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## MEDICAL-LEGAL ASPECTS OF PULMONARY DISEASE

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### INTRODUCTION

**T**HIS ARTICLE on the medico-legal aspects of pulmonary disease is written by physicians for lawyers. It is hoped that the medical facts and concepts presented will assist interested members of the legal profession in finding areas of further legal import. The few diseases which have been chosen are frequently involved in workmen's compensation proceedings and lend themselves more to legal evaluation than many other diseases, although it is appreciated that those diseases not discussed have found and may again find their day in court. Beyond the scope of this article are: the role of individual susceptibility, the responsibility of the individual, and the responsibility of communities where disease is more prevalent, all of which demand further analysis and evaluation.

### PNEUMOCONIOSIS

Pneumoconiosis is a disease of the lungs caused by the inhalation of noxious dusts. It develops because of occupational exposure to offending agents. The employer legally has certain obligations. In some states, departments of industrial hygiene attempt to keep the hazard at a minimum. Occasionally, a union group will exert pressure on an employer or an industry to correct certain conditions which contribute to

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the hazard. If the employee becomes disabled because of pneumoconiosis, state workmen's compensation laws will generally compensate him for his disability, but, in most states, the condition must be disabling before compensation is awarded.

Most dusts are incapable of producing significant diseases. Only a few dusts produce sufficient tissue reaction in the lungs to impair pulmonary function. A knowledge of the occupations in which causative agents are encountered is essential in order to evaluate causally related disability. The materials encountered, the working conditions, the methods used, the protective measures employed and the hygiene practiced must be ascertained and evaluated. Different dusts are capable of producing varying degrees of hazard. Thus, the pneumoconiosis may be divided into major pneumoconioses and minor pneumoconioses. The major pneumoconioses are silicosis, abestosis, talcosis and coal miner's pneumoconiosis. Among the minor pneumoconioses are those caused by anthracite, diatomaceous earth, silicates (mica, clays, feldspar), and the vegetable dust pneumoconioses such as mill fever, byssinosis, bagassosis, farmer's lung, weaver's cough and others. In addition, there is a group of benign pneumoconioses such as baritosis, siderosis, stanosis, graphosis, and exposure to limestone, marble, cement, and other dusts. Whether or not impaired pulmonary function ultimately results depends on the development of fibrosis in the lungs or the magnitude of the damage to the bronchioles, air sacs, and pulmonary blood vessels.

#### SILICOSIS

Whether or not an individual develops disabling silicosis depends on a number of factors:

1. *The substances with which the employees came into contact while at work.* Quartz, quartzite, sandstone, flint, tripoli, diatomaceous earth and silica sand contain high contents of silicon dioxide which is the causative agent in silicosis. Thus, quartz is almost pure silicon dioxide. Granite contains sixty per cent pure silicon dioxide. Silica sand has a very high content of silicon dioxide. Mining, tunneling, quarrying, highway construction, foundry work, sand blasting, and the making of pottery are some of the industries in which high quantities of silicon dioxide are encountered.

2. *Dust particle size.* Inhaled particles over three micra in diameter do not produce typical silicotic nodules in the lung. Particles which range from one to three micra in diameter generally produce nodular

silicosis if inhaled in sufficient quantities for long enough periods of time. Particles less than 0.5 micra produce a diffuse fibrosis with some nodulation in the lungs. The potency of the particles of silica increase remarkably with the decrease in size.

3. *Duration of exposure.* Usually, in most industries, from five to thirty years of exposure are required to produce disabling silicosis. However, exposure to dusts of extremely high silicon dioxide content of minute particle size (1 to 3 micra) and in high particle concentration may produce silicosis after only one and one-half or two years of exposure. Such cases commonly occur among workers engaged in the manufacture of scouring powders where special types of extremely fine sands in heavy concentrations are used.

4. *Conditions under which the employee worked.* In the mining and tunneling industries it is important to know how far underground the work was being done, how confined the space was in which the employee worked, whether wet or dry drilling was employed, whether a ventilator was present, and what devices, types of respirators, or masks were used. Wet drilling greatly reduces the amount of dust in the atmosphere.

5. *Concentration of dust particles.* This can be ascertained by industrial hygienists or sanitary engineers. Less than 5,000,000 particles per cubic foot of air of any dust will not produce disabling pneumoconiosis even if inhaled for long periods of time.

Thus it can be seen that the length of time for silicosis to develop will depend on the factors enumerated above. Inhalation of high concentrations of dusts of extremely small particles with high silicon dioxide content where no protective devices were used will produce silicosis in a much shorter period than under circumstances where the particles were much larger, contained less silicon dioxide, were not present in heavy concentrations, and where the employees wore adequate protective devices. In addition, there is an individual susceptibility which renders some employees more prone to develop silicosis than others working in the same environment.

The major symptom of silicosis is shortness of breath which begins insidiously and progresses relentlessly to the point where the individual becomes disabled. The disease may then be complicated by tuberculosis, emphysema, or pulmonary heart disease.

It remains the expert physician's task to determine not only whether the individual has an industrial disease, but also whether it is disabling.

An individual with X-ray findings of simple silicosis may not be disabled.

#### ASBESTOSIS

Asbestosis is caused by a hydrated magnesium silicate. Asbestos fibers from twenty to twenty-five microns in length are the most active in the production of a disease. Asbestos fibers are used as insulation. Exposure exists in those working with materials used for insulation, as in pipefitters, pipecoverers, and shipfitters. It also occurs in the manufacture of clutch facings, brake linings and undercoating.

As in the case of silicosis, the occupational history must reveal the nature of the dust involved, the circumstances of exposure, general ventilation in the areas where the work was being done, local exhaust ventilation and personal protection (masks or respirators). A detailed history of every job held should be taken. The types of material handled, operations involved, dust present, and duration of exposure should be ascertained. Asbestosis develops after a shorter period of exposure than silicosis. The length of exposure often is between five and ten years. The degree of disability is determined by medical evaluation which may include studies of pulmonary functions.

It is extremely important to know that there is an increasing incidence of cancer of the lung and malignant tumors of the pleura in individuals who have asbestosis when compared with the incidence of such tumors in the general population. This relationship has important medico-legal implications in determining causal relationships in individuals with asbestosis who develop these extremely serious diseases.

#### BERYLLIUM DISEASE

The inhalation of beryllium dusts produces systemic disease. Beryllium is used extensively in the atomic energy program because of its effectiveness as a moderator and reflector of neutrons. It was used in the manufacture of fluorescent light, but is no longer used in this industry. Beryllium forms an alloy with many metals such as copper, nickel and aluminum. The greatest utilization of beryllium is as a beryllium-copper alloy.

Beryllium disease may be either acute or chronic. The acute form of the disease may be either dermal or respiratory. In the dermal type, the exposed parts of the body such as the face, neck, arms and hands

present patches of skin eruptions. The skin often is swollen, itchy and has a burning sensation. There often is inflammation of the membranes of the eyes, nose and throat. Abrasion of the skin with implantation of one of the beryllium salts produces a nodule which persists until surgically removed.

Respiratory aspects vary from a mild rhinitis or tracheobronchitis to fulminating pneumonitis. The worker complains of a cough which may become productive of blood-streaked sputum, fatigue, anorexia, substernal discomfort and palpitation. At the onset of symptoms, X-rays disclose no specific changes. Within two to three weeks after onset of symptoms, X-rays usually reveal a ground glass haziness followed by areas of infiltration and occasionally by discrete nodules. These findings will persist for a number of weeks to several months, but often disappear before symptoms subside.

In the chronic form of beryllium disease, the striking feature is the delay between the last exposure and the onset of symptoms. The interval may vary from one month to more than ten years. The inhalation of irritant fumes has been cited as a stress factor initiating the onset of the symptoms of the disease. A correlation between the intensity and duration of exposure and the severity of the disease has not been demonstrated. This disease has occurred in persons residing in the neighborhood of a beryllium plant without any established work exposure. The onset of the disease is insidious. Often the worker complains of frequent colds that persist, slight coughs, loss of appetite, weight loss and fatigue. This is followed by the appearance of a progressive condition of shortness of breath, which increasingly limits the worker's activity. Often failure of the right ventricle of the heart (cor pulmonale) is responsible for death.

The presence of beryllium in biopsied lung tissue does not necessarily mean that the individual has had an occupational exposure to beryllium. Beryllium may be found in the soil in certain areas, in the atmosphere and in certain types of coal. The findings of beryllium in the urine likewise does not justify the diagnosis of beryllium disease. It merely means an individual has been exposed to beryllium. The patch test (1% solution of beryllium salt as a patch) is controversial. Some believe it is useful as a diagnostic tool and as a screening test. Others believe that by using the patch tests, normal persons might become sensitive to beryllium and thus made more susceptible to the disease.

The course of beryllium disease is so variable that it is not possible to predict the degree of disability to be expected nor the outcome of the disease.

#### PULMONARY TUBERCULOSIS

Pulmonary tuberculosis is an infectious disease caused by the mycobacterium tuberculosis. The tubercle bacillus enters the susceptible human host by inhalation of particles excreted from an infected individual. Previously negative, the tuberculin skin test then becomes positive a few weeks after this first infection. This primary phase becomes inactive in most people, but the body may continue to harbor live bacilli through a latent phase.

Secondary causes of tuberculosis are largely socio-economic. The primary phase occurs in an environment in which tubercle bacilli are present in a form that may be inhaled readily. A patient with open "tuberculosis" may spread droplets of bacillus laden sputum by cough and thus contaminate the atmosphere. This circumstance is prevalent in crowded living or working quarters (such as the crowded slums and poorly ventilated sweat shops of a previous era), military barracks, sailors' quarters, public facilities, and hospitals. Most pulmonary tuberculosis in the past has developed in the younger age groups, children and teenagers. Today however, the average age of the development of a positive tuberculin test is much later.

The reinfection phase of tuberculosis occurs in a tuberculin positive individual and is currently considered to be endogenous (*i.e.*, a focus in the body that has harbored latent or inactive tubercle bacilli acquired during the primary stage becomes activated, and clinically active tuberculosis develops). The circumstances in which the reinfection phase of pulmonary tuberculosis occurs are less well known. Activation or reactivation of a latent focus occurs more frequently with alcoholism, diabetes, gastrectomy, long hours of hard work, stress, dissipation and malnutrition.

The steps taken to prevent disease must interrupt the transmission of the tubercle bacilli from the patient to the person with a negative tuberculin test. The greatest effectiveness in prevention must be achieved before the primary phase. Many industrial units have incorporated this principle into environmental control to protect the workers from disease, and the company from negligence suits or claims

under workmen's compensation laws. Adequate working space, ventilation, and, where necessary, germicidal ultraviolet lamps are provided. Tuberculin testing and chest X-rays have become part of the required pre-employment physical examinations for many employers.

Proof of contraction of tuberculosis in work areas requires the proof of exposure to an individual with active tuberculosis.

When active clinical disease has developed, the determination of the duration and degree of disability, and the prognosis for return to work is a matter of individual assessment, although general principles may be described.

During the acute stage of pulmonary tuberculosis, the patient may not be able to work because of disabling symptoms. Hospitalization may be required because of fever, weight loss, distressing cough, blood spitting, shortness of breath, and other related symptoms. When antibiotic treatment has become effective, the patient may be able to return to work before complete healing has occurred if the symptoms have improved. There is little risk to the patient and others on the job with him if the cough and sputum production have been controlled by specific medicine, even though the sputum may still contain tubercle bacilli during the early phase of healing. The resultant improvement in patient morale, the decrease in cost of hospitalization, and the reduction of wage loss is thus greatly enhanced.

When pulmonary tuberculosis is in its active phase, is undiagnosed, and is unaffected by treatment, the threat of infection of the unsuspecting tuberculin negative bystander is great. The early detection of disease by physicians in industrial medical departments, by public health surveys, and in private practice with the use of tuberculin tests and periodic X-rays has resulted in a decrease in the number of people who are sources of infection and in the number of new cases each year. Continued vigilance is still necessary.