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INDUSTRIAL DISEASES OF THE CHEST*

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A DISCUSSION of industrial chest conditions would be incomplete without a review of silicosis, but that familiar occupational entity is now so well-known that it seems preferable to consider some of the unrelated ailments which simulate it rather than the various phases of the disease itself. The distinction is important because of economic, medicolegal and employee health implications, and the far-reaching effects of inaccurate diagnosis on both management and labor demand recognition and correction.

Unfamiliarity on the part of some physicians concerning more recent developments in the field of industrial chest diseases is regrettable, but insistence on making dogmatic diagnoses in spite of the lack of accurate information is inexcusable. It is most difficult to overcome the effect on a worker when once he has been told by his family physician that shadows observed in his chest x-ray are the result of silicosis when actually there has been no significant exposure. Such interpretations frequently result in unjustified claims for compensation as

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well as unnecessary apprehension on the part of the employee.

Chest conditions whose x-ray patterns simulate silicosis may be classified as industrial or nonindustrial. Among the causative agents responsible for lung affections in the industrial group are dusts from such organic materials as cotton, sugar cane and tobacco. Cases of byssinosis have been reported in this country but the disease seems to be less prevalent than in England, where the Byssinosis Act of 1940 awards compensation for total disability following periods of exposure up to 20 years.

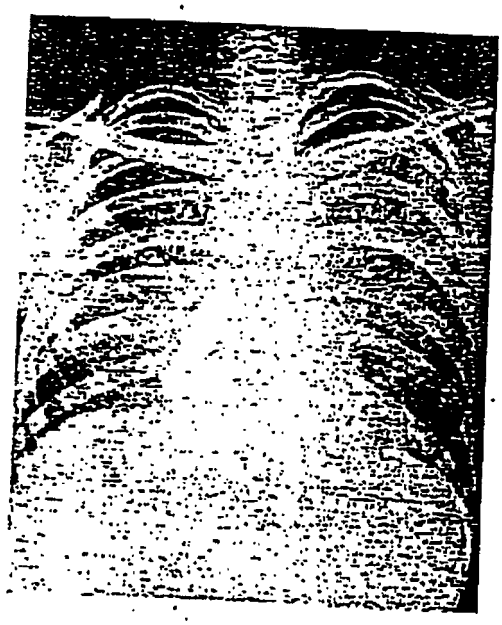
Ritter and Nussbaum,[1] following a thorough investigation of the cotton industry in Mississippi in 1944, concluded that no evidence was found to justify the existence of byssinosis as a clinical entity among employees in a cross section of the cotton industry in that state. They did find that certain allergic individuals showed asthmatic reactions to high concentrations of cotton dust but no other manifestations of actual disease were evident.

Castelden and Hamilton-Patterson,[2] in the British Medical Journal, in 1942, described symptoms consisting of dyspnea, cyanosis, violent cough, scanty sputum, and mild elevation of temperature in workers exposed for one to two months to fibers of bagasse, a waste product of sugar cane after the extraction of sugar. The condition was also described by Gillison and Taylor,[3] who stated that the

1. Ritter, W. L., and Nussbaum, M. A.: Occupational Illnesses in Cotton Industries II. J. Indust. Hyg. & Tox., 27:2, 47, February, 1945.
2. Castelden, L. L. M., and Hamilton, J. L.: Patterson Bagassosis: An Industrial Lung Disease. Brit. Med. J., 2:473, October 24, 1942.
3. Gillison, J. A., and Taylor, F.: Bagassosis: Further Notes on Four Cases. Brit. Med. J., 2:577, November 14, 1942.

x-ray appearance suggested a bilateral pneumonia of the influenzal type which resulted in varying degrees of fibrosis or else cleared up completely. The reaction in the lungs to this dust was considered a possible allergic manifestation. Observation of 4,000 workers in the tobacco industry over a period

FIGURE 1



Bagassosis
Courtesy of Dr. J. Hannon

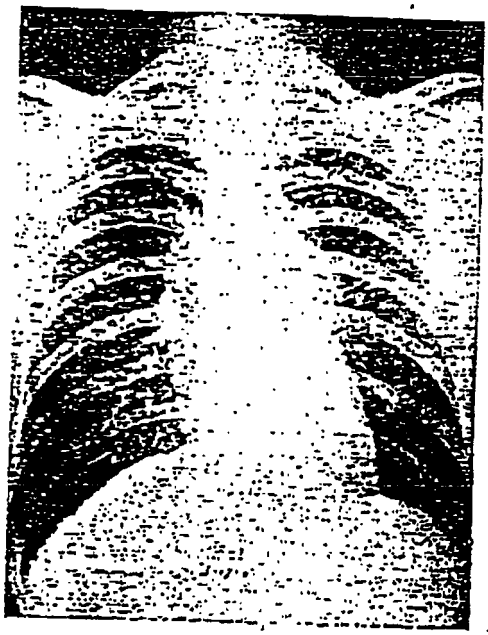
of 10 years convinced C. F. Long[4] that "Tabacosis does not develop in the lungs of those who inhale tobacco dust." His study covered an investigation of 1,247 employees with known tobacco dust exposure as compared to 1,007 with no exposure.

Dusts such as those produced from limestone, chalk, marble, carbon, and cement may produce accentuated linear markings in the x-ray but they are relatively inert. They do not cause definite symptoms and frequently are only discovered in routine chest x-ray surveys. Barium, iron and tin may produce radiopaque deposits in the perivascular lymphatics and interalveolar septa which closely simulate silicosis in its discrete nodular form but these residues do not appear to have real significance except from the medicolegal aspect. A possible exception may be found in Stannosis or changes in the lungs resulting from exposure to tin and its compounds. Observations by some workers in this field in recent years have aroused suspicion that such deposits may not be entirely innocent. However, thus far nothing definite in the way of toxic or pathologic manifestations has been established.

The nodulations in arc welders, described by Sander and Enzer[5] in 1938, demonstrated that an x-ray pattern almost indistinguishable from silicosis can result from the inhalation of iron oxide fumes. Exposure to free silica in these cases was lacking and postmortem findings on one case showed no evidence of fibrosis. Histologically there were simple deposits of pigment about the

4. Long, C. F.: Tobacco Dust and the Human Lung. Industrial Medicine, 8:9, September, 1939.
5. Sander and Enzer: Chronic Lung Changes in Electric Arc Welders. J. Indust. Hyg. & Tox., 20:5, May, 1938.

FIGURE 11



Silicosis

blood vessels which gave a typical Prussian-blue reaction for iron. The striking similarity between these cases and a number in our own experience among foundry grinders and burners led to the inauguration of an experimental study on the effect of air-borne grinding dusts on laboratory animals. The work was begun approximately a year and a half ago under the direction of the

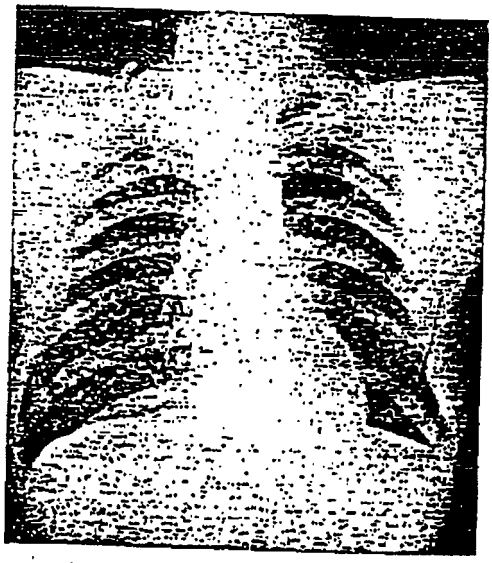
late Dr. L. U. Gardner at the Saranac Laboratory, Saranac Lake, New York. Analysis of representative samples of the dust in this industry indicated that while free silica was not present in sufficient amounts to produce silicosis, enough iron oxide in respirable sized form did exist to account for the roentgenographic changes observed. Preliminary findings in the lungs of animals after eight to ten months' exposure to this dust show faint diffuse dust pigmentation grossly in guinea pigs and white rats, but microscopically only scattered inactive dust cells without evidence of actual fibrosis. Chest x-rays of rabbits exposed to inhalation of the same dust for ten months were essentially negative and the dust had no apparent effect on accelerating tuberculous infection in those animals in which the disease had been introduced.

The occupational fibrosis which results from the inhalation of asbestos fibers, particularly those over 2 microns in length, is believed to be caused by a mechanical plugging of the alveoli or the formation of fibrotic sleeves along the terminal bronchioles. The condition is observed most frequently in such operations as mining and crushing crude asbestos. In 1940 Gardner[6] stated that at that time 27 percent of exposed workers were involved and in those with more than 15 years experience the percentage rose to 60. The characteristic x-ray finding is the so-called "ground glass" appearance which differs considerably from the nodular pattern of silicosis.

Within the last few years lung changes have

6. Gardner, L. U.: The Pathology and Roentgenographic Manifestations of Pneumoconiosis. J.A.M.A., 114: February, 1940.

FIGURE III



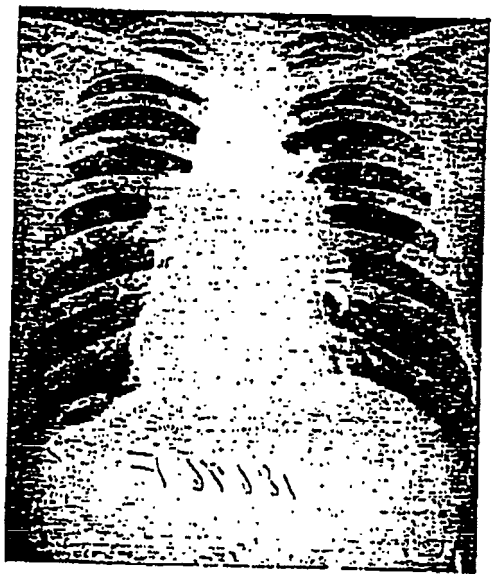
Siderosis

been observed in some workers in the beryllium industry. X-ray findings range from diffuse haziness in both lung fields to discrete large or small nodules which may disappear after one to four months. While the action of beryllium is thought to be a toxic one, affecting the skin, nasal mucous membranes and pulmonary tissues, its exact effect has not been definitely established. Van Ordstrand[7]

7. Van Ordstrand et al.: Beryllium Poisoning. J.A.M.A. 129:15, December 15, 1945.

and his associates have reported beryllium poisoning in 170 workers in an industry in which the metal was used in the manufacture of precision instruments during the war. Chemical pneumonitis developed in 38 employees in this group and progressed without exacerbation or remission either to complete recovery or to death. Postmortem examination on 5 cases showed typical pneumonitis without definite fibrosis. In 1946 Hardy and Taber-

FIGURE IV



Sarcoidosis
Courtesy of Dr. P. Hamberger

shaws[8] reported 17 cases of delayed chemical pneumonitis occurring in a fluorescent lamp manufacturing concern where beryllium compounds were used. Six deaths occurred in this group and recovery was very slow in the remaining individuals. A few are still disabled. The distinctive feature of the disease was the delay in onset following exposure, and progression of the illness in spite of removal from the hazard.

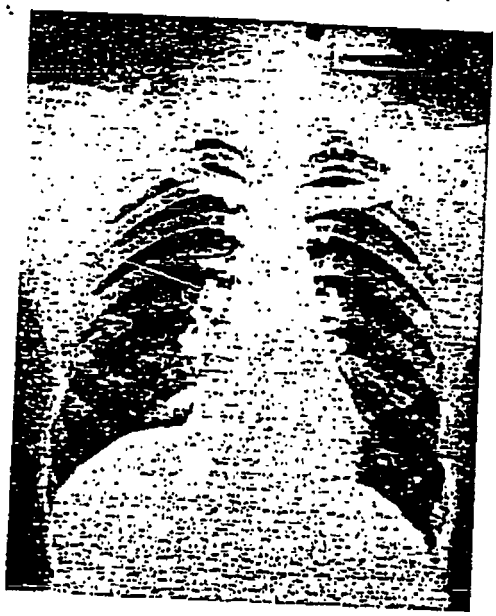
Mycotic diseases such as aspergillosis, monilliasis, bronchial asthma due to wood dust spores, coccidioidomycosis, blastomycosis and actinomycosis while attacking the mucous, cutaneous, subcutaneous, osseous and visceral structures, may produce x-ray patterns in the lungs which can be very difficult to distinguish from nodular silicosis. The roentgenographic changes vary from accentuated linear markings and miliary sized densities which may coalesce, to discrete nodular opacities scattered throughout the parenchyma of the lungs. Clinical evaluation correlated with x-ray findings and the history should facilitate the diagnosis in individual cases. These diseases are usually not considered industrial. They are endemic in certain regions but in spite of the lack of occupational prevalence, cases of mycotic infection do occur in certain industries. Such an instance was reported by Coe[9] in September 1945, where a worker in the Chicago stockyards, who was exposed to dust which was mixed with hay, corn and straw, developed aspergillosis and received an award for compen-

8. Hardy, H. L., and Tabershaw, I. R.: Delayed Chemical Pneumonitis Occurring in Workers Exposed to Beryllium Compounds. J. Indust. Hyg. & Tox., 28:5, September, 1946.
9. Coe, G. C.: Ann. Int. Med., 23, 423-425, September, 1945.

sation from the Industrial Commission of Illinois.

Infection with Coccidioides immitis produces coccidioidomycosis, a disease endemic in Central

FIGURE V



Miliary Calcification

California, Arizona, and Texas. It may be encountered occasionally in any region and should not be confused with coccidiosis, which refers to an infection by one of the animal parasites included under the order of coccidia of the class Sporozoa. The

symptoms resemble those of influenza or bronchopneumonia and may imitate tuberculosis. X-ray findings include discrete nodular shadows or confluent areas of pneumonitis, depending on the stage of the lesion. Densities appear in the bases of the lungs or radiate from the hilus into the upper lobes. Cavitation may occur which may be most difficult to differentiate from tuberculosis. In making a diagnosis it is essential to consider not only the clinical history, exposure and x-ray findings, but also the cutaneous reaction to coccidioidin and the identification of Coccidioides immitis spherules in the sputum.

Other conditions whose x-ray appearances may simulate silicosis are miliary tuberculosis, metastatic carcinoma of the lungs, vascular changes associated with age and hypertension, mitral stenosis, miliary calcification, bronchiolitis, polycythemia vera and Boeck's sarcoid. Here again careful consideration of occupational exposure and evaluation of the physical examination should eliminate the possibility of error in arriving at a satisfactory diagnosis.

The occurrence of a number of cases of sarcoidosis in certain industries using beryllium and its compounds has given rise to speculation as to whether this condition should be classified as an occupational disease. Sarcoidosis was first described in 1875, at which time it was thought to be a skin condition. Later it was found that the process involved the lungs, bones, lymph nodes and other organs. The etiology is unknown but histologically, according to King,[10] the lesions resemble

10. King, Donald S.: Sarcoid Disease as Revealed in the Chest Roentgenogram. Am. J. Roent. & Rad. Therapy, 45:6, April, 1941.

those of tuberculosis. Giant cells may be seen but caseation does not occur and demonstration of the tubercle bacillus is rare, either by staining methods or animal innoculation. Tuberculin tests are usually negative even with strong dilutions. A study by Longcope and Pierson failed to show the presence of fungi in the lesions of sarcoidosis. In a recent editorial in *The American Review of Tuberculosis*, Pinner[11] states that of all authors who have expressed a positive opinion as to the causative agent in sarcoid lesions, the majority believe that the tubercle bacillus is directly or indirectly involved.

Katz, Cake & Reed[12] note that pulmonary lesions are common in sarcoidosis but x-ray films of the chest are not a guide to the severity of the disease. They have observed that widespread lung involvement may occur where there is little evidence of a generalized process. On the other hand, extensive lesions may be present in other organs when very little can be seen in the chest roentgenogram. A bilateral nodulation resembling miliary tuberculosis is frequently observed in the x-ray while at times a soft infiltration similar to caseous pneumonic tuberculosis, with or without cavitation, is present. Often the only pulmonary manifestation is marked bilateral, symmetrical enlargement of the hilar and peribronchial lymph nodes. The lesions have a tendency to resolve completely, leaving no residual fibrosis. In King's series of cases this occurred in

11. Pinner, Max: Editorial on the Etiology of Sarcoidosis. Am. Review of Tuberculosis, 54:6, December, 1946.

12. Katz, Sol, Cake, C. P., and Reed, H. R.: Sarcoidosis. New England J. Med., 229:493, 503, September 23, 1943.

an average time of twenty-two months. Gardner[13] recently stated: "While this disease occurs here and there throughout the population at large it has frequently been attributed to pulmonary irritants of industrial environments. Recently a considerable number have been found in industries using beryllium and its compounds. It has not occurred in every plant using these materials. It has not been possible to reproduce a sarcoidosis with beryllium compounds in animals." His conclusion was that much remains to be done before it can be stated that sarcoidosis is an industrial disease.

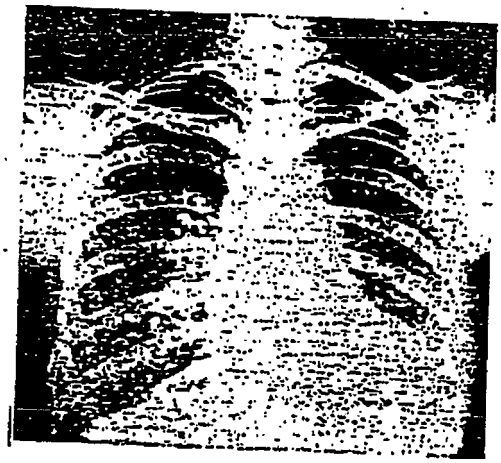
Miliary calcification or "wheatena," as it is sometimes called, is a condition frequently seen in the chest x-rays of people living in certain states such as Missouri, Mississippi, Ohio, and Kansas. The multiple, dense, sharply defined nodular shadows have on occasion been diagnosed as silicosis but their characteristic appearance, particularly when considered in connection with the possibility of occupational exposure, should eliminate such an interpretation. The actual causative agent in the production of these densities has not been definitely established but it is thought to be the result of healed infection of obscure origin. High, Zwerling & Furclow,[14] reporting on 113 cases of miliary calcification, tested 108 with tuberculin and histoplasmin. Of these, 104 cases, or 96.3 percent, reacted to histoplasmin, while only 4 had negative reactions to this antigen. None reacted only to tuberculin. This finding appears to be strong evidence

13. Gardner, L. U.: The Diagnosis of Silicosis. Indust. Hyg. Dig., 3:11, November, 1943.
14. High, R. H., Zwerling, H. B., and Furclow, M. L.: Disseminated Pulmonary Calcification. Public Health Report: Tuberculosis Control Issue, 62:1, January, 1947.

that disseminated calcifications are not caused by tubercle bacilli but probably by the agent producing sensitivity to histoplasmin.

Five cases of rheumatic mitral stenosis, presented by Hurst, Bassin & Levin[15] in 1944, showed linear and nodular patterns which might easily be confused with silicosis. The nodular shadows in the x-ray films were thought to be due to hypertrophied blood vessels seen in horizontal section or "caught on end," vascular engorgement throughout the pulmonary fields or calcium deposits resulting

FIGURE VI



Polycythaemia Vera
Courtesy of Dr. J. Hannon

15. Hurst, A., Bassin, S. and Lewin, L.: Miliary Densities Associated with Mitral Stenosis. Am. Review of Tuberculosis, 49:3, March, 1944.

from healed small infarcts sometimes associated with severe passive congestion. A case of mitral stenosis, confirmed at autopsy, in which the roentgenogram indicated diffuse fibrosis, was presented by D. A. Hanson[16] in the *Lancet* of September, 1945. The patient had worked as a bricklayer for 15 years and a sandblaster for 5 years, but the postmortem examination revealed no macroscopic evidence of silicosis.

CONCLUSIONS

From consideration of the conditions herewith presented it is obvious that the diagnosis of industrial chest conditions is not a simple matter. Too frequently an opinion is based on the x-ray alone without taking into consideration all the factors involved. Unless there has been definite exposure to hazardous dust, as demonstrated by actual counts of respirable sized particles of free silica in the breathing zone of the worker, one should be extremely cautious about making an interpretation of silicosis even though the roentgenogram strongly suggests it.

16. Hanson, D. C.: Mitral Stenosis and Silicosis. Lancet, 12:2, September 29, 1945.