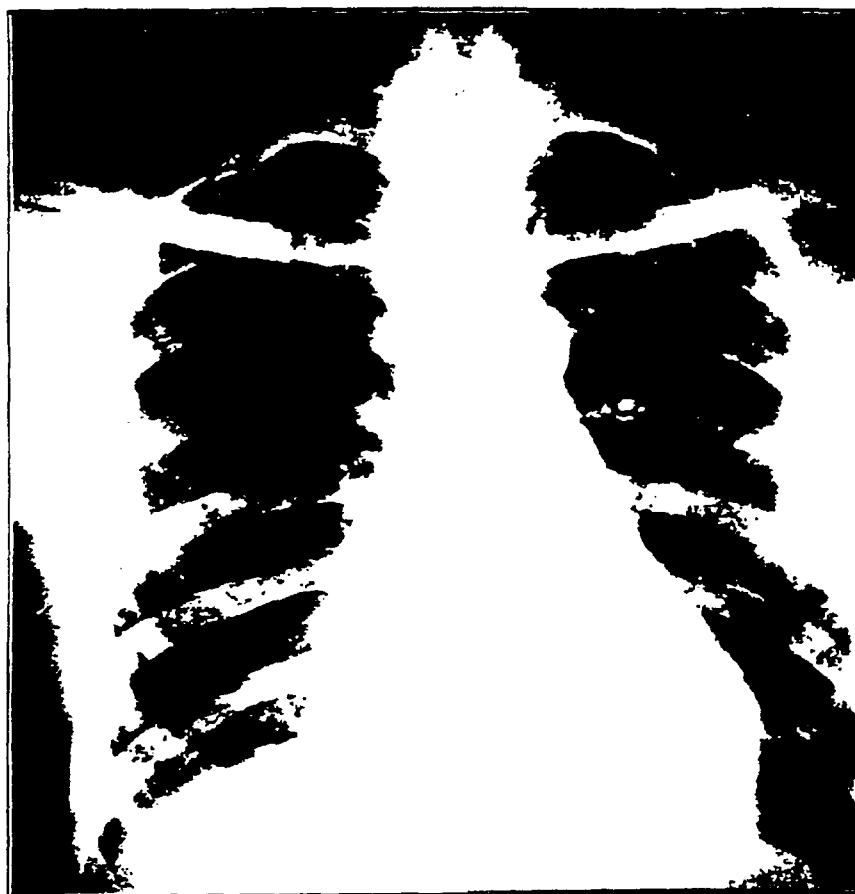


FIGURE 29

Asbestosis moderately advanced. Same individual as illustrated in Fig. 28. Examination made five years after that illustrated in Fig. 28. Note the relatively marked changes in the right lower lobe, which include thickening of the parietal and diaphragmatic pleura, obliteration of the costophrenic sulcus, slight thickening of the interlobar pleura, some narrowing of the interspaces and enlargement of the cardiac silhouette. There is evidence of hyperventilation in the upper lung fields.

marked thickening of the pleura and occasional thickening of the pleuropericardium. It is my feeling that these definite changes in the pleura can be adequately explained upon the basis of an underlying infection of the lung, extending to the pleura with subsequent thickening of the structure, due to the powerful respiratory movements and cardiac pulsations. (Figures 30 and 31)

The haziness of the lung fields, called attention to by almost every writer, can be explained by pleural thickening and a lack of ventilation. One should be careful in distinguishing between the shadow caused by pleural thickening and that resulting from the subcostal muscles. This has been discussed under the healthy chest.

I have been forced to place less and less importance upon observations concerning the trunk or vascular markings. These shadows are so changeable with variations in phases of respiration and cardiac pulsations that, unless one is re-examining patients and employing critical technics, such as making exposures during forced inspiration and during the systolic or diastolic phase of the cardiac cycle, one is absolutely confounded with changes in the size of the shadows, entirely within physiologic limits.

However, when changes develop sufficiently, one can see disturbance in the movements of the domes of the diaphragm and of the ribs, and obliteration of the costophrenic sulci. From this point on, the pathologic process in the lungs seems to advance fairly rapidly, exactly as one would expect it to do. This can readily be demonstrated by comparing the vertical diameter of the thorax in serial examinations of an individual who has a well developed asbestosis. If a period of at least five years is allowed to elapse, the vertical dimension is materially reduced. This observation corresponds to the limitation of the movement of the domes of the diaphragm, and, in a rough way, indicates the ability of the individual to



FIGURE 30

Asbestosis moderately advanced. Close-up of the right lower lung field in the individual illustrated in Fig. 29.



FIGURE 31

Asbestosis moderately advanced. Note the position of the ribs, the hypoventilation of the lung fields and thickening of the parietal pleura, which, in turn, are responsible for the ground-glass appearance of the lung fields. The interlobar pleura between the upper and lower lobes is thickened. From its position, one can readily see the extent of scar contraction already existent in the right lower lobe. The vascular trunk shadows have largely lost their identity.

ventilate the lung fields. The shadows are more pronounced in the bases and diminish as they approach the upper third of the lung field.



FIGURE 32

Asbestosis markedly advanced. The lesion occupies the lower lung fields and there is hyperventilation in the upper lung fields. Note the indistinct outline of the cardiac silhouette. This is probably due to thickening of the pleuropericardium and superimposed shadows in the lung fields. Note the dependent position of the ribs.

My comments thus far have been limited entirely to the lungs and pleura. The heart and pericardium also suffer in asbestosis, much more so in this occupational disease, in fact, than in any dust hazard with which I am acquainted. With a moderately advanced degree of asbestosis, the shadow of the cardiac outline is obscured by the shadows of superimposed lung structures and involvement of the pleuropericardium. (Figure 32) One rarely sees the cardiac shadow sharply demarcated, as observed in ordinary roentgenograms. The pleuropericardium becomes thickened and, in some instances, markedly so, for the same reason that the pleura over the lower lung fields and the domes of the diaphragm become thickened. Pulsations of the heart and infections extending to the pleura in this region play a major role, I believe, in producing the marked thickening of the pleuropericardium. These changes and those of the lung probably account for the right-sided hypertrophy and ultimate decompensation of the heart. In some instances, the cardiac silhouette is quite enlarged. Realizing, therefore, that ventilation of the lungs is seriously interfered with and that cardiac action is impaired, one can readily understand why these patients are embarrassed with dyspnea, weakness, and cyanosis.

In my present state of knowledge, if I were called upon to make a classification of asbestosis from a roentgenographic standpoint, I should include only two stages; asbestosis moderately advanced, and asbestosis markedly advanced; or asbestosis, first and second degree. I feel quite certain that the roentgenologic diagnosis of the clinically early stage of asbestosis is not entirely reliable. (Figure 33)

Prognosis. — It seems to be the rather general opinion that the prognosis in pulmonary asbestosis among factory workers is to be regarded as grave without adequate protection, in a large number of cases, among those who work



FIGURE 33

Asbestosis, an unusual type. This may be due to the fact that the individual has had some silica exposure. The shadows in the middle third of the right lung field are not typically nodular.

where there is great concentration of dust. The condition is apparently progressive, even after cessation of occupation. Merewether and Price⁷⁹ state that, with continued exposure to high concentration dust, fibrosis may be fully developed

in from seven to nine years, and that death may result in about thirteen years. With less concentration, fibrosis may not be fully developed for fifteen, twenty, or twenty-five years. Inhalation of dust in high concentration results in a more marked degree of fibrosis in a shorter time than when the concentration is lower.

There is no disease among the industrial hazards in which it is more important to have serial examinations than in the asbestos industry. Almost every investigator has been hampered by either no pre-employment roentgenologic examinations of the chest or relatively few opportunities for re-examinations of the individual exposed to asbestos dust.

Shull⁸⁶ states that it would seem that improvement could be expected in the early cases, but that, as the disease progresses, improvement is less likely. Sparks⁸⁶ feels that it is too early in the study of this disease to say very much on the subject. Lanza⁸⁵ believes that it is by no means certain that asbestosis progresses as does silicosis after withdrawal from dust exposure, nor that infection seems to be so closely and intimately associated with asbestosis as with silicosis.

My experience in this condition is also limited, but I feel quite certain that, when asbestosis can be diagnosed by roentgenologic examination, the condition is likely to become progressive, due to the important role exerted by fixation of the lung structures secondary to the pleural involvement. This can be readily demonstrated in serial roentgenograms, even in the absence of fine lung detail. The striking feature is the reduction of the perpendicular diameter of the chest.

Predisposition to Tuberculosis.—The condition of pulmonary asbestosis being a new child in the pneumoconiosis family, a great amount of interest centers around the incidence of tuberculosis. Among the early case reports referred to in the beginning of this occupational section, pulmonary tuberculosis was reported as an accompanying condition in a

few instances. Cooke's case had advanced tuberculosis. Simson reported 1 case which died with tuberculosis and 1 without evidences of the infection. He thought that we could not as yet formulate any definite opinion as to the frequency of the predisposition. Simson quoted Hoffman⁹⁷ as reporting 13 deaths among asbestos workers, 3 of whom had tuberculosis. He also quoted Collis⁹⁸ as reporting 5 deaths from tuberculosis among less than 40 workers at a factory. Wood and Gloyne⁹¹ referred to 3 cases showing evidences of tuberculosis at autopsy. Haddow⁸⁸ observed many asbestosis cases, among whom 4 died. He did not believe that the condition predisposed to the infection, at least not as a rule. Merewether and Price⁷⁹ stated that asbestosis differed from silicosis in the distribution of the fibrous tissue, in more rapid development, in roentgenographic features, and, possibly, in a *lessened susceptibility to tuberculosis*. Merewether^{80, 81} states that, out of 374 cases examined, evidence of active tuberculosis was found in 3. He thinks that, while there is no outstanding susceptibility proved, the question has not been fully settled as yet. I believe, however, that when tuberculosis does complicate asbestosis, that one can readily suspect or diagnose the tuberculous lesion with a fair degree of accuracy, due to the fact that the tuberculous process often occurs in the upper lobes, where there is usually very little evidence of asbestosis. If a basal tuberculous process were superimposed upon asbestosis, it would be very difficult, if not impossible, to diagnose it. (Figure 34)

Differential Diagnosis. — Passive Congestion of the Lungs as a Result of Cardiac Decompensation. — In this condition, the enlargement of the heart shadow, the abnormalities of the cardiac silhouette, especially in cases of mitral stenosis, and the clinical picture serve to establish the presence or absence of passive congestion. If asbestosis is absent, the appearances are likely to disappear with restoration of com-

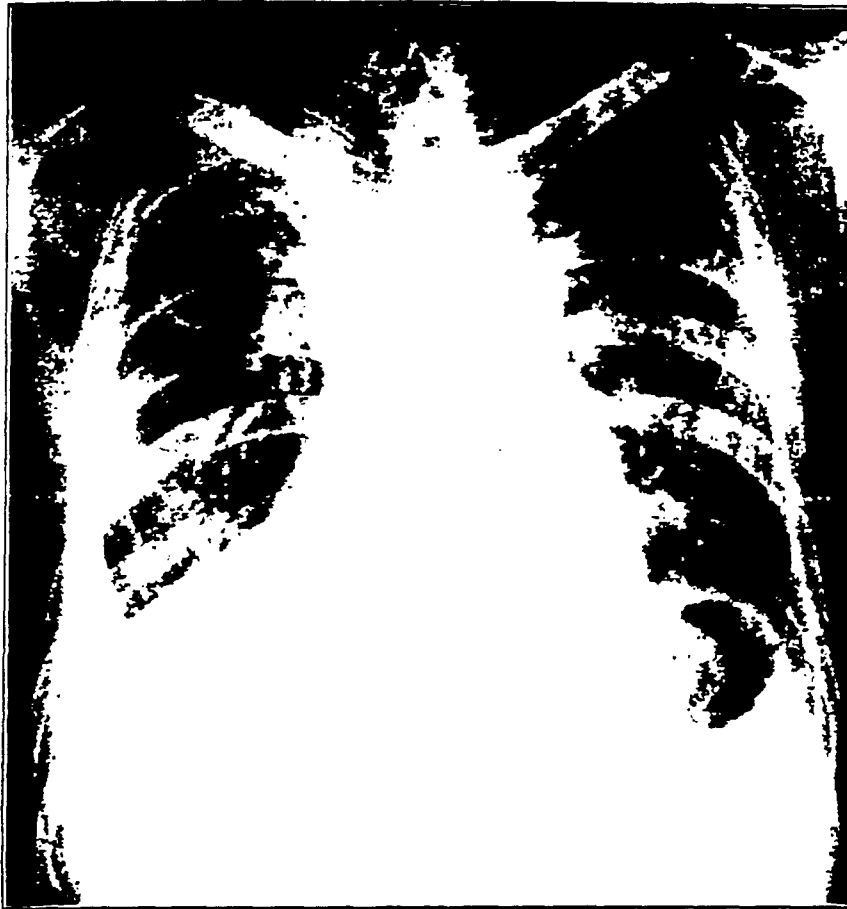


FIGURE 34

Asbestosis with a complicating tuberculous lesion in the right upper lobe. It is entirely possible that tuberculosis may account for the entire appearance found in this individual, but it was subsequently shown that this individual not only had tuberculosis but also had asbestosis in the lower lung fields.

pensation, but not altogether in long-standing cases. Marked emphysema is usually associated with asbestosis and is apt to cause a certain amount of rotation of the heart, so that the sagittal roentgenogram does not convey a direct impression of cardiac size or shape. The lateral view is always necessary to clear up the diagnosis. Any phase of asbestosis is likely to have its roentgenographic appearance greatly accentuated by passive congestion.

Advanced Bilateral Bronchiectasis. — The dilated bronchi, retained secretions and associated tracheobronchitis characteristic of this condition will produce the appearance of greatly accentuated trunk shadows, which may simulate that of the moderately advanced phase of asbestosis.

Other conditions that simulate asbestosis include polycythemia or erythremia and infiltrating malignant metastases, especially those extending from the mediastinum.

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The author has drawn rather freely from a number of the writings in which he has collaborated, in view of the fact that their observations largely represent his present views. When present opinions have differed, a discussion of the reasons for the change is included in the text.

IV. PATHOLOGY

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INTRODUCTION

DEFINITIONS

THE term *silicosis* will be used in this chapter to denote the changes which take place in the body tissues as a result of the inhalation of dust containing silicon dioxide, generally referred to in this connection as "free silica." The International Conference held at Johannesburg¹ in 1930 proposed in interpreting this term that the following conditions be satisfied, viz., that the silica must reach the lungs (a) in a chemically uncombined condition, although it might be mixed with other dusts; (b) in fine particles of the order of less than 10 microns in diameter; (c) in sufficient amount, and over a certain period of time.

For some years pathologists regarded free silica as the only siliceous dust likely to produce the disease, but recently "combined silica" or various mineral silicates found in industrial dusts have been included in the indictment and a new term, *silicatosi*s, has come into use to denote the types of disease thus produced.

The chief example of this new group of diseases has already become sufficiently important to receive a special designation, *asbestosis*, a disease resulting from the inhalation of the dust of asbestos, which contains the silicates of iron and magnesium.

As the diseases caused by these various substances differ

in their pathology, it will be necessary to describe them separately, but before doing so certain general considerations from a pathologist's point of view require discussion. Just as a chronic infective disease such as tuberculosis is regarded from the three-fold point of view of the organism, the portals of entry and the tissue reaction, so in the study of a pulmonary dust disease the nature of the dust, the conditions of its entry into the body, and the tissue reaction which ensues all need separate consideration.

Nature of the Dust. Mavrogordato² (1930) has classified the dusts which enter the lung into four types: (1) Dust that is harmless; (2) dust that is usually harmless but which may contribute to the lung mischief when inhaled in association with a harmful dust; (3) dust that is usually harmless and may actually have a prophylactic value when inhaled in association with a harmful dust; (4) dust that is harmful. The first group does not concern us here. In the second group may be placed such dusts as iron ore and coal dust in association with silica. The third group is a doubtful one. At the moment one would not feel justified in placing any dust in this category with certainty, for the pneumoconioses take many years to develop and surprises are not uncommon. The fourth and last group must include nowadays not only free but also combined silica.

Free silica occurs mostly in the form of mineral quartz. In the British Silicosis Compensation Scheme of 1928 it is referred to as silica rock, quartzose sand or 'any dry deposit or dry residue of silica.'

Combined silica includes a large number of silicates which tend to increase with industry. The commonest of those in use which are under suspicion as possible producers of pulmonary changes are enumerated by Middleton (1936).²⁰

The size of the dust particle is important. Only very small particles reach the lung tissues. Probably the majority, as

Mavrogordato suggests, are about the size of the common pathogenic micro-organisms.

— *The shape of the particle* has also a bearing on the resultant lesion. Broadly speaking there are 3 morphological types: (1) Small particles with irregular surfaces and sharp edges and diameters approximately equal. This is the common shape for quartz particles. (2) Long fibres of asbestos, which may extend up to 100 microns in length. Even if we regard the particle as inert, size and shape are of great importance because they determine the penetrating power of the particle. The long needle-like asbestos fibre readily "engages" in the respiratory bronchiole and easily comes to rest athwart the lumen of any narrow passage it may enter, whereas the small silica particle whose diameters do not differ greatly can readily penetrate to the peribronchial lymph spaces and the mediastinal glands. (3) Minute needles of fibrous sericite — a hydrous silicate of aluminium and potassium. These vary from 2 to 5 microns in length and have been described by W.R. Jones³ (1933) in silicotic lungs as occurring in 'piled-up bundles of needles,' hundreds of which are present, he states, for every grain of quartz that can be recognised. Although sericite had been previously described, it was not regarded as of great importance until the work of Jones. He regarded it, rather than the uncombined silica or quartz, as the important causative factor in silicosis. At the present moment the matter must still be regarded as sub judice. Obviously a series of carefully controlled pathological observations is needed in workers who have been exposed to the dust of quartz particles alone and sericite particles alone, but according to Drinker and Hatch⁴ (1936) quartz free fibrous sericite is difficult to obtain. Middleton has pointed out, however, that in Britain workers exposed to sericite without uncombined silica are found in

the china clay industry and that so far there has been no high incidence of silicosis in this industry.

The experimental evidence in laboratory animals falls outside the scope of this chapter.

Non-siliceous particles are found in practically all cases of silicosis and may cause confusion. Carbon, for example, is generally present in the shape of very fine amorphous particles, but coal dust may occur as elongated sharp jagged particles often up to 10 microns in length.

In the course of recent investigations on dust sampling, Briscoe, Matthews, Holt and Sanderson⁵ (1937) noted that dust which had been exposed for some time to the moist atmosphere of mines lost an unusually large amount of water on ignition, in striking contrast with the relatively small loss of weight on drying at 100°C, indicating that the dust had taken into firm combination a relatively large amount of water. In other words the comminution of the minerals had exposed fractured surfaces on which, at numerous points, there existed free and unsatisfied valency forces whereby water was attracted and held as water of constitution in the hydrated particle. Hydration of this nature can apparently liberate bases held in the crystal lattice. The writers found for instance that alkali was liberated in amounts equivalent to about one-sixth of the total alkalis present in the minerals. Obviously important new possibilities are opened up by these researches. Freshly formed dangerous dusts yield to water both alkali and soluble silica in larger amounts than do 'dead dusts' — i.e., dusts which have been exposed to air for some time.

The Portal of Entry. Probably only a small fraction of the inhaled dust reaches the pulmonary alveoli. The greater portion is deposited in various parts of the respiratory tract and expelled therefrom. In the case of asbestos workers, for

instance, the fibres can be found in the nose and throat and even occasionally in the conjunctival sac. The histological examination of the walls of the larynx, trachea and large bronchi in cases coming to autopsy usually reveals no lesions of asbestosis. Probably the same is true of silicosis though the writer knows of no investigations on the point. It may be assumed, therefore, that mechanical forces come into play to drive out the invading dust. Such are the muscular movements of the eyelids, the cleansing effect of the lachrymal secretion, the action of cilia and the outward flow of mucus in trachea and bronchi. In the case of cilia these protective phenomena have been demonstrated by Leonard Hill⁶ (1928). Using the trachea of horse, ox and sheep, he found that a suspension of lamp black in Ringer's solution was carried along the mucous membrane by the cilia at a rate which might be as fast as 3 centimeters in one minute and that fine particles such as those of iron and aluminium filings were readily passed along the trachea in this way. If a small area of ciliated epithelium was damaged by injury, cautery, or various chemicals (including chemical fumes) the transport of suspended particles was greatly reduced or even inhibited altogether. Irvine⁷ (1930) has suggested that repeated inhalation of silica dust may lead in the course of time to a dry bronchitis and bronchiolitis with denudation of epithelium. In this case the portal of entry is opened wide, as it were, and the penetrating dust will meet with little or no resistance. No doubt, too, some types of particles may be more readily expelled than others. These various factors may explain why different dusts seem to have a predilection for different parts of the lung.

The Tissue Reaction. Obviously the tissue reaction must, to some extent, be dependent on a variety of factors, viz., the duration or intermittent nature of the dust exposure, the size of the dose, the admixture of dusts, the age and sex

of the worker, and the condition of the respiratory passages prior to exposure. These factors take long years to observe in conjunction with employment histories and post-mortem findings and in many industries the necessary data for assessment are not yet available. Meanwhile the interaction of the dust particle and the tissues may be briefly and tentatively summarised as follows : —

(1) Many dust particles are doubtless expelled en route, but if a particle reaches the end of the journey in the distant parts of the portal of entry it "stays put." It does not multiply or become bacteriolysed like a micro-organism but remains as a more or less insoluble and irremovable irritant particle.

(2) The end of the journey for the silica particle is the peribronchial lymphoid tissue, but in the case of the asbestos fibre it is the respiratory bronchiole and the alveolus ; hence the difference in the pathological picture.

(3) Although bacteria may be found mixed with the dust, they are for the most part non-pathogenic organisms ; the dust particle can therefore be regarded as a non-infective irritant, and the tissue reaction is fibroblastic, ending in the production of the collagenous fibre.

(4) The amount of plasma exudation is minimal. The exudative reaction in the lung is characteristic of the bacterial diseases.

(5) Although we speak of the silica particle and the asbestosis fibre as being practically insoluble, many investigators believe that a colloidal silicic acid is actually formed by a slow disintegration of the particles in the lung and that the morbid processes are the result of a toxic action. There is indeed experimental evidence in the work of Gye and Purdy⁸ (1922) in favour of such a view in the case of silica sol. Asbestos fibres are known to persist in the lungs of persons who have not been exposed to the dust for more than twenty years and any disintegrating action must therefore

be extremely slow. Sundius, Bygden and Bruce,⁹ (1936) indeed, consider that this chemical theory of slow disintegration of dust particles is still an open question.

(6) It follows that if the dust particle is practically insoluble or only very slowly soluble, the lung must in the course of time become choked with irremovable foreign matter. This dust accumulates chiefly in the lymphatic drainage system.

(7) The process of phagocytosis, so prominent a part of the tissue reaction to bacterial diseases of the lung is much modified. Many of the particles, notably the asbestos fibre, are too large to be engulfed by a single phagocyte and a group of these cells can often be seen surrounding but not engulfing the meal which is obviously too large for them. Again, the particle even if engulfed cannot be digested and disposed of in the same way as the more soluble protoplasmic bacterium. This may be one reason why the phagocyte appears to remain so long in situ in the silicotic tissue in experimental animals. Lastly the phagocyte is for the most part a large mononuclear macrophage and not the small polymorphonuclear leucocyte from the blood stream. The latter is the phagocyte, par excellence, of pyogenic cocci in the lung.

(8) Lymphoid hyperplasia. This is one of the striking features of the tissue reaction. Small collections of lymphoid tissue are normally situated on the outside of the walls of the small bronchi but they are so small as generally to escape notice. These minute lymphoid aggregations absorb much carbon pigment and are a striking feature in the lungs of all persons exposed to the dust of large towns irrespective of silicosis. They should be distinguished from true lymphatic glands. They become markedly hypertrophied as the result of the drift of the silicotic particles into them. In the course

of time collagenous fibres are laid down and constitute the first simple silicotic nodules.

SILICOSIS

MACROSCOPICAL APPEARANCES

As a rule the body of the silicotic is not markedly wasted unless tuberculosis has supervened. Cyanosis may be present. Deformity of the chest wall is rare, whereas in the fibroid disease of the lung of non-industrial origin which begins generally in childhood, flattening and deformity are common.

Lungs and Pleurae. On opening the thorax the lungs as a rule do not collapse. They are heavier than normal and frequently bulky. Pigment is variable in amount. In the British cases it is generally pronounced owing to the carbonaceous dust of the industrial towns and may indeed be extreme in amount. Emphysema is almost the rule, bullae at the apices being especially common, whilst all the free borders may be affected.

The essential feature of the silicotic lung, however, is the nodular fibrosis. This begins with small raised hard nodules or islets of fibrosis immediately beneath the pleura and in the peribronchial regions of the lung. The former can be felt by running the fingers lightly over the pleura and are less pigmented than those in the lung. At first the nodules are no bigger than miliary tubercles but as the disease progresses adjacent nodules coalesce into composite nodules or small irregularly shaped masses which look rather like pieces of hard black rubber embedded in the lung. They can be detected by the touch as the knife cuts through the lung substance and they stand out prominently on the cut surface as the crepitant lung tissue collapses, but a true 'gritty' feel is

only imparted to the knife, as a rule, by extreme cases such as the stone mason's lung. In this case small particles of quartz can actually be dug out of the nodule with the point of the knife. The nodules are usually more plentiful in the upper lobes where they readily tend to run together into masses of large size.

As the presence of the nodule is the essential feature for diagnosis, attempts have been made to arrive at a classification in terms of distribution and severity of the lesion. Simson and Strachan¹⁰ (1930) in describing simple silicosis as it exists in the South African mines give the following classification which is used by the Miners Phthisis Bureau :

- (1) Slight — lesions small and moderately numerous, or medium-sized to large but sparse ;
- (2) Moderate — lesions numerous and small or moderately numerous and large ;
- (3) Well marked — lesions numerous and large.
- (4) Very well marked — lesions very numerous and large.

These authors state that in estimating the degree of nodulation present the whole lung substance has to be considered for the obvious reason that the distribution may be limited or unequal. The term small is used for islets of fibrosis with diameters up to 2 millimetres, medium from 2 to 4 millimetres, and large from 5 millimetres up to a centimetre. The term sparse is used to describe the distribution of nodules on the cut surface of the lung of the order of one nodule in a square with a side of 5 centimetres ; moderately numerous signifies one nodule per square with a 3 centimetres side, numerous when the square has a side of 2 centimetres and very numerous when the square has a side of less than 2 centimetres.

The nodule must be distinguished from calcified deposits and from the small fibrotic patches of obsolescent tubercu-

losis. As noted above, the silicotic nodule in the British cases is usually darkly pigmented, whereas calcified deposits and patches of obsolescent tuberculosis are much less so.

In long standing cases the aerated lung tissue between the nodules collapses, thus causing the groups of composite nodules to run together into the large hard, dark pigmented masses already described. These masses are commonest in the upper lobes; they may be wedge shaped with the apex of the wedge towards the hilum like deposits of caseous tuberculosis; they are frequently subpleural, and beneath the apical pleura are associated with emphysematous bullae, and eventually they may occupy the greater part or even the whole of the lobe. (Figures 1 and 2)

In addition to this characteristic fibrotic nodule the pleura generally shows evidence of old pleurisy in the shape of thickening, usually patchy, with obliteration of the interlobar fissures, and the production of sessile adhesions closing portions or the whole of the pleural sac. A layer of soft plastic fibrin, the result of a recent terminal pleurisy is often found at the autopsy, covering the old thickened pleura. Pleural effusion is rare.

In the lung the most important change apart from the fibrotic nodule, is the presence of emphysema. This is so common as to form almost an integral part of the picture. It occurs in the shape of bullae beneath the apical pleura, and along the thin anterior margins of the lungs, similar to the emphysematous lesion in other lung diseases. But it is seen also in another characteristic form, viz., a compensatory emphysema of the deeper portions of the lung tissue giving rise to an irregular honeycomb appearance.

The later stages of the disease are often characterized by a septic bronchitis with muco-pus plugging the bronchi, and a terminal reddening of the lower lobes from congestion



FIGURE 1

Silicotic lung showing massive nodules best marked in upper lobe.

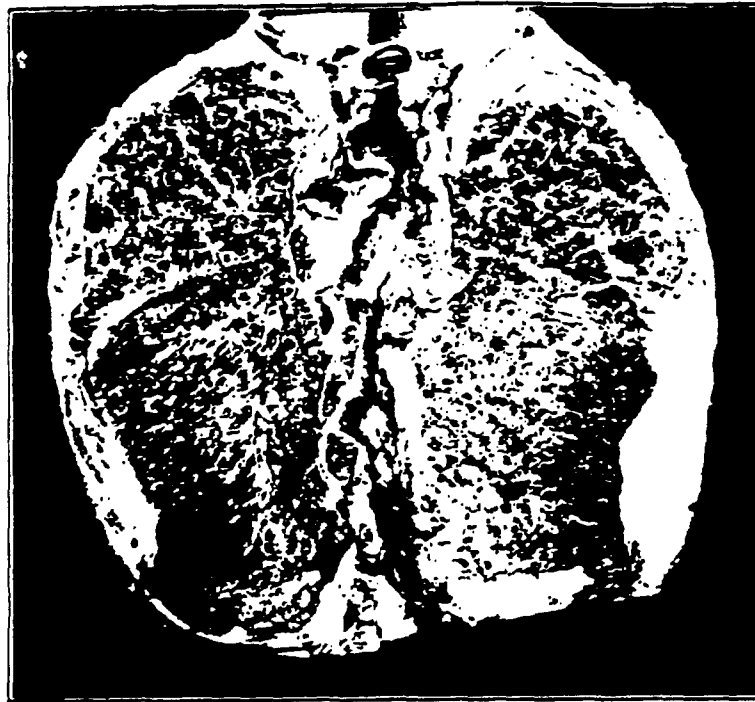


FIGURE 2

Silicotic lung showing extensive nodulation especially in lower lobes.

which may go on to a true red hepatization. These terminal conditions will be referred to again under the complications and sequelae of the silicotic lung.

In uncomplicated cases little of importance may be found in the other organs. Myocardial degeneration is not uncommon and is considered by some pathologists to be due to the obstruction in the pulmonary circulation as a result of the fibrosis, but it must be remembered that, as a rule, silicosis as seen in the autopsy room is a disease of the elderly industrial worker whose heart is likely to show signs of wear and tear in

any case. Although silicosis is believed to show a biochemical aspect there is as a rule no obvious toxic effect on distant organs in man, such as one finds in gout for instance, though lesions in the kidneys and other organs have been obtained by Gye and Purdy⁸ (1922) and others in experimental animals. Chronic interstitial nephritis may be found in silicosis but there are generally other reasons for its presence, (e.g. atheroma) and one cannot with any confidence claim that such lesions are simply the result of a silicotic toxin.

MICROSCOPICAL APPEARANCES

The Silicotic Nodule. The silicotic nodule is best studied when small and discrete and for this purpose the earlier the nodule is obtained the better. The key to the situation is to be found in the minute aggregations of lymphoid tissue which are normally located, in the lungs of man, in relation to the bronchioles, saccular ducts, small branches of the blood vessels and pleura. These aggregations of lymphoid tissue have been carefully described by W.Snow Miller¹¹ (1937) and should be distinguished from the lymph nodes which are found in intimate relation with the cartilaginous bronchi. These small collections of lymphoid tissue are simply minute aggregates of lymph corpuscles without the organised structure of supporting framework and capsule of connective tissue which a true lymph node of the cartilaginous bronchus possesses. In relation to the bronchioles they are found lying between them and the accompanying branch of the pulmonary artery. They are also to be seen along the course of the pulmonary artery and the pulmonary vein, in the interlobular septa and beneath the pleura, probably in this case in association with minute radicles of the pulmonary veins but not necessarily so. Miller has examined the lungs of elderly persons in order to determine the distribution of this lymphoid tissue in relation to the usual carbon pigment which

accumulates in the lung with age. The masses of lymphoid tissue around the bronchioles and the branches of the pulmonary artery were found to be hypertrophied into a common mass, whilst those associated with the pulmonary veins and the venous trunks in the interlobular septa were also hypertrophied. He further noted that these masses were usually found where alveoli were in the direct line of the air current against the septa. The lymphoid masses beneath the pleura are similarly hypertrophied.

These small masses of lymphoid tissue, therefore, are the sites of the formation of the silicotic nodule, a point which has been well stressed by Strachan and Simson¹⁰ (1930). Pathologists get very few opportunities of examining the nodule in its very early stages in man and the picture tends to be obscured by subsequent events, especially by the presence of pigment, but probably the sequence of events is as follows.

Amorphous particles of silica together with carbon pigment reach the saccular ducts and alveoli. Here they are taken up by large macrophages, generally designated by the unscientific but expressive term 'dust cells,' and reach the masses of lymphoid tissue described above. At this site these 'dust cells' come to rest and are surrounded by fibroblasts. In the course of time they degenerate and disappear leaving behind them the insoluble particles of silica and carbon pigment. Meanwhile the fibroblasts have given place to collagenous fibres and one or two minute capillaries may be formed from neighbouring vessels. The early nodule is now complete. When suitably stained it resembles a small round wicker basket in which the interwoven wicker work may be taken to represent strands of collagenous fibres. (Figure 3) In the centre are a few small capillaries, and lying between the fibres are particles of silica and carbon pigment. Surrounding the whole, new fibroblasts are formed producing in their turn more collagenous fibres. By this process the older fibres in the

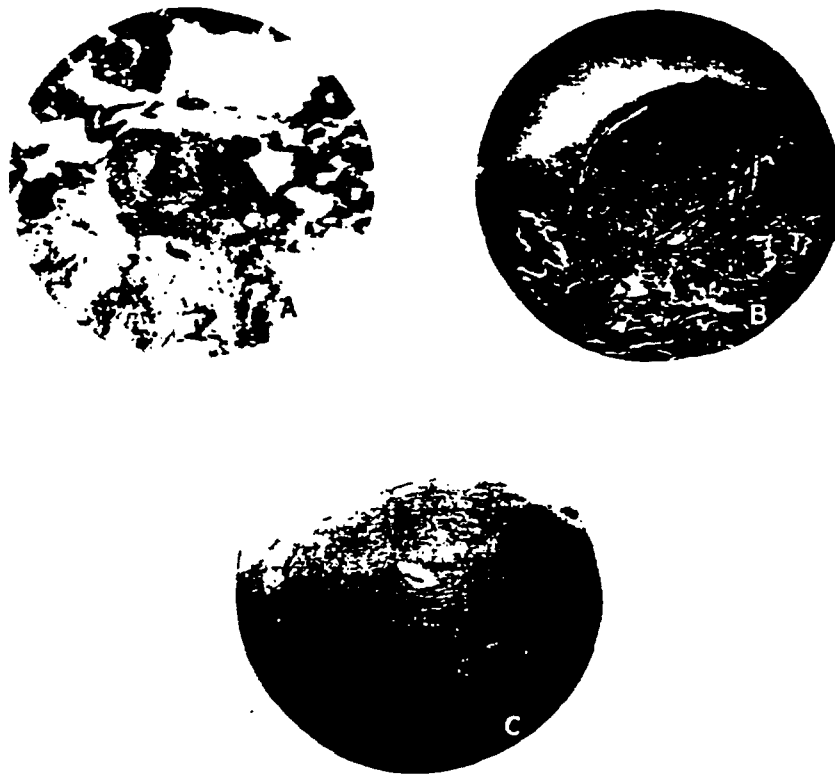


FIGURE 3

Discrete silicotic nodules. *a.* early stage ($\times 80$) ; *b.* intermediate stage ($\times 40$) ; *c.* late stage ($\times 40$).

inner parts of the nodule are compressed and come to lie closer together and have a hyaline appearance when stained. The capillaries too are compressed and may ultimately disappear. The picture is now less like a wicker basket and more like a large onion with tightly packed concentric layers. A circle of capillaries can often be found around the nodule in this stage, especially when the nodules are subpleural. In this

late stage the South African writers have also described both a fatty degeneration and a pre-calcification phase. In the course of time discrete nodules come into juxta-position by reason of the collapse of intervening alveoli. They then form an irregular composite nodule. As a rule the individual nodules forming this composite mass can still be recognised and around it there may be small areas of emphysema. (Figure 4)

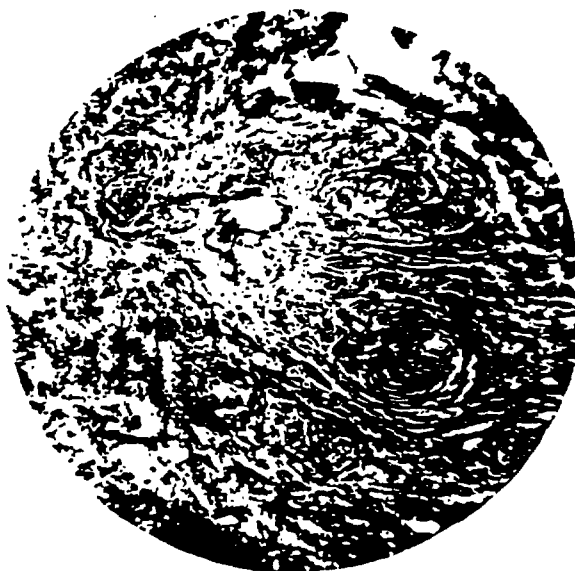


FIGURE 4

Composite nodules of silicosis (x 60).

The remaining portions of the lung show collections of macrophages in the alveoli, whilst desquamation of lining epithelium of the small bronchi is common. Hypertrophy of the aggregations of lymphoid tissue is the rule.

THE COMPLICATIONS AND SEQUELAE OF SILICOSIS

The following are the chief complications and sequelae noted in the post-mortem room.

Emphysema. This has already been referred to as an almost constant finding. Occasionally an emphysematous area may become sufficiently large to form a cavity filled with mucus and inhaled pigment from neighbouring bronchi. Such a cavity may be mistaken for a tuberculous or bronchiectatic cavity but as a rule it does not possess the well defined wall of either of these.

The Heart. The patient often dies with all the symptoms of cardiac failure. But this is a notoriously unsatisfactory term, and at the autopsy there is usually nothing to be found except a pale, soft, flabby heart muscle with some dilatation of the chambers. There are no valvular lesions, and histologically there is little to be made out.

Septic Bronchitis and Pneumonia. Secondary infection with one or other of the organisms associated with bronchitis and pneumonia is common, the streptococcus being the organism most frequently found. On section of the lung mucus exudes from the bronchi around which are areas of broncho-pneumonia.

The combination of a septic broncho-pneumonia and the nodular fibrosis of silicosis may be confused with chronic fibro-caseous tuberculosis; it is never safe to pass over the condition without microscopical investigation.

Histologically the lesions resemble those of septic bronchitis and pneumonia in general, but mention must be made of the hypertrophy of the minute aggregations of lymphoid tissue in the bronchial wall which may be confused with early microscopic lesions of tuberculosis.

Bronchiectasis. Dilatation of bronchi may supervene as a secondary result of the silicotic fibrosis. It is not very com-

mon and as a rule does not reach the stage of a fully developed foul smelling purulent bronchiectasis.

Tuberculosis. Next to septic bronchitis and pneumonia, tuberculosis is the commonest sequela of silicosis. Indeed, so common and so intimate a part of the pathological picture is it that some writers have declined to admit that such a disease as simple silicosis exists, and have attributed the whole condition to a chronic form of tuberculosis. Silicosis accompanied by tuberculosis is often referred to as 'infective silicosis.' This form of the disease will therefore be treated separately.

Carcinoma. Bridge and Henry¹² [1928] have proposed that cancer, in order to be classified as of industrial origin, should fulfil the following conditions: (1) The rate of incidence in the occupation under review should exceed that in the general population to a significant extent, and (2) in the occupation concerned there should be sufficient association of the worker with a substance proved experimentally to have carcinogenic properties. Suggestions have been made from time to time, notably in the case of the Schneeberg miners in Germany (Rostoski¹³ [1928] Schmorl¹⁴ [1928]) and again recently by Dible¹⁵ [1934] in England that carcinoma of the lung is likely to supervene upon pneumoconiosis of long standing. The data we possess at the moment however are not sufficient to satisfy Bridge and Henry's postulates and we cannot do more than regard the association of silicosis and carcinoma (and of asbestosis and carcinoma) with suspicion. The varieties of cancer found in association with silicosis are those found in intrathoracic malignant neoplasms in general (Gloyne¹⁶ [1930]). They are: (1) growths involving the mediastinal glands (2) growths involving the bronchi.

The primary growth involving the mediastinal glands is a rapidly growing white tumour situated in the neighbour

hood of the bifurcation of the trachea and along the course of the two main bronchi. The glands become adherent to each other as they enlarge and coalesce into an irregular collar of growth surrounding the lower end of the trachea, the two main bronchi, and the great vessels. For many years this type of growth was described histologically as a lymphosarcoma, but it is now generally regarded as a carcinoma, an oat cell carcinoma, by reason of its characteristic oval cells, though it is not clear from what part of the respiratory tract it arises. The deep layer of cells of the epithelial lining of the main bronchi has been suggested.

The other type of malignant growth is more clearly bronchial in origin. It may arise in large or small bronchi and may be single or multiple. It needs to be carefully distinguished from the type of growth described above which has a predilection for the mediastinal glands. These primary bronchial growths extend along the bronchial wall pushing their way through the silicotic tissue as they grow. Ulceration and necrosis with cavity formation is common. This last point is important because the growth cavity may easily be confused with a pyogenic abscess or bronchiectatic cavity. Microscopic sections, therefore, should never be neglected. Histologically they are, for the most part, squamous cell carcinomata possessing the usual characteristics with prickly cell arrangement, keratinisation, and cell nest formation.

The precise relationship of these tumours to the silicosis in the matter of causation is of course impossible to define. This subject will be referred to again under the heading of asbestosis and carcinoma where the pathological relationship has been worked out further.

SILICOSIS AND TUBERCULOSIS

If, when silicosis is accompanied by tuberculosis, the two pathological processes were always found side by side as they

are in silicosis and pneumonia, there would be little difficulty in differentiating between the two diseases. Unfortunately, this is not always so. The two are often intimately associated in such a way as to produce what is tantamount to a separate disease. In theory, there are three possibilities in the modes of onset and of progress : (1) the tubercle bacillus is already implanted in the lung before the silica exposure begins, the tuberculous focus being either (a) quiescent or (b) active ; (2) the tubercle bacillus reaches the site as a secondary infection in a lung which has already become silicotic, the silica exposure (a) having ceased or (b) being still in operation ; (3) the tubercle bacillus and the silica particles reach the healthy lung more or less simultaneously. This third possibility must be rare. The legal mind is very much alive to these possibilities and questions relative to them are frequently asked of medical witnesses in court. Unfortunately Nature does not always paint her pathological pictures with an eye to these fine distinctions and we must be careful to distinguish between the post-mortem appearances actually found and the inferences drawn from them as to the mode of onset. For descriptive purposes we may classify the various findings of the post-mortem room into three main groups, though it will be realized that in actual practice the picture is not always so well defined : (1) silicosis with obsolescent tuberculosis ; (2) silicosis with open tuberculosis ; (3) tuberculo-silicosis.

Silicosis with Obsolescent Tuberculosis. Three cardinal signs of obsolescent tuberculosis are generally recognisable : (1) dense pleural adhesions, especially in the upper part of the pleural sac tethering the apex of the lung to the apical pleura ; (2) fibrotic contraction and scarring of the visceral pleura and subjacent lung tissue without adhesions, again especially well marked at the apices ; (3) calcified nodules. In the presence of a chronic fibrotic disease of the lung such

as silicosis these signs are often difficult to interpret. Calcification is probably the most reliable, but the South African pathologists have described small deposits of calcium in the silicotic nodule, and of course, calcification of non tuberculous origin may take place in a vessel wall or around a foreign body. A calcified nodule beneath the pleura, or attached to the wall of a small bronchus, or in a mediastinal gland generally means obsolescent tuberculosis. If calcified deposits are found in the mesenteric glands, this fact may be taken as corroborative evidence. Generally speaking, however, silicosis found in the presence of obsolescent tuberculosis does not present any unusual features.

Silicosis with Manifest Tuberculosis. Frank active tuberculosis when it occurs is most commonly a terminal event. The following varieties are to be found: (1) pleural effusion, tuberculous empyema, or pyopneumothorax; (2) old cavities, often multilocular, with fibrotic walls having silicotic nodules adjacent thereto or actually forming part of the wall; (3) rounded foci of caseous tuberculosis with a sharply defined black rim of compressed pigmented lung tissue surrounding the foci; (4) miliary spread from an older tuberculous focus; (5) tuberculous broncho-pneumonia (which cannot always be distinguished from that due to other organisms without microscopic examination). We have here a composite picture resulting from the invasion of the pulmonary tissues by two very different types of invaders. The one is an insoluble particle which reaches the distal parts of the bronchial tree but does not reproduce, the other is a parasite with a capacity for multiplication and for being lysed by the tissues. The former remains strictly bronchogenic in its spread, the latter soon reaches the blood stream and is disseminated widely. This applies also to the next group, but to a less marked degree.

Tuberculo-Silicosis. It is convenient if not essential to have

a special term by means of which to designate a form of silicosis accompanied by tuberculosis in which the one disease is modified in association with the other. The term tuberculo-silicosis is used in this chapter in this sense only.

The modification is generally in favour of the tuberculosis, which is frequently of such a chronic fibroid type that it cannot be recognised until histological examination is made.

The first and essential feature is the excessive fibrosis. The second characteristic is the closely interwoven texture of the two disease processes, tuberculosis and silicosis, in contradistinction to the picture seen in silicosis with manifest tuberculosis. The third is the tendency to calcification.

Tuberculo-silicotic nodules vary a good deal in appearance as a result of the distribution of pigment, the silicotic portion being as a rule darkly pigmented and the tuberculous portions free from pigment. The result is that at least four different types of nodules may be recognized : (1) a nodule with a soft gray tuberculous centre and a pigmented silicotic periphery (2) a nodule with a darkly pigmented silicotic centre and a gray tuberculous periphery (3) a pigmented silicotic centre surrounded by a ring of small gray satellite tubercles (4) tuberculous and silicotic nodules side by side, the one compressing the other. These various types of nodules may result in the affected portions of the lung having a mottled or marbled gray-black appearance. (Figure 5)

Histologically the tuberculo-silicotic nodule has the following characteristics : (1) a tendency to be more irregular in shape than the simple silicotic discrete nodule, the foci running together more readily into composite nodules (2) collagenous fibres form only part of the nodule, the rest being caseous material (3) minute deposits of calcium are not uncommon (4) giant cells are few and generally lie just outside the nodule (5) tubercle bacilli are variable, the more caseous looking the nodule the greater is the likelihood of

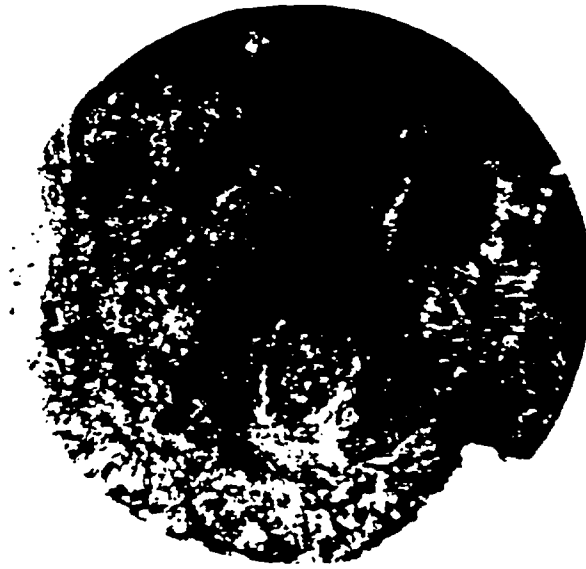


FIGURE 5

Tuberculo-silicotic nodulation, the upper half of the photomicrograph showing caseous nodules, the lower a tuberculo-silicotic nodule with a caseating centre (x 80).

finding them ; they should be searched for in the neighbourhood of the giant cell (6) lastly, small branches of the pulmonary artery may exhibit an obliterative thromboarteritis which may be mistaken for silicotic nodules.

Of course, not all nodules found in a case of tuberculo-silicosis exhibit these characteristics ; simple silicotic nodules and uncomplicated fibro-caseous tuberculous nodules occur.

Kettle and Archer¹⁷ [1933] have investigated some of the problems encountered in this disease, which are particularly difficult in Great Britain, where a low-grade tuberculosis with excessive fibrosis is common apart from dust hazards. They believe that the differential histological diagnosis depends upon the amount and distribution of collagen

in the tissue reaction. This is a matter of degree and they cannot define any delimiting standards. They suggest, however, that a chemical analysis for silica will often help when taken in conjunction with the other findings. In their experience an 'ordinary dried lung' (amongst which may be included uncomplicated tuberculosis) does not contain more than about 0.2 per cent of silica whereas in the tuberculo-silicotic lungs they investigated, analyses up to 2.7 per cent were obtained. It goes without saying that a number of different portions of lung should be selected for analysis. (Figure 6)

SOME UNUSUAL PATHOLOGICAL VARIETIES OF SILICOSIS

The range of industries whose workers encounter free silica is a wide one and it is not surprising that certain forms of the disease should, from the pathologist's point of view, appear unusual. At least three of these call for special descriptions.

Silicosis in Coal Miners. This condition is occasionally referred to as anthraco-silicosis and is met with in a variety of forms in those parts of the coal field which show a silica risk. A great deal has been written on this subject and a distinction is not always made between pure anthracosis and anthracosis with silicosis. One of the best recent accounts is that of Cummins and Sladden¹⁸ (1930) dealing with miners in the Welsh coal field in the 'hard headings' of which silica dust is present. The outstanding feature of their cases was a lung showing deep black to dark gray consolidated areas on a background of chocolate coloured or gray aerated lung substance. The consolidated areas usually varied from small 'maculae' in the lower lobes to large confluent areas in the upper. Histologically the characteristic appearances were a diffuse fibrous hyperplasia of the lung with massive dust accumulation in the alveoli and lymphatics together with



FIGURE 6

Quartz particles from stone-mason's lung. Photographed with dark ground illumination ($\times 1200$). (Courtesy of *Tubercle*)

the occurrence of fibrous nodules, some of which showed little or no lamination while others were markedly laminated or whorled.

Silicosis in Hematite Miners. Stewart and Faulds¹⁹ (1934) have described a series of cases in which both hematite and silica dust were present in large amounts in the lungs (sidero-silicosis). The hematite found was a ferric oxide producing a fine dry dust and gave to the affected parts of the lungs a bright red colour. The condition appeared to resemble closely the coal miner's lung in its histology, a diffuse or massive fibrosis with much dust accumulation and a 'few discrete rounded nodules of the usual silicotic type.'

In the one case the fibrotic areas appeared as bright red areas on a dark brown aerated lung background, in the other, the fibrotic areas were almost coal black on a background of chocolate coloured or gray aerated lung. Both these types of disease are of much interest to the pathologist and doubtless other similar conditions will in the course of time be described.

Broadly speaking there are two large groups of cases (1) one in which the free silica dust is preceded or accompanied by massive accumulation of a fine non-siliceous, non-toxic dust which acts chiefly in choking up the alveoli and the whole lymphatic drainage system of the lung, (2) a group in which the free silica is accompanied by a silicate which may have toxic and lethal properties of its own. (In this connection the writer is indebted to Dr.E.R.A.Merewether for a lung which shows the effects of exposure to free silica and asbestos.)

Acute Silicosis. Of recent years a number of cases of so-called 'acute silicosis' have been described, characterized generally by a short exposure to a siliceous dust and a rapidly fatal issue. The records have been collected and reviewed by Middleton²⁰ (1936) who gives the employment history of the largest group so far reported. In most instances there has been something unusual in the kind of dust to which the worker has been exposed. Often it has been a highly siliceous one mixed with an alkali or powdered soap for the manufacture of abrasive soap powders, and the suggestion has been made that a silica sol may be quickly formed in the presence of alkalis which is responsible for the rapid course of the disease.

Descriptions of the pathology are still very meagre. The first autopsy record was that of MacDonald²¹ (1930) and colleagues. They described the lower lobes of the lung as being solid, greenish-gray in colour somewhat resembling

marble, with innumerable tiny pinhead nodules of a lighter gray colour, whilst the upper lobes were still aerated but contained similar nodules. Microscopically there were large fibrotic patches which were practically acellular and were surrounded by small-celled infiltration. They found no histological evidence of tuberculosis and no tubercle bacilli.

Kettle ²² (1932) after examination of material from cases of acute silicosis considered that the sections showed 'acute tuberculosis enhanced, no doubt, by the silica present.'

Stuart McDonald ²³ (1932) recorded an example in which post-mortem examination revealed minute silicotic nodules in the lung not unlike miliary tubercles and strongly resembling the lesions of experimental silicosis in animals.

Chapman ²⁴ (1932) described a case in which the lungs, at autopsy, were about half the normal size, the middle two-thirds being shrunken, fibrotic, and almost as hard as stone. Histologically the fibrotic nodules were found to have hyaline centres.

Obviously the scanty nature of the pathological material available at present prevents our constructing a satisfactory picture of the pathology of the disease, and there is a danger of confusing the issue, as Kettle pointed out, with cases which are primarily acute tuberculosis. A distinction should be drawn between (1) workers who have had a very short but intensive exposure to siliceous dust but whose lungs, at autopsy, contain large and even massive patches of fibrosis resembling those of the ordinary chronic form of silicosis and (2) those with an equally short exposure but with miliary silicotic nodules resembling those of the experimental animal, as in Stuart McDonald's case. (The natural life of the laboratory animal is short and therefore more strictly comparable with an acute than a chronic silicosis in man.) It might be best, at the moment, to confine the term acute silicosis to this

second group. In any case it is inadvisable to include in the definition cases showing any evidence of tuberculosis, which can play such queer tricks in association with silicosis.

ASBESTOSIS

The word asbestosis was introduced into medical literature by Cooke²⁵ (1927) and Stuart McDonald²⁶ (1927) to designate that type of pneumoconiosis induced by the inhalation of asbestos dust. Several varieties of the mineral are used in industry, including chrysotile (white) crocidolite (blue) and amosite, the first named being the commonest. A good account of the geology of the mineral is given in a monograph by Hall²⁷ (1930).

NAKED-EYE APPEARANCES

In an uncomplicated case the pathological signs seen at autopsy are chiefly confined to the thoracic cavity, as follows.

Pleurae. Old dense adhesions are common, and are nearly always sessile, producing in the end complete symphysis of the pleura; the long pedunculated bands so frequently seen in tuberculosis are rare. Apical adhesions, however, occur and the interlobar fissures may be completely closed.

In the least affected portions the pleura retains its transparency but as the disease advances it assumes a typical ground-glass appearance and eventually exhibiting stiff, yellow and horn-like plaques some 2 to 3 inches in diameter. (The older writers on non-industrial fibroid lung were conversant with this type of thickening and likened it to cartilage.) At the apices there may be definite scarring, but whether this is to be regarded as an invariable sign of healed tuberculosis is doubtful. Here and there, generally near the apices also, the pleura may be thin over emphysematous bullae protruding from the lung. Lastly, covering and obscuring these signs of

old pleurisy are seen evidences of recent inflammation in the shape of reddish patches of lymph arranged in thin flat gauze-like layers or as small shaggy processes. Occasionally little patches of recent haemorrhage may be seen, such patches becoming browner and darker with age.

Displacement of the mediastinum is unusual.

The changes seen in the parietal pleura are similar to those found in the visceral pleura, but less marked and the horn-like plaques are absent.

Pleural effusion is rare, owing to the obliterative character of the disease.

Lungs. The asbestos worker is generally a town dweller and therefore the pathological picture, like that of silicosis, is superimposed upon a certain amount of anthracosis which forms a background. This confusion between the two is however greater than in silicosis.

The asbestosis lung is generally large and of increased weight, though as the disease progresses it may become contracted from excessive formation of fibrous tissue. It does not collapse when the thorax is opened, and like fibroid lungs in general, is firm, tough, and dense. (Figure 7)

On section blue-black patches of irregularly polygonal shape about $\frac{1}{8}$ to $\frac{1}{4}$ inches across are seen; they are hard and rough to the touch and are surrounded by a gray framework of thickened interlobular septa. This forms the typical unit of the asbestotic lung, corresponding to the silicotic nodule. In the upper lobe these polygonal areas are usually discrete with unaffected lobules lying between them. In the lower lobe they tend to run together and become confluent. Engorgement of the lung or an actual bronchopneumonia frequently provides a deep red background for the blue-black polygonal areas. Much pigment and the blue-black polygonal patches of asbestosis are found in excess in the peripheral portions of the lung immediately beneath the visceral pleura,



FIGURE 7
The asbestosis lung.

exemplifying the 'pleural drift' of the dust so common in pneumoconiosis. The interbronchial and other lymph glands of the middle mediastinal group which drain the lung, are intensely pigmented and fibrotic.

The pigmented polygonal areas of asbestosis are roughly coincident in size and shape with pulmonary lobules, and if examined with a hand lens the cut ends of small bronchi, sometimes dilated, can be seen. The fibrotic nature of the lung is better appreciated when the specimen has been fixed. The pulmonary vessels and bronchi now show thickened walls and the cut ends stand widely open, but the writer has not seen a typical extensive purulent bronchiectasis such as is sometimes described in chronic fibroid lung of non-industrial origin.

Occasionally the cut surface of the lung shows a honeycomb appearance due to the breaking down of alveolar structure analogous to the spongy emphysema described by Cummins and Sladden¹⁸ in anthracosis. This is commoner in asbestosis than in silicosis. Cavitation without tuberculosis, such as is described by Cummins and Sladden in anthracosis, has not been found in these areas.

Mediastinal Glands. The absorption of asbestos fibres into the mediastinal glands, is a much more difficult task for the lung than is the absorption of carbon or even small particles of free silica; but in the course of time the glands become small and hard with a thick fibrous envelope, and trabeculae are seen as white lines traversing them. When bronchopneumonia supervenes, they become secondarily enlarged and reddened.

Other Organs. Apart from the complications and sequelae to be dealt with later, there are few naked-eye changes in other organs. As in silicosis the heart muscle may be thin, pale, and flabby, and the spleen hard and fibrotic, but there is little else of importance.

MICROSCOPICAL APPEARANCES

The Asbestos Fibre and the Asbestosis Body. The asbestotic lung is essentially a dusty lung. It contains in addition to the asbestos fibres and the bodies formed from them, carbon pigment common to all town dwellers, jagged carbonaceous particles such as are present in coal miners' lungs, blood pigment, and metallic impurities of the asbestos dust which tend to accumulate in the neighbourhood of the asbestosis bodies. These various structures are apt to obscure the real causative factor, the asbestos fibre. If asbestos dust be examined with dark-ground illumination, it will be seen r.



FIGURE 8

Asbestotic lung showing the respiratory bronchioles packed with asbestosis bodies, fibres and pigment ($\times 360$).



FIGURE 9

Asbestotic lung showing reticular fibrosis of the alveolar walls
(X 150).

consist of fibres of varying length mixed up with particulate matter of irregular shape and often of reddish colour, representing various mineral impurities. The fibres themselves are straight and highly refractive, and of course, vary in length according to the thoroughness with which the crude material has been broken up. They may extend across the whole field of the microscope or they may be no longer than an anthrax bacillus. They can even be broken up sufficiently small to make a suspension which can be inoculated intravenously into the blood stream of the rabbit without producing emboli. In the raw material the fibres are held together in thick, straight bundles which, when crushed, are split up into

smaller and smaller bundles, until eventually very fine fibres are obtained. No branching has been observed. Under dark-ground illumination the fibres appear to be brightly illuminated like the filament in an electric bulb, and when very thin have a bluish luminosity. They are readily distinguishable from the irregularly shaped silica particles of the silicosis lung. (Figures 8, 9 and 10)

The asbestos fibre is of a highly resistant nature, hence its use in industry. It will withstand heat, and is not destroyed by mineral acids. It has been stated that with high-power magnification minute pores can be perceived in the fibre which render it capable of absorbing, to a certain extent, dyes



FIGURE 10

Asbestos fibres (crocidolite). Photographed with dark ground illumination ($\times 1200$).

such as methylene blue. The writer has not been able to confirm this.

The fibres are found in large numbers in the lungs. Their detection is readily achieved by destroying the surrounding lung tissue with concentrated sulphuric acid, but in ordinary stained sections the presence of these fibres is often obscured by other foreign particles.

In the lung of man two types of tissue reaction take place. The one is the thickening of the fibre, as a result of deposition of iron-containing material along its course, to form the asbestosis body. The other is the cellular reaction to the fibre.

The asbestosis body has had a curious history. In 1914 Fahr,²⁸ demonstrating to the Medical Society of Hamburg specimens and photomicrographs of the lungs from a case of pneumoconiosis occurring in an asbestos worker, observed the presence in the lung of a large number of crystals and stated that similar structures had been seen in 1906 by Marchand and Riesel, who had speculated as to whether they were due to the inhalation of asbestos dust or were a haemoglobin derivative. No pictures are given in Fahr's communication, but it is probable that what Marchand and Riesel and Fahr saw was the asbestosis body. However, these descriptions attracted little attention at the time and seem to have been forgotten.

The next step came in 1927 when Cooke and Hill²⁹ and Stuart McDonald²⁶ described certain 'curious bodies' found in the lungs of an asbestos worker who had died in 1924. Cooke had previously recorded the case in 1924 but had not at that time mentioned these bodies. Cooke and Hill wrote of them as follows: 'They are yellowish-brown in colour and do not stain with the usual stains, but give the Prussian blue reaction. There is a uniformity in appearance, group distribution, and fructating heads that make one think of a fungus. The hyphae are verruciform and discoid, and definite spores

are seen.' Pathologists to whom the material was submitted suggested various possibilities, viz., diatomaceae, casts of small cavities, particles of asbestos fibre that had become coated with colloidal matter, fungoid bodies.

Later in the same year, Stuart McDonald, to whom post-mortem material had been submitted from the same case by Cooke, gave a fuller description of the bodies. He regarded them as foreign bodies associated with the asbestos and was satisfied that they were neither vegetable nor animal in origin, and quoted another case of an asbestos worker in whose lungs he had found similar bodies.

The first illustrations were given by Cooke and Hill in their original description. Subsequently they were depicted by Simson³⁰ (1928) in autopsies of South African cases. His paper also included a microphotograph of the bodies produced experimentally in the lung of the guinea-pig by Mayr and Ordato, the first occasion on which the bodies had been so produced.

At this stage Stewart and Haddow³¹ (1929) made the suggestion that the bodies should be called 'asbestosis bodies,' a term which has now been generally adopted.

Finally Gloyne³² (1929) showed in the following experiment that the body consisted of an asbestos fibre coated with a substance soluble in a strong acid.

A wet preparation of the bodies was placed on the stage of the dark-ground illumination microscope, and the position of a readily recognisable group of these bodies noted. Concentrated sulphuric acid was then run very gently under the cover slip, the bodies being kept under observation all the time. They began slowly to dissolve, and in the middle of the structure of each appeared a typical asbestos fibre. At the end of half an hour practically all the material of which the bodies were composed had disappeared, leaving only faint ghost-like outlines with refractile asbestos fibres within them.

The diversity in shape of the bodies makes it difficult to write a satisfactory description of them. The following summary comprises the chief points: (1) Marked variation in length (24 to 60 microns) and breadth (12 to 24 microns). Gardner and Cummings³³ record them as long as 250 microns in experimental animals. The short forms are sometimes ingested by phagocytes. (2) Golden-yellow colour. Gardner and Cummings have pointed out the resemblance in colour to haemosiderin deposits in tissue in which haemorrhage has occurred. (3) Homogeneous structure. (4) The central fibre. This can be best seen by reducing the illumination. The fibre may sometimes be detected projecting beyond the body. (5) No differentiation with polarised light (Cooke and Stuart McDonald). (6) Tendency to be arranged in irregular clumps and clusters. (7) Very early forms unsegmented ('sausage shaped'). (8) Later forms crenated, segmented, or resembling a series of oval discs or beads strung together in necklace form. (9) Bulbous or pointed extremities (or one bulbous and one pointed suggesting 'heads and tails'). (10) Generally quite straight, occasionally curved, very rarely S-shaped. When examined in a wet preparation slight pressure on the cover slip will sometimes cause them to bend owing to the elasticity of the central fibre. (11) Forms seen 'end-on' look like knobs or door-handles. Small secondary knobs and bosses sometimes seen thereon.

The bodies do not stain with the ordinary aniline dyes, but according to Gardner and Cummings, Wright's blood stain gives them a 'greenish cast.' They will also retain neutral red slightly, but these partial reactions cannot be called true staining reactions. Cooke and Hill and Stuart McDonald, in their original descriptions, noted that the bodies could be stained by means of the Prussian blue reaction, and Gloyne has shown that they become black on treatment with ammonium sulphide. Haematoxylin, which also has an affinity

for iron, can be made to colour them to some extent, but it is more a deposit of stain than a true staining reaction.

With regard to their formation, no such bodies have been found in crude asbestos or in asbestos dust, and it seems practically certain that they are only produced when the fibres gain access to living tissues and remain there for a considerable period of time.

Gardner and Cummings showed that the golden-yellow material which covers the fibre contained iron, whilst Simson and Strachan put forward the view that the iron-containing substance was of cellular origin.

From a study of a large number of different forms of asbestosis bodies, the present writer has endeavoured to build up a complete picture of formation from the original inhaled fibre. Figures 11₁ to 11₃ show deposition of some material on the fibres to a varying extent. When completely covered the body thus built up has a sausage-like form without any differentiation from end to end (Figure 11₄). These thickened forms next begin to show definite fissures (Figures 11₅ to 11₇), giving the appearance of incomplete segmentation at irregular intervals. In Figure 11₈, this segmentation has advanced a stage further, the fissures now extending almost to the mid-line of the body and occurring at irregular intervals along its length. These fissures produce the appearance of a head or knobs at the ends (Figures 11₉ and 11₁₀). As they become deeper and more numerous a completely but irregularly segmented body results (Figure 11₁₁). When the fissures reach the straight central fibre they appear as (a) a small number of long segments as in Figures 11₁₂ to 11₁₈, or (b) a large number of short segments as in Figures 11₁₉ to 11₃₄. These short segments may be regular in size and shape resembling beads on a necklace (Figures 11₃₂ to 11₃₄), or the segmentation may be complete at one end of the body and incomplete at the other.

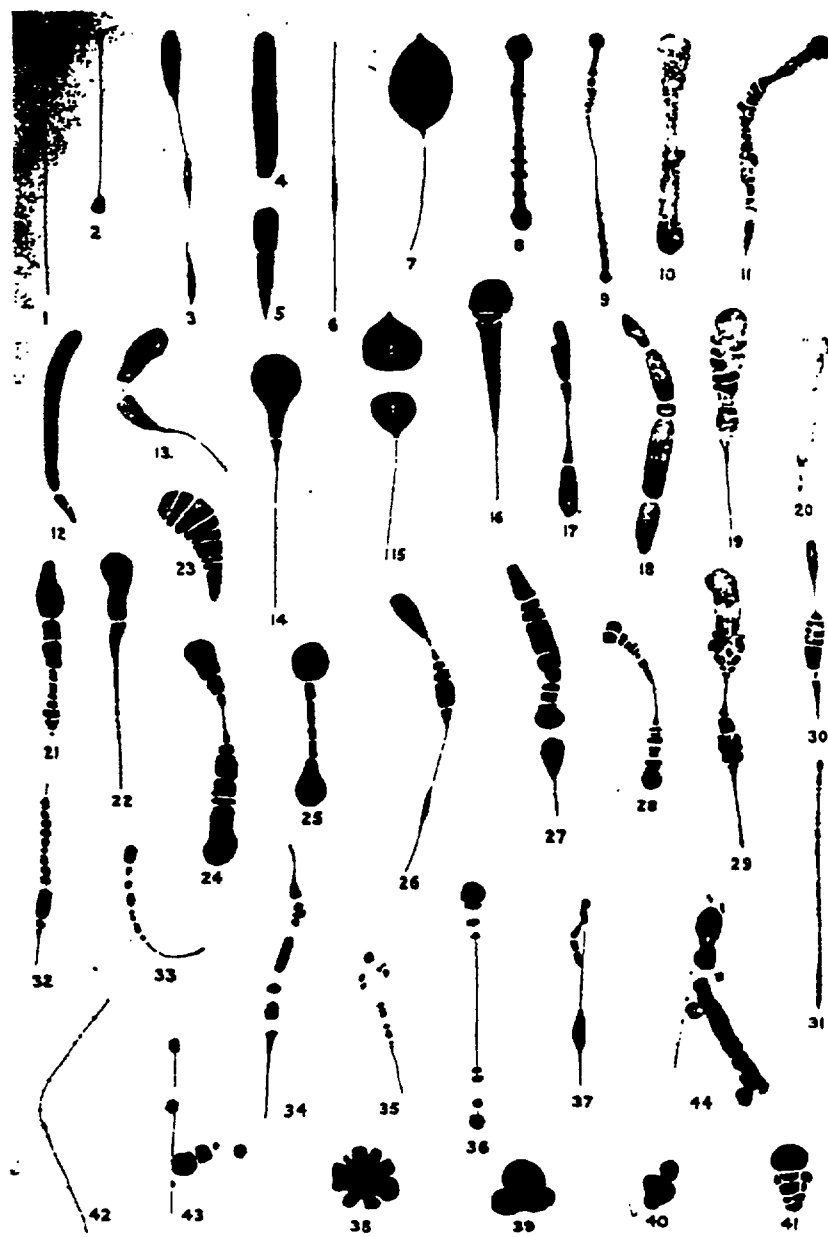


FIGURE 11
Stages of formation of the asbestosis bodies.
(Courtesy of *Tubercle*)

In this final stage some of the segments or even small portions break away leaving bare lengths of fibre between the remaining segments (Figures 11₃₅ to 11₃₇). Very occasionally these broken fragments can be actually seen in the process of breaking away. This is well shown in Figures 11₃₅ and 11₃₇. Bodies viewed end-on or obliquely are seen in Figures 11₃₅ to 11₄₁.

The asbestosis body would appear, therefore, to be formed from the original fibre by a process of (1) deposition of some material around the fibre so as to thicken it; (2) fissure of this material giving rise to the appearance of segments; and (3) fragmentation or the separating off from the asbestosis body of fractured portions of the deposited material.

The central fibre having been satisfactorily demonstrated, the question arises, of what does the golden material consist which forms the segmented coating? Iron certainly enters into its composition, for, as far as we know, all asbestosis bodies give an iron reaction. This metal is present in crude asbestos in varying amounts, but it is also present in haemoglobin. On the whole, the evidence is in favour of the iron being derived from the tissues — probably from blood pigment — but the point has not been conclusively settled, and it seems probable that the golden yellow substance is the product of a colloidal reaction between the fibre and blood protein.

Asbestosis bodies have considerable power of resisting the ferments of the gastro-intestinal tract when swallowed with sputum and have been found in the faeces. They also resist heat. The effect of acids varies a good deal. In sections of lung tissue they resist decalcifying fluid, which contains 7½ per cent of nitric acid, for several weeks. Concentrated sulphuric acid leaves only a mere semblance of a ghost-like body with a central fibre. Hydrochloric acid has the effect of bleaching the bodies; the yellow colour is taken out almost immediately

leaving a colourless skeleton of the body, suggestive of a minute hyaline urinary cast. Nitric acid has less effect and glacial acetic acid none. The bodies will resist 20 per cent caustic soda for twenty-four hours without losing their golden-yellow colour. They become black when digested with trypsin.

The inference from these reactions would appear to be that the asbestosis body consists of at least two portions surrounding the fibre — viz., an acid resistant material which forms a framework and gives the body the characteristic shape, and a golden-yellow pigment contained therein which is not acid resistant.

The question now arises, what inference can be drawn from the presence of the asbestosis body? Since these structures have not been found in any other disease, their presence is an indication of exposure to asbestos dust, whilst the fact that the bodies have not been found in asbestos dust itself implies that their formation takes place in the body — probably as a result of a colloidal reaction with the blood proteins in which the iron plays an important part. They have been found in the guinea-pig by different observers from seventy days to six months after exposure to dust. Simson has recorded the bodies in a human lung with a history of only two months' exposure.

Lastly, can it be inferred from the presence of the asbestosis body that fibrosis necessarily exists in the lung? The presence of the asbestosis body implies a tissue reaction to the fibre, but it does not necessarily follow that this reaction runs *pari passu* with the production of pulmonary fibrosis. On one occasion the writer found fine collagenous fibres in the tissues of a guinea-pig as early as twelve days after subcutaneous inoculation of asbestos fibres, but the amount was very minute. A heavy dose of fibres may be needed — and even then years may elapse — before pulmonary fibrosis is sufficient to be detected by physical signs or roentgenogram during life. It

would be unsafe, therefore, to accept the presence of the asbestosis body as undeniable evidence of fibrosis; all we can say at present is that the asbestosis body is an expression of a tissue reaction to a foreign body acting as a benign irritant, and that fibrosis of an appreciable extent may or may not be a part of that tissue reaction.

There is some evidence that asbestosis bodies are not formed with the same facility in all the tissues of the body. Thus they have not yet been described in the skin warts to which the workers are liable. The writer has examined without success sections of a wart which was known to have been present for many weeks, and has failed also to find them in the skin of a guinea-pig seven months after the asbestos fibres had been introduced into the cutaneous tissues. Dewirtz also does not make any mention of asbestosis bodies in sections of warts he examined from the skin of Moscow factory workers. Furthermore, even the lung varies in its reaction to asbestos in different animals. Gardner and Cummings³³ (1931) record that the lung of the rabbit apparently offers a much less favourable environment for the production of the bodies than does the lung of man and of the guinea-pig. The rat also does not produce bodies readily, whilst Schuster³⁴ (1931) failed to find them in the lungs of a dog which had been kept on factory premises for the purpose of ratting for several years, though fibrosis of the lungs was present.

The Lungs. The tissue reaction to the asbestos fibre in the lungs depends partly on the non-absorbable character of the particle and partly on its silicate content. There is little leucocytic reaction, the cells chiefly concerned being the lining epithelium of the respiratory bronchioles and the large macrophage which is the striking feature of the cellular reaction in many of the non-pyogenic pulmonary diseases. The bronchial epithelial cells do not phagocytose the asbestos fibre. They are simply desquamated into the lumen of the tube as

a mechanical result of the irritant action of the fibre. The macrophages are identical with the so-called 'dust cells' of the lung and surround the fibre in an attempt to engulf it. Frequently the fibre is too long to be completely engulfed and looks as though it was sticking into the cell with one end protruding, like a pin in a pincushion. Whether this is in all cases a true statement of facts is not quite certain owing to the liability of the long asbestos fibre to lie obliquely rather than in one plane. (Figure 12)

In addition to these tissue phagocytes, asbestosis giant cells and fibroblasts are seen. The former are foreign body giant cells probably resulting from partial degeneration of small collections of the above-described large phagocytes which have



FIGURE 12

Asbestosis bodies (low power) in unstained section of lung (x 520) (Reproduced from article by W.Burton Wood and D.S.Page) (Courtesy of *Tubercle*)

lost all their envelopes. In early lesions, e.g. in the guinea-pig, the cytoplasm has a pigmented stippled appearance in contradistinction to the almost structureless look of the tuberculosis giant cell. Sometimes the faint outlines of the degenerated individual cells which form this composite 'cell' can be seen. The cell also varies considerably in size according to the number of phagocytes composing it. Finally, the nuclei of the asbestosis giant cell are large and less numerous than those of the tuberculosis giant cell and have not the same tendency to be arranged round the periphery. The fibroblast does not appear to differ in appearance in asbestosis from the fibroblast of other varieties of pneumoconiosis. It is found around bronchioles and alveoli, in interlobular septa and in the subpleural connective tissue. (Figure 13)

The trachea and main bronchi are little affected in asbestosis. The bodies can be seen on the surface of the epithelium, having been carried there by bronchial secretion, and occasionally they may be seen in the mucous or fibrous layer of the wall of the main bronchus surrounded by phagocytes and free pigment. The writer has not yet seen them actually in the tissues of the trachea. In the intra-pulmonary bronchi, from the point where they enter the lung to the point where they terminate in respiratory bronchioles, the most marked change is the desquamation of the epithelium, and lying amongst the desquamated epithelial cells asbestosis bodies, fibres and loose pigment are frequently to be found, often plugging the lumen. The basement membrane is thickened and the fibromuscular coat surrounded by a ring of collagenous fibres. The elastic fibres generally remain intact, but in some places they are found to be contracted together as though definitely increased and when ulceration takes place they may be found in the sputum. Asbestosis bodies may be found in any part of the wall.

Gardner and Cummings³³ (1931) have shown by animal



FIGURE 13

The asbestosis giant cell showing phagocytosed asbestosis bodies and fibres (x 1200) (Courtesy of *Tubercle*)

inoculation experiments, however, that the chief site of asbestos dust localisation is the respiratory bronchiole. In man, asbestos fibres, pigment, cellular debris, phagocytes and asbestosis bodies—the latter frequently in radially arranged clumps—are all to be found choking the lumina of these tracts, blocking the narrow alveolar ducts, and filling the air sacs and alveoli. Lastly, around these distal portions of the bronchial tree and of the alveoli a fine network of collagenous fibres is formed, ultimately producing a generalised reticular fibrosis which is a distinguishing feature of the disease. When this diffuse fibrosis has reached an advanced stage all alveoli in the area may be compressed out of recognition. Whorled nod-

ules such as those of silicosis are rare but are occasionally found in these areas of advanced fibrosis. Asbestosis bodies are occasionally present in the proximal parts of the plexus of lymphatics around the smaller bronchi, and become more numerous towards the respiratory bronchioles and are plentiful at the level of alveolar ducts. The lymphatics accompanying the branches of the pulmonary artery less frequently show asbestosis bodies or pigment. In the small lymph-nodes draining the lung tissue asbestosis bodies as a rule are not plentiful owing to the difficulty the fibres encounter in entering the afferent ducts. The aggregations of lymphoid tissue in the walls of the small bronchi are hypertrophied.

Changes in the vascular system are relatively unimportant, presumably because the toxicity of asbestos is not marked. There may be an increase of collagenous fibres around branches of the pulmonary artery and asbestosis bodies may also be seen lying in the connective tissue between the artery and the wall of the bronchiole. The capillaries are of course involved in the reticular fibrosis of the alveoli wall of which they are the chief constituents and may be compressed almost to the point of occlusion. The pulmonary venules are also involved in this fibrosis. Lynch and Smith (1930) have reported asbestosis bodies in the thrombi of veins.

The Pleura. The surface of the visceral pleura is generally extensively denuded of epithelium, with patches of fibrin containing a few desquamated endothelial cells lying on the denuded surface. The subendothelial portion is much thickened by the formation of collagenous fibres with many fibroblasts, and new formed blood-capillaries are numerous, especially in the deeper layers. Asbestosis bodies are scanty and tend to be short and stumpy — probably the larger fibres do not penetrate so far — and usually lie surrounded by connective tissue in small groups. Fibres are also occasionally seen, singly or in sheafs. The elastic layer is generally intact.

The interlobular septa may show asbestosis bodies, fibres and pigment with or without collagenous thickening. In two cases whorls of connective tissue resembling discrete silicotic nodules were found by the writer near the point where the septum joined the pleura — which is the point at which the pulmonary deep lymph-vessels in the septum anastomose with the subpleural lymph-vessels.

Histology of other Organs. H.L. Stewart, Butcher and Coleman³⁵ (1931) have found asbestosis bodies in the spleen. The present writer has failed to find them in any of the abdominal organs, even in the walls of the stomach and intestines where swallowed sputum might conceivably carry the bodies.

To summarise, the typical histological lesions of asbestosis are three : (1) The pouring out of large macrophages into the most distal portions of the bronchial tree and of the alveoli to surround and as far as possible phagocyte the invading fibres ; (2) the formation of the asbestosis giant cell ; (3) a generalised reticular fibrosis surrounding the bronchioles, alveolar ducts, alveoli and air sacs, capillaries and venules, the interlobular septa and pleura, ultimately resulting in a complete obliteration of all lung structure.

COMPLICATIONS AND SEQUELAE

The chief complications and sequelae of pulmonary asbestosis are as follows : —

- (a) Purulent bronchitis.
- (b) Bronchopneumonia, which is best seen in the parts of the lung least affected with the asbestosis. This point is probably of importance because it explains why bronchopneumonia is such a fatal disease in asbestosis — it puts out of action the last remaining functioning portions of the lung.
- (c) Pulmonary tuberculosis. In a series of 100 cases published by Burton Wood³⁶ (1934) and the writer, active

tuberculosis was found in 21. The cases coming to autopsy have been of the acute caseous type (in one case with a generalised miliary spread), with few Langhans giant cells and no obvious sign of healing. Asbestos fibres, asbestosis bodies and tubercle bacilli have been found lying side by side in the caseous centres, but there is evidence in animal experiments (Gardner and Cummings) that asbestosis bodies disappear in caseous areas. In the series quoted above there was one case of pyopneumothorax with lardaceous disease following treatment by collapse therapy. A number of cases of healed and obsolescent tuberculosis have also been found at autopsy. These are probably not more numerous than in post-mortem work in general, but they indicate that the supervention of asbestosis does not necessarily mean the lighting up of quiescent tuberculosis.

(d) Empyema. In the series mentioned above there was one case of tuberculous empyema.

(e) Bronchiectasis. Dilated bronchi are occasionally met with, but bronchiectasis with fusiform or cavity-like dilations containing foul pus have not been encountered by the writer.

(f) Carcinoma has been found in 6 cases examined by the writer at autopsy. Until more statistics are available it is impossible to draw any very definite conclusions as to the relation of the two diseases. The points noted up to the present are (1) 5 out of the 6 cases of carcinoma have been of the squamous variety, the sixth being an oat cell carcinoma; (2) 4 out of the 6 have been associated with asbestosis which was fairly advanced and of long standing; (3) in 4 cases the carcinoma consisted of an isolated nodule only; (4) the nodules were in the portions of the lung most affected by the asbestosis—i.e. basal or peripheral; (5) 2 only showed secondary growths, one in pericardium, thoracic wall, liver and kidney, the other in thoracic vertebrae; (6) histologi-

cally the squamous carcinoma appeared to advance along the mucosa of the small bronchi, pushing aside the pigment, asbestos fibres and asbestosis bodies as it grew. whilst in the 1 case of oat cell carcinoma observed, the tumour grew outwards from a centre in a compact mass without any tendency to confine itself to prolongation along and infiltration of the bronchial mucosa; (7) death occurred in 4 of the cases whilst the growth was still small (i.e. not more than 1 to 1½ inches in diameter.)

CLINICAL PATHOLOGY AND POST-MORTEM EXAMINATION

Laboratory investigations of silicosis and asbestosis have been few. The following is a brief summary, and the reader is referred to text books on biochemistry for the more detailed account of silica estimations.

Sputum. Patients usually produce very little sputum until the late stages of the disease when the material expectorated is often the product of intercurrent bronchitis and, owing to the excess of mucus, may be of small value. The sputum of asbestosis is more likely to yield helpful information than that of silicosis by reason of the asbestosis body.

Asbestosis. The sputum is generally thick and mucoid — frequently so thick that it resembles semi-coagulated egg albumin. In the latest stages small nummules of pus appear. The asbestosis body may occasionally be found by examination of a direct smear, but as a rule a concentration method is necessary for its detection. The one in general use is that originally devised by Stewart and Haddow³¹ (1929). It is carried out as follows: — About half an ounce of sputum is dissolved in an equal quantity of undiluted formalin, centrifuged, the deposit washed and examined microscopically. This may be done by drying and fixing a direct smear of the deposit (which need not be stained) or by mounting a small

quantity under a cover glass as in examining a urine deposit. Stewart³⁷ (1934) suggests that the occurrence of asbestosis bodies in clusters is indicative of tissue destruction in the lung sufficient to liberate the clusters from the small bronchioles in which they have been formed.

Stewart and Haddow (1929)³¹ have also obtained the bodies by direct lung puncture, whilst the present writer has obtained them in faeces in a case in which it was believed that the sputum was being swallowed.

Silicosis. Here there is no distinctive structure to fall back upon and the detection of particles of free silica by direct illumination and the ordinary microscopic technique is impossible. Burke³⁸ (1935) has described a technique which unfortunately requires a larger amount of sputum for its performance than is available in the great majority of instances. A large quantity of sputum (100 cc or more) is gently heated in a beaker until most of the water is evaporated; 50 cc of aqua regia is then added and the mixture left at room temperature for twenty-four hours with occasional shaking. It is then centrifuged at high speed for one hour, the deposit smeared on chemically clean slides and incinerated to a white or pale grey ash. This deposit is examined by polarised light for doubly refractile particles which are generally angular or needle-shaped and vary in size from 1 to 5 microns. These particles can be further identified as quartz by mounting in immersion fluids of known refractive indices.

Blood. Schlomka and Nolte³⁹ (1935) have made blood counts and sedimentation rate estimations on a series of patients with silicosis. Their results indicate that this method of approach may be helpful in marking the onset of tuberculosis. They found that in various types of silicosis accompanied by tuberculosis the blood sedimentation rate was increased, the total leucocyte count was higher and the propor-

tion of polymorphonuclear cells in the differential count was increased, when compared with the figures obtained in simple silicosis.

King and Stantial ⁴⁰ (1933) record methods of estimating the silica in the blood by a micro-determination technique of analysis developed from those used by chemists in estimating small quantities of silicic acid in various natural waters. Briefly this consists in the reduction of a yellow silicomolybdic acid complex to give a blue colour with aminonaphthol-sulphonic acid, a blue colour being more accurately matched with a colorimeter than a yellow. The methods are too long and complicated for abstraction here and the original papers should be consulted. Both soluble and total silica are examined, the former with 2 cc and the latter with 5 cc blood. The authors state that there appears to be an absence of any insoluble particulate silica and of any organic compound of silicon in the blood so that the figures for soluble and total silica are approximately equal.

Urine. King and Stantial ⁴⁰ have also applied the above methods to the estimation of silica in small quantities of urine, viz., 5 cc.

Silica is a natural constituent of animal tissues and the deductions which can safely be made in cases of silicosis call for a good deal of experience. King and Dolan ⁴¹ (1934) found that silica which was absorbed from the lung or other parts of the body into the blood stream was rapidly excreted in the urine. The elimination of silica in the sputum was higher than in persons having no silica exposure. Attempts made to influence the absorption of silica from the lungs by the administration of alkali were inconclusive. Comparing the blood silica content of non-silicotic and silicotic persons (all of whom had tuberculosis) King and Dolan were unable to detect any significant differences. The results obtained by these workers accord with those obtained with numerous

other biochemical substances in disease. There appears to be a 'tissue threshold' which enables the body to maintain the constituents of the blood on a fairly even keel until the last stages of disease. Biochemical investigations, therefore, in the present stage of our knowledge do not help the clinician very greatly in assessing the functional capacity of the silicotic or asbestotic lung, though they are invaluable in all research work on the subject.

Autopsies. The following details in the making of post-mortem examinations in these cases have, in the writer's practice, been found worth attention.

The lungs should be opened in such a manner that they can be compared with the roentgenogram and the chest diagram of physical signs made by the physician during life. It makes things unnecessarily difficult for those who have to assess compensation claims to find that the lungs have been cut to ribbons in all directions. The writer's practice is to place the lungs, with trachea and bronchi attached, on the operating table with their posterior surfaces uppermost and then to make serial horizontal slices, which can be replaced, from the mid-line of the mediastinum outwards to the pleura. If this operation can be postponed until the lungs have been hardened in formalin, so much the better.

The natural tendency of the pathologist is to reserve the most diseased portion of the lung for histological examination. Whilst this is in general sound practice, the portion thus selected is often so altered by fibrosis or tuberculosis that satisfactory study is difficult. The portion of lung which, naked eye, looks least affected by the disease is often the best for microscopy. Here the silicotic nodules may be found lying in the midst of relatively healthy tissue and can be examined readily. In cases of doubt a portion of interbronchial lymph gland should also be selected: if there are silicotic nodules anywhere in the specimen they are most likely to be found

here. A minimum of three sections should therefore be chosen, (1) the most typical portion of advanced disease; (2) the healthiest looking piece of lung, and (3) an inter-bronchial lymph gland. If possible (1) and (2) should include pleura, whilst portions of lung representing intermediate stages are also selected as required in difficult cases. The writer uses a chest diagram similar to those employed by physicians and marks thereon the position of all portions of lung selected for histological study.

Histology. In silicosis where the demonstration of the whorled nodule is all important, a good stain for the collagenous fibre is essential. No doubt pathologists will differ on this point, but the writer has a strong preference for Van Gieson's stain. Reliance on a simple routine haematoxylin stain is liable to lead one astray, especially in tuberculo-silicosis. The only safe rule is to stain three sections from each block of tissue with (1) haematoxylin and a good counter stain for cellular structure, (2) a collagenous fibre stain such as Van Gieson or Mallory and (3) Ziehl Neelson for tubercle bacilli.

These rules apply with less force to asbestosis. A warning may be given, however, with regard to the resistance properties of the asbestosis body.

In studying the microscopical picture of the different organs one error must be guarded against, viz., the assumption that the asbestosis body has been actually formed at the site in which it is found. Although these bodies probably do not easily migrate far, if at all, during life from the position in which they are formed, they are easily moved in dead tissues by various manipulations. Moreover, they are highly resistant to cleansing, and glassware should be carefully cleansed by concentrated sulphuric acid. In forming an opinion as to whether an asbestosis body has been actually formed where it is found lying in the tissues the following points are help-

ful: (a) the presence of amorphous brown pigment, phagocytes and fibrous tissue around: (b) its relation to surrounding structures (bronchiole, capillary, etc.) The cardinal point in the histology is the liability of the asbestos fibre to be held up at the distal end of the respiratory bronchiole.

Silica particles cannot be detected satisfactorily in ordinary sections mounted in Canada balsam and viewed with transmitted light. With a mounting fluid of high specific gravity such as bromobenzene, particles are more readily detected, but it is only by the use of crossed nicols in a petrological microscope that silica particles can be demonstrated with any certainty. Viewed in this way the quartz particles stand out clearly on a dark background. For the actual technique of the petrological microscope the reader is referred to special mineralogical textbooks.

Irwin⁴² (1934) has devised a simple method of micro-incineration which enables the observer to detect silica particles *in situ* in silica nodules in sections. After removal of the paraffin from the section (which should not be more than 5 microns thick) in the usual way, the slide is heated to 550–600 C. After cooling the incinerated section is mounted in concentrated hydrochloric acid covered with a coverslip and examined by direct illumination for quartz particles. A small furnace is needed for the incineration. The microscope slides vary a good deal in their resistance to the necessary heat and in the writer's experience there are a good many casualties in the way of cracked slides. The ash pattern obtained with silicotic nodules by this method is characteristic. A somewhat similar technique has been used by Schutz and Braun (quoted by Siegmund and Köppenhofer 1935).

Dark ground illumination also has its uses. It may be employed with advantage to detect the characteristic asbestos fibres in wet or dried fibres of sputum and in sections. Siegmund and Köppenhofer have also used it in silicosis (quoting

it as the method of Timms) but the present writer has found so many pitfalls that he has discontinued it.

Extraction of Asbestos and Silica Particles from Lung. The extraction of asbestos fibres and asbestosis bodies from the cut surface of the lung is usually a simple matter. A direct smear on to a slide or a film from the centrifuged deposit of a little tissue fluid which has been expressed from the organ is usually sufficient. In the old stone-mason's lung, also, quartz particles can sometimes be obtained readily by removing a sub-pleural nodule with the point of a scalpel and compressing it between two slides.

But in most instances silica particles are much more difficult to isolate. The best technique is that devised by Jones³ (1933) for the isolation of sericite fibres and termed by him 'sliming.' Portions of the lung freed from preservative by preliminary washing are treated by gradual disintegration with strong nitric acid over a period of several days. The slime thus obtained is diluted about ten times with hot water and the mixture filtered through No. 54 Whatman's filter paper (this is important, since from the ash of many filter papers minute fibres of cellulose which may cause confusion under the petrological microscope are detached). After repeated washings the residue is carefully dried (the fumes given off are inflammable) and the material ignited in the usual way. The light ash is removed with a mixture of bromoform and benzene in a Sollas separator and the minerals which sink to the bottom are washed with benzene, then alcohol, dried, and mounted on slides in Canada balsam. A petrological microscope is necessary for the identification of particles.

Chemical Analysis of the Lung. This is done by chemical assay of the wet or dry ash of the lung. In both cases it is necessary to remove the organic matter first. Jones recommends the method of wet ashing (as described above for the examination of silica particles) on the ground that a dry ash

may contain a high percentage of constituents such as phosphoric oxide, sodium oxide, etc., which are removed by his wet ash technique. The majority of analyses have, however, been carried out by dry ashing. This consists in mincing up large slices of lung, drying at 100° until friable, powdering in a coffee mill, mixing and drying again at 110°, ashing a small portion (e.g. 10 gms) in crucible, weighing ash and assaying it (e.g. by gravimetric method of fusion with alkaline carbonates, precipitation of silica by repeated treatment with hydrochloric acid, ashing the precipitate and determining the true silica content of this ash by treatment with hydrofluoric acid) (Kettle and Archer¹⁷ [1933]).

King and Stantial⁴⁰ (1933) have devised a micro-determination technique by means of which small quantities of tissue can be analysed (about 1 gram fresh tissue or 0.25 grams pulverised dried tissue) as in the case of blood and urine mentioned above. The advantages of using such a method are obvious in that they afford an opportunity of comparing areas of diseased lung by chemical and histological methods which the ashing of a large slice of lung does not.

No hard and fast line can be drawn between the percentage of silica in the dried lung in silicotic and non-silicotic persons, but probably it would be safe to put the upper limit for the non-silicotic lung at about 0.2 per cent. Silicotic lungs often yield analyses of 1 to 2 per cent.

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V. EXPERIMENTAL PATHOLOGY

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EXPERIMENTAL PNEUMOCONIOSES

It was once maintained that silicosis, the best known form of pneumoconiosis, could not be studied by experimental methods for it was a chronic disease which took years to evolve and was commonly complicated by the most chronic manifestations of an infection like tuberculosis. However, experience has demonstrated the fallacy of this contention. With pure silica it has been possible not only to reproduce tissue changes that simulate the lesions of human silicosis in several short-lived species but to demonstrate that such changes alter native susceptibility to tuberculous infection. It has been necessary to curtail the essential factor of time but this has been largely compensated by exaggerating the reciprocal factor of dosage. While this necessary adjustment may have produced some distortion of the resultant lesions so that they are not always identical with those in human silicosis, the same kinds of cells participate in the reaction and the type of tissue change is the same. (Figure 1, *a* & *b*) All of these effects are specific and cannot be produced with non-siliceous materials. On the other hand, the mixed dusts, composed of silica and other minerals, which apparently cause silicosis in different industries have not uniformly excited comparable reactions in animals. In searching for an explanation for his apparent failures, the experimenter is beginning to discover principles which may be much more important than his successful reproduction of silicosis with pure silica. It would now appear that the non-siliceous components of a mixed dust are not merely inert diluents of the silica but



FIGURE 1

a. Vertical section lungs of white rat. Inhaled pure chalcedony 9 months, then normal atmosphere 5 months. *b.* Detail of above. Note marked swelling of collagen.

that they exert specific effects upon the silica either in the atmosphere or after it has been inhaled. Evidence is accumulating from various sources to indicate that some types of mineral particles may accelerate the action of silica on the tissues; others may retard it or perhaps prevent its inhalation from the atmosphere. The conditions governing such modifications have not been defined but the experimental methods now developed offer a means of attack. Certainly a silicosis that develops with unusual rapidity should be capable of experimental reproduction. The limitations imposed by the span of life in small animals may preclude a complete evaluation of inhibitory effects. Nevertheless experiments should point out principles which govern reaction to various combinations of dusts and they may disclose the manner in which silica injures the tissues.

Other forms of pneumoconiosis have not been studied so thoroughly. The lack of significant degrees of reaction to non-siliceous dusts has been sufficiently well established to assert with a reasonable degree of certainty that the pneumoconioses of this class are essentially pigmentations with little or no effect upon respiratory function. Finally, there is the group of conditions caused by silicates, which Badham designated as 'silicatosis.' Only one of these, asbestosis, which may be produced by several of the fibrous silicates, is generally recognized as a disease. Its early manifestations have been reproduced in animals but the evolution of its more advanced phases still awaits demonstration. The evidence of other forms of silicatosis is largely roentgenographic. Experimental studies with this group of minerals have thus far yielded nothing to support a belief in their capacity to provoke progressive fibrosis.

The basic facts that have already been discovered in an intensive study of man in various dusty environments and the reliable methods of procedure now in possession of the

experimenter offer hope that in time he may be able to answer the perplexing questions that still enshroud the reaction to all kinds of inhaled dusts.

With these general considerations in mind the achievements of the experimental pathologist can now be examined. Much of his work remains to be done; some of it will have to be repeated because of the discovery of certain pitfalls of which he was not aware in his earlier efforts. But many of his observations are undoubtedly sound and can be used as the basis for further investigation.

EXPERIMENTAL METHODS

The experimentalist has a variety of methods at his command. Obviously the most satisfactory way to study the effect of *inhaled dust* is to make animals inhale it. This procedure is expensive, time-consuming and requires very special equipment but it constitutes the only means by which the effect upon the lungs of atmospheric suspensions of dusts or mixtures of dusts can be determined. It permits investigation of the physical properties of such dusts as they exist in the atmosphere. It allows the protective mechanisms of the upper respiratory tract to come into play and to admit or exclude the different components of a dust from the lungs. It permits the foreign particles to accumulate slowly and without gross trauma to the tissues and it does not interfere with the natural mechanisms of elimination. In fact the inhalation method is the only one to demonstrate decisively whether a given kind of dust suspended in the atmosphere will injure the lungs.

All other methods merely indicate whether particles of a certain composition are capable of producing reaction in the tissues. They are most useful in eliminating the substances which are not irritating and which consequently do not need further investigation. Based upon the *injection* of aqueous

or saline suspensions of particles, they have the advantage of being easy to perform, of requiring no special apparatus and of producing results in relatively short periods of time. All of them are subject to the one fundamental objection that they test the action of an aqueous rather than an air-borne suspension of particles and it is now known that the properties of such suspensions are not necessarily identical. With proper precautions this objection may not be particularly serious as long as pure substances are being tested. In dealing with mixtures of harmful and benign minerals exclusive reliance upon injection techniques may lead to false conclusions. Many of the proposed procedures are also unsatisfactory because such overwhelming doses of the substance under investigation are injected that confusing non-specific inflammatory reactions are produced. This is particularly true when the particles are introduced directly into the organ where their effects are to be observed. In most instances the test organ is not the lung but this is probably of no moment for experience has demonstrated that free silica and all non-siliceous minerals *in particulate form* have the same effect upon connective tissues regardless of the organ in which they are located. Apparent exceptions are the *fibrous* silicates known collectively as asbestos, which seem to exert their specific action only in the lungs. In spite of these objections, injection methods have their place and the ones which are technically satisfactory have contributed materially to the solution of the pneumoconiosis problem.

One injection test, (Gardner and Cummings' 1933) ¹ in the writer's opinion obviates the non-specific effects that have been mentioned and results in localization of particles by physiological mechanisms. It is based upon repeated intravenous injections of small fractional doses of particles suspended in physiologic salt solution. Full grown rabbits are injected by ear vein with one gram of particles, 1 to 3
