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Etiology and Aggravation in Thoracic Medicine

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ETIOLOGY AND AGGRAVATION IN THORACIC MEDICINE

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INTRODUCTION

IN THE practice of thoracic medicine, misconceptions about the etiology and/or the aggravation of thoracic diseases are commonplace. These errors are frequently perpetuated by patients and occasionally by well-meaning lawyers and well-trained physicians. Superficial analysis indicates that most of these misconceptions arise from the concept of *post hoc ergo propter hoc.* In diseases affecting the thorax, multiple etiological factors commonly operate over prolonged periods of time. Under such circumstances it is extremely difficult to sort out cause and effect. Thus, in light of the imperfections in diagnosis, misconceptions about etiology and aggravation of disease can be appreciated.

There are substantial limitations and uncertainties to any medical diagnosis. The semantic derivation of the word "diagnosis" comes through the Greek words whose meaning is "to know through" or "to understand by means of the manifestations of." A complete diagnosis in the modern sense of the term is a disease entity in which the causative mechanisms are clearly understood, and the manifestations readily appreciated both in clinical signs and symptoms and in laboratory findings. This type of disease entity frequently can be treated satisfactorily. Pulmonary tuberculosis is a useful example since its specific cause is known and it produces characteristic symptoms. The disease may be diagnosed by physical examination, X-ray findings, and recovery of the tubercle bacillus from the patient's sputum or other body secretions. This disease usually can be successfully treated by appropriate chemotherapy.

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The second order of the diagnosis consists of diseases which are readily identified by clinical findings and laboratory results, have a well recognized pathological pattern, but whose etiology is obscure. No specific treatment is available for most of these diseases. An example of a disease with an unknown etiology is pulmonary neoplastic disease. The exact causative mechanism is unknown, although there is considerable medical evidence in favor of certain theories.

The third order of diagnosis, frequently called the "clinical syndrome," consists of a series of symptoms and physical signs often accompanied by certain laboratory findings, but with several different pathological manifestations. Although the pathological findings may clearly separate one of these diseases from another, there is frequently no plausible theory to explain the etiology and little or nothing that can be done for the patient other than symptomatic or general measures. This type of disease is illustrated by the various forms of interstitial fibrosis. Under the microscope a distinct pathological entity may be apparent and specific histological and histochemical details separate some of the diagnostic entities. Nevertheless, the clinical expression of these different types of fibrosis may be similar. The precise pathological entities can rarely be diagnosed from either the X ray or other manifestations in the living patient. Since there is no specific treatment for any of the different types of fibrosis, it is often of mere academic interest to establish a specific pathologic diagnosis.

There is a fourth level of diagnosis which occurs when a person's name is used for the title of a disease or syndrome—"eponym diagnosis." These should be eliminated from current diagnostic nomenclature, and pathological or physiological descriptive terms used instead. Also to be deplored is the introduction of catch phrases with the advertising flair (Black Lung, Pink Puffer, Blue Bloater). These diagnostic labels reflect the imperfection of medical knowledge.

An intelligent assessment of etiological and aggravating factors in diseases of the thorax requires recognition that the disease falls into a category of a complete diagnosis, an incomplete entity, a symptom complex, or an eponym diagnosis.

When legal questions arise concerning thoracic diseases, an expert medical opinion may be sought. Although any licensed physi-

cian may proclaim himself a thoracic disease specialist, most attorneys will seek a properly trained and certified consultant. A specialist in internal medicine who concentrates his efforts on diseases involving the thoracic organs qualifies as a thoracologist. He first graduates from medical school, and usually undertakes appropriate training in internal medicine to qualify for examination of the American Board of Internal Medicine. When this is complete, he seeks further training and experience leading to another examination. When this is successfully completed, he is certified as a subspecialist in pulmonary diseases. The heart, the great vessels and the esophagus are located within the thorax, but diseases of these organs fall within the province of the cardiologist and the gastroenterologist. The physician skilled in internal medicine does not practice surgery himself, but is called upon frequently to evaluate whether the patient may benefit from surgical intervention, or whether the patient has sufficient pulmonary or cardiac function to undergo surgery.

Physical trauma produces disease and aggravates pre-existing disease. A stab or bullet wound to the chest is ordinarily cared for by the surgeon and either results in successful re-establishment of thoracic integrity, or may rapidly prove fatal. If consulted following thoracic trauma, the internist is more likely to be concerned with precipitation of symptoms or aggravation of an underlying disease from blunt injury without penetration of the skin or thoracic cavity. Should blunt trauma produce a rib fracture, then internal bleeding and even puncture of the lung with pneumothorax can occur resulting in serious intrathoracic hemodynamic alterations, which may be fatal if left untreated. Inhalation of irritating or harmful dusts or chemicals is another form of trauma which may produce or aggravate disease.

LUNG FUNCTION

The primary function of the lungs is to maintain an equilibrium between the gaseous environment and the concentration of oxygen and carbon dioxide in our blood and tissues. Pathologic processes alter one or another of the several subdivisions of lung function and eventually disrupt gaseous equilibrium. These subdivisions can be classified as ventrillatory function, distribution of air within the lungs, diffusion of air from the air spaces into the blood and per-

fusion of the lungs by blood via the circulation. By appropriate physiological measurement the thoracologist can identify such abnormalities as airway obstruction, volume loss, or decrease in diffusion capacity.

PNEUMOCONIOSIS

This group of diseases is due to identifiable inhaled irritants. Silicosis is the best known and results from the inhalation of silica in particles from 0.5 to 5 microns in size. The disease is not likely to develop unless particles of the appropriate size exist in concentrations greater than five million per cubic foot of air,[1] and the individual is exposed for a prolonged period of time. The duration of exposure required to produce the disease will vary with the intensity of exposure and the susceptibility of the individual. The silica particles are deposited in the alveoli of the lung and produce fibrous tissue.[2] In a susceptible individual an exposure as brief as several months can result in silicosis. Since it may take as long as fifteen or twenty years, there may be confusion as to which job caused the disease in workmen who change employers but continue to be exposed. Even a relatively low level of exposure, if continued for a long period of time, will produce changes apparent by chest X-ray examintion. To determine if these changes have produced disease, the functions of the lung should be measured. Silicosis is manifest by a reduction in both volume measurements and flow rates. In early cases the major abnormality may be a reduction in the exercising diffusing capacity.[3]

Many classifications of silicosis are based solely upon X ray abnormalities with no consideration of functional consequences. The most useful classification is that recommended by the International Labor Organization, as modified by Cochrane.[4] This divides pneumoconiosis into both simple and complicated, and lists definitions for three degrees of each group. The presence of infection in the lungs of a patient with silicosis greatly aggravates any functional im-

1. Lanza, *Silicosis* in THE PNEUMOCONIOSES 2 (Lanza, Grune & Stratton ed. 1963).

2. Nagelschmidt, *A Review of the Theories of Silicosis,* in INDUSTRIAL PULMONARY DISEASE, A SYMPOSIUM 59-68 (King & Fletcher ed. 1960).

3. BATES & CHRISTIE, RESPIRATORY FUNCTION IN DISEASE 385 (1964).

4. Cochrane, Davies, Chapman, & Rae, 13 BRIT. J. IND. MED. 231 (1956).

pairment which may exist. A common cause of infection in the silicotic lung is the tubercle bacillus. The combination—silico-tuberculosis—is a common cause for an entity called "progressive massive fibrosis."[5] The exact role of tuberculosis in the production of this catastrophic combination is unclear. Tuberculosis may be either the major causative mechanism, or a significant contributing factor.[6] In Chicago and Cook County the foundry industry is a common source of silicosis. In the manufacture of metal products requiring the pouring of hot metals into molded forms covered with sand, some workers are at high risk, particularly those who "shake out" or clean the "burrs" or rough edges from the fresh castings, those who prepare the molds, the "core makers," and those who pour the metal into the mold, particularly if they do this from an open overhead crane. We have also encountered silicosis among fire brick layers who reline blast furnaces using a special high silica content brick. The bricklayer who uses common brick or the construction worker has no appreciable silica exposure. Silicosis develops in workers engaged in sandblasting, sometimes after only a short period of employment. I recall one patient who claimed to have worked constantly under a protective hood with an external source of air, but still developed silicosis.

Not every patient who is exposed to the risk of developing silicosis acquires the disease. If the patient is exposed for a sufficient period of time, he develops abnormalities, visible by X ray, consisting of fine nodular shadows scattered throughout both lungs. This X-ray abnormality results from the deposition of particles of silica dioxide and the reaction of the lung and lymphatic tissue to the retained dust. In the first stage of fine disseminated nodules, the fibrotic reaction does not interfere with lung function, and the patient is essentially asymptomatic.[7] When and if the nodules increase in size, a greater volume of lung tissues becomes affected. The possibility of functional impairment increases directly with the number and size of the nodules. When these nodules appear to coalesce, thereby creating large dense

5. CHRISTIE, PNEUMOCONIOSIS 530 (Beeson & McDermott ed. 1967).

6. Cochrane, *Epidemiology of Coal Workers Penumoconiosis*, in INDUSTRIAL PULMONARY DISEASES, A SYMPOSIUM (King & Fletcher ed. 1960).

7. JONES, KUSHNER, STEELL, & RENZETT, DEFINITIONS AND CLASSIFICATIONS OF NONINFECTIOUS REACTIONS OF THE LUNG (1966).

shadows on the X-ray film, there is definite functional impairment and symptoms.[8] When the nodulation reaches the advanced stage described as progressive massive fibrosis with large, dense shadows just off the root of the lung, the disease has entered its final and usually fatal stage. Silicosis is totally and completely preventable but almost as totally and completely untreatable once acquired. The patient with end-stage progressive massive fibrosis has been fatally harmed by his exposure to silicon dioxide, although tuberculosis is a common contributor. Any patient in whom there appears to be a coalescence of nodules on his X-ray film, progressive increase in the size of nodules, or deterioration in his volume or flow measurements has probably been harmed and should definitely be removed from further exposure. The patient with stage one or simple silicosis—fine discrete nodules not associated with symptoms or functional impairment—has silicosis but may wish to continue working in his present position. Responsibility for preventing silicosis rests squarely upon company management and can best be achieved by cooperation between the industrial health engineers and the physicians involved. Disease prevention requires removal of silica particles or, preferably, altering the industrial process so that silica particles are not produced.[9] Because the disease cannot be cured it is obviously unwise to await the development of symptoms or functional impairment before removing the patient from his silicogenic environment. On the other hand, it is also an error to remove from the active labor force every person with simple silicosis, which is detectable only by X ray.

OTHER PNEUMOCONIOSIS

Inhalation of other dusts can produce disease entities similar to silicosis. The clinical, radiographic, and functional differences from silicosis depend upon the particular dust inhaled, as well as the particle size and concentration. The importance of particle size can not be overstressed, since large particles are filtered out by the upper ais passages and only those particles smaller than five microns can reach the alveoli. Particles smaller than one-half micron penetrate to the alveoli, but few are deposited, as they are carried out with the ex-

8. Becklake, *Pneumoconioses*, in HANDBOOK OF PHYSIOLOGY 1603-05 (1965).

9. Hunter, *The Prevention of Silicosis*, in INDUSTRIAL PULMONARY DISEASES, A SYMPOSIUM 225-69 (King & Fletcher ed. 1960).

pired air and, thus, are not a factor in causing disease.[10] Inhaled particles which may be hazardous to health include asbestos, tin oxide, talc, diatomaceous earth, beryllium, and coal dust. Diatomasceous earth comes from the deposit of organic algae and plants (diatoms) from the Miocene period. It is mined principally in California and also in Germany, France, and Italy. It is widely used in dynamite manufacturing, filtering water, making beverages and drugs. When heated and calcinated, it can create a particularly severe form of silicosis.[11] Beryllium is a metal which at ordinary temperatures exists in a gaseous state. If inhaled it may produce a granulomatous type of pulmonary lesion. The immediate effect is a beryllium pneumonia, but fibrosis of lung tissue occurs in the chronic form of the disease. Berylliosis is peculiar in that workers in a plant with equal exposure are affected to variable degrees.[12] Even outside workers around the plant and people living within a moderately distant area from the source of the beryllium may inhale the fumes and, thus, develop the disease.

COAL WORKERS PNEUMOCONIOSIS

"Black Lung" is slang for coal workers pneumoconiosis, which was described in English and Welsh coal miners who worked at the face of the coal vein in the production of coal. These workers did the drilling, undercutting, blasting, or loading of coal.[13] The pathological process consists of formation of a nodule of dust and fibrous tissue that blocks the alveolar ducts and results in distal focal emphysematous changes.[14] It is important to distinguish coal workers pneumoconiosis from the much more prevalent and benign anthracosis. The latter consists of the accumulation of carbon pigment in the hilar lymph nodes and lung parenchyma of city dwellers. Because this is associated with carbon pigment deposition in the pulmonary

10. Davies, *Deposition of Dust in the Lungs: A Physical Process*, in INDUSTRIAL PULMONARY DISEASES, A SYMPOSIUM 44-58 (King & Fletcher ed. 1960).

11. Smith, *Diatomaceous Earth Pneumoconiosis*, in THE PNEUMOCONIOSIS, 26-33 (Lanza, Grune & Stratton ed. 1963).

12. Van Ordstrand, *Berylliosis*, in THE PNEUMOCONIOSIS 73-102 (Lanza, Grune & Stratton ed. 1963).

13. Gough & Heppleston, *The Pathology of Pneumoconioses*, in INDUSTRIAL PULMONARY DISEASES, A SYMPOSIUM 23-36 (King & Fletcher ed. 1960).

14. Perlman, *A Summary of the Conclusions from an International Conference on Coal Workers Pneumoconiosis*, AM. REV. RES. DIS. 102-243, 247 (1970).

lymphatics and in other portions of the lung tissue, it is referred to as "tattooed lungs" or simple pneumoconiosis and seldom produces symptoms.[15] Anthracosis differs from coal workers pneumoconiosis primarily in the amount and particle size of carbon inhaled. Anthracotic nodules can be found in almost every city dweller's mediastinal lymph nodes, but only those working at the coal face in a coal mine are subject to the development of focal emphysema and coal workers pneumoconiosis. This entity should also be distinguished from silicosis that occurs in coal miners who mine hard coal, particularly in areas which require drilling of rock in order to mine the coal. For example, the coal mines in Illinois contain no appreciable amount of silica and, therefore, Illinois coal miners are not subject to silicosis. The only possible source of silica in an Illinois coal mine comes from sand sprayed upon the rails for purposes of braking the vehicles which are used to pull the trucks containing the coal. This does not produce the particle size, nor the concentration, appropriate for silicosis. An Illinois coal miner may have worked in an anthracite coal mine in Pennsylvania, Kentucky or some other state, moved to Illinois and continue to mine coal. Some coal mining in Illinois has been done by the strip mining process which is not associated with coal workers pneumoconiosis but, unfortunately, is attended by considerable disruption of appearance and ecology of the Prairie State.

ASBESTOSIS

The inhalation of asbestos fibers can result in an alteration of lung function and structure known as asbestosis. The asbestos fibers are long, spindle shaped and pointed at the ends. Although the larger particles are filtered out in the upper air passages some of the fibers which penetrate into the lower airways may be as long as fifty microns.[16] Asbestosis has been observed among men employed as miners, millers, and manufacturers of asbestos products and in workers who apply asbestos as insulation to pipes, ducts, and as automo-

15. Ball, *The Natural History and Management of Coalworkers Pneumoconiosis,* in INDUSTRIAL PULMONARY DISEASES, A SYMPOSIUM 241-54 (King & Fletcher ed. 1960).

16. Smith, *Asbestos,* in THE PNEUMOCONIOSIS 13-25 (Lanza, Grune & Stratton ed. 1963).

bile undercoating. Asbestos usage has increased steadily and the possibility of widespread contamination exists.[17] Asbestos is used in the brake lining materials of American made automobiles and this may be a source of environmental contamination by asbestos. Asbestos fibers surrounded by fibrous tissue reaction have been seen at autopsy in the lungs of patients who never worked with asbestos. Examination of the sputum of healthy non-asbestos workers revealed peculiar objects which were thought to be asbestos fibers. These were called "ferruginous bodies" because the coating took a histochemical stain containing iron. The finding of ferruginous bodies in the sputum and lung tissue of the general population raises the possibility that these are derived from inhaled asbestos.[18] The question has not been settled since it was learned that not all ferruginous bodies were due to asbestos inhalation.[19]

ASBESTOS AND NEOPLASTIC DISEASE

There is firm evidence linking the inhalation of asbestos with the production of neoplasms. A tumor of the pleural space, called a mesothelioma, was first noted to be more common among asbestos workers and those living near asbestos mines and plants.[20] [21] A long term study of asbestos workers revealed that no conclusions could be drawn regarding development of mesothelioma and asbestos exposure prior to twenty years.[22] Among asbestos workers observed for longer than twenty years the mortality rate from cancer of all types was increased two and one half times the expected incidence. Mesothelioma, an otherwise very rare disease, was common in these patients. Asbestos exposure is not the sole factor in this increased incidence of cancer. Among asbestos workers who

17. Lynch, *Brake Lining Composition Products*, 18 J. AR. POLLUT. CONSTR. ASS'N. 824 (1968).

18. Utidjian, Gross & de Treville, *Ferruginous Bodies in Human Lungs*, 17 ARCH. OF ENVIRONMENTAL HEALTH 327 (1968).

19. Gaensler & Addington, *Asbestos or Ferruginous Bodies*, 280 NEW ENG. J. MED. 488 (1969).

20. Fowler, Sloper, & Werner, *Exposure to Asbestosis and Mesothelioma of the Pleura*, 2 BRIT. MED. J. 211 (1964).

21. Owen, *Diffuse Mesothelioma and Exposure to Asbestos Dirt*, 2 BRIT. MED. J. 214 (1964).

22. Selikoff, Churg & Hammond, *The Occurrence of Asbestos Among Insulation Workers in the United States*, 132 ANN. N.Y. ACAD. SCI. 139 (1965).

smoked, the incidence of bronchogenic carcinoma was ten times that anticipated, but among those asbestos workers who did not smoke cigarettes there were no cases of bronchogenic carcinoma or mesothelioma.[23] Cases of mesothelioma have been reported where the patient did not give a history of asbestos exposure when first questioned, but subsequently remembered a short period of asbestos work twenty-five years previously.[24]

Does the asbestos worker who dies of bronchogenic carcinoma twenty or more years after working with asbestos have a legitimate claim for damages? It would appear that he has as much reason to recover damages from the cigarette manufacturer as he does from the asbestos manufacturer.[25]

ORGANIC DUSTS

The inhalation of dusts originating from plants or animals may produce diseases which are specific for a particular occupation. Farmer's lung is an asthmatic or allergic type of shortness of breath which follows exposure to moldy feed grains.[26] When the susceptible farmer shifts the grain within his barn during the winter, he frequently develops sudden onset of shortness of breath, tightness in the chest and physiological evidence of airway obstruction.[27] This disease entity is not well known in the workmen's compensation litigation field because many farmers are self-employed. Nevertheless, it is a well recognized disease entity resulting from exposure to a specific and known condition of employment. The disease may become chronic if exposure is repeated, with permanent physiological changes even though symptomatic improvement follows removal from the offending feed grains and mold spore.[28]

23. Selikoff, Hammond & Churg, *Asbestos Exposure, Smoking, and Neoplasia*, 204 J.A.M.A. 106 (1968).

24. Gracey, Cugell, Nam, Calloway & Buckingham, *Pulmonary Complications of Asbestos Exposure*, 59 CHEST 77-81 (1971).

25. Wright, *Asbestos and Health 1969*, 100 ANN. REV. OF RESPIRATORY DISEASES 467-79 (1969).

26. Bishop, Melnick & Raine, *Farmers Lung*, 32 QUART. J. MED. 257 (1963).

27. Emmanuel, Wenzel, Bowerman & Lawson, *Farmers Lung*, 37 AM. J. MED. 392 (1964).

28. Rankin, Jaeschke, Calties & Dickie, *Farmers Lung: Physiologic Features of the Acute Interstitial Granulomatous Pneumonitis of Agricultural Workers*, 57 ANN. INT. 606 (1962).

A number of other specific disease entities have been described following exposure to specific employment conditions. Among these are maple bark stripper's disease,[29] pigeon breeder's lung,[30] vineyard sprayer's lung,[31] malt worker's lung,[32] coffee worker's lung,[33] thatched roof worker's lung,[34] pituitary snuff inhaler's lung,[35] small pox handler's lung,[36] furrier's lung,[37] byssinosis (cotton worker's lung),[38] and bagasossis,[39] which is the result of handling of the sugar cane residue or bagasse.

A common feature to all of these diseases is an immunological reaction to the specific inhaled substance, which results in an allergic form of asthmatic bronchitis. If the offending material is repeatedly inhaled, a pneumonitis or alveiolitis may occur and progress, resulting in permanent tissue change. The colorful terms paprika splitter's lung[40] and wheat weevil disease[41] are additional examples of lung disease due to organic dusts. They may also be considered as specific subtypes of farmer's lung.

When the manifestations of these diseases are primarily acute shortness of breath, then the most dramatic changes in pulmonary function are the reduction of airflow measurements. In the more chronic form of farmer's lung, diffusion measurements are impaired and there is a reduction in the volume measurements. Some of these

29. Towey, Sweany & Huron, *Severe Bronchial Asthma Apparently Due to Fungus Spores Found in Maple Bark*, 99 J.A.M.A. 453 (1932).

30. Reed, Sosman & Barbee, *Pigeon-Breeder's Lung*, 193 J.A.M.A. 261 (1965).

31. Pimentel & Marques, *Vineyard Sprayer's Lung*, 24 THORAX 678 (1969).

32. Riddle & Grant, *Allergic Alveolities in the Maltworker*, 22 THORAX 478 (1967).

33. Van Toorn, *Coffee Worker's Lung*, 25 THORAX 99 (1970).

34. Blackburn & Green, *Precipitins Against Extracts of Thatched Roofs in the Sera of New Guinea Natives with Chronic Lung Disease*, 2 LANCET 1396 (1966).

35. Mahon, Scott, Ansell, Manson & Fraser, *Hypersensitivity to Pituitary Snuff with Miliary Shadowing in the Lungs*, 22 THORAX 13 (1967).

36. Evans & Foreman, *Smallpox Handler's Lung*, 56 PRO. ROY. SOC. MED. 274 (1963).

37. Pimental, *Furrier's Lung*, 25 THORAX 387-98 (1970).

38. Schilling, *Byssinosis in Cotton and Other Textile Workers*, 2 LANCET 261-319 (1956).

39. Buechner, Prevatt, Thompson & Blitzo, *Bagassosis*, 25 AM. J. MED. 234 (1958).

40. HUNTER, DISEASES OF OCCUPATIONS (4th ed. 1969).

41. Lunn & Hughes, *Pulmonary Hypersensitivity to the Grain Weevil*, 24 BRIT. J. OF INDUST. MED. 158 (1967).

diseases have characteristic features such as the Monday morning dypnea of the cotton carding room workers. In other diseases, circulating antibodies to the specific dusts have been demonstrated and immune globulin (IgG) and compliment have been identified by immunoflourescence along the alveolar capillaries. In animal experiments the inhalation of fur dust by guinea-pigs produced lung changes similar to those found in patients with furrier's lung disease. There seems to be little doubt that the disease is caused by the inhaled dust. The extent to which the patient may be disabled can best be measured by pulmonary function studies.

CHEMICAL IRRITANTS

Numerous volatile chemicals produce respiratory symptoms—mostly the result of direct irritation—but some also provoke progressive tissue reaction. Acute toxic, and even fatal, inhalation of various oxides of nitrogen (NO & NO_2) has been reported in workers who entered the upper port of agricultural silos that were partially filled with silage. This has been called "Silo Filler's Disease"[42] and is distinct from farmer's lung. The oxides of nitrogen accumulate as a result of fermentation of the silage. Similar toxic gases accumulate in mines after explosions[43] and in some industrial processes.[44] Following non-fatal exposure a severe chemical pneumonia develops within several hours. Resolution is prolonged and permanent lung changes occur resulting in pulmonary insufficiency.[45] Inhalation of smoke containing nitrous oxides may produce the acute chemical pneumonia of silo filler's disease. Artificial smoke bombs containing zinc chloride may produce an acute, fatal interstitial pneumonitis.[46] The inhalation of ammonia, sulfur trioxide (SO_3), phosgene, and Lewisite[47] have been reported to cause or aggravate chronic pulmonary disease, but this relationship is difficult to prove.

42. Lowry & Schuman, *Silo Filler's Disease—A Syndrome Caused by Nitrogen Dioxide,* 162 J.A.M.A. 156 (1956).

43. Becklake, Goldman, Bosman, & Freed, *The Long Term Effects of Exposure to Nitrous Fumes,* 76 AM. REB. TUBER. 398 (1957).

44. Drake & Warrack, *Bronchiolitis From Nitrous Fumes,* 13 THORAX 327 (1958).

45. Leib, Davis, Brown & McQuiggan, *Chronic Pulmonary Insufficiency Secondary to Silo Filler's Disease,* 24 AM. J. MED. 471 (1958).

46. Milliken, Waugh & Kadish, *Acute Interstitial Pulmonary Fibrosis Caused by a Smoke Bomb,* 88 CANAD. M. A. J. 36 (1963).

47. Nishimoto, Burros, Miyanishi, Katsuta, Shigenobo & Kettel, *Chronic Ob-*

The inhalation of cadmium oxide produces an acute chemical pneumonitis which may be fatal,[48] or which may produce a type of emphysema.[49] Exposure may occur in welders who use cadmium-containing metals or in workers who use copper-cadmium alloys.[50] The inhalation of ozone may produce a similar acute chemical pneumonitis.[51] Volatile hydrocarbons (gasoline, kerosense, benzene, etc.) are inhaled frequently, but it is questionable if they produce pulmonary damage. Tolune Di-isocyanate is used in the manufacture of some plastics and inhalation is reported to produce bronchial changes,[52] but there are no measurements of the effects on pulmonary function.

DRUGS AND RADIATION THERAPY

Therapeutic agents for hypertension[53] (Hexamethonium & Apresoline) and lukemia[54] (Busulfan) have been associated with the development of interstitial pneumonia manifested by progressive dyspnea. The best indication that the pulmonary changes are due to the drugs is apparent reversal when the drug is withdrawn. Somewhat similar but milder changes have been reported after use of the antibiotic, Furadantin. Therapeutic radiation may cause a pneumonitis that heals with fibrosis.[55]

The possible harmful effects of any therapeutic measure must be weighed against the course of the underlying disease and the intended beneficial result. It is often difficult to separate manifestations of the disease from effects of the treatment.

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structive Lung Disease in Japanese Poison Gas Workers, 102 AM. REV. RESP. DIS. 173 (1970).

48. SPENCER, PATHOLOGY OF THE LUNG 80 (1962).

49. Lane & Campbell, *Fatal Emphysema in Two Men Making a Copper Cadmium Alloy*, 11 BRIT. J. INDUST. MED. 118 (1954).

50. Bonnell, *Emphysema and Protienuria in Men Casting Copper Cadmium Alloys*, 12 BRIT. J. INDUST. MED. 181 (1955).

51. Jaffe & Estes, *Ozone Toxicity in Cabins of High Altitude Aircraft—A Review and Current Program*, 34 AEROSPACE MED. 633 (1963).

52. Trenchard & Harris, *An Outbreak of Respiratory Symptoms Caused by Toluene Di-isocyanate*, 1 LANCET 404 (1963).

53. Peterson, Dodge & Helwig, *Pulmonary Changes Associated with Hexamethonium Therapy*, 103 A.M.A. ARCH. INT. MED. 285 (1959).

54. Oliner, Schwartz, Rubio & Damashek, *Interstitial Pulmonary Fibrosis Following Busulfan Therapy*, 31 AM. J. MED. 134 (1961).

55. Smith, *Radiation Pneumonitis*, 87 AM. REV. RESP. DIS. 647 (1963).

DISEASE OF QUESTIONABLE ETIOLOGICAL RELATIONSHIP

The most common disease incorrectly alleged to be due to adverse work conditions is tuberculosis. It results from infection with the tubercle bacillis, but the disease may not occur until many years after exposure. Other forms of the tubercle bacillus include Mycobacterium avium which infects birds but is of very low pathogenicity for man, and Mycobacterium bovis—a disease of cows which formerly accounted for considerable human tuberculosis. Elimination of this disease from cattle has resulted in its disappearance as a human disease. Reactivation of tuberculosis following trauma may occur, particularly if extensive. Claims for damages because of reactivation of tuberculosis often occur when a patient sustains relatively minor trauma to the chest or shoulder and has an X-ray film taken in order to determine if a fracture is present. The coincidental and fortuitous discovery of pulmonary tuberculosis on such an X-ray film is the basis for a subsequent claim of traumatic aggravation. Since the tuberculosis is usually asymptomatic and its discovery following trauma is actually beneficial since it leads to adequate treatment in an early stage, this can hardly be the basis for an injury claim.

PNEUMOTHORAX

Penetrating wounds of the chest cause the entry of air into the pleural space and result in collapse of the lung. Blunt trauma can produce a rib fracture, the fractured end of the rib then penetrates the visceral pleura and air escapes from within the lung into the pleural space resulting in a traumatic pneumothorax but without penetration of the skin. A rib fracture is essential for this sequence to occur. Traumatic pneumothorax can be distinguished from spontaneous pneumothorax which results from rupture of the air spaces of the lung with leaking of air into the pleural space and lung collapse. Spontaneous pneumothorax is caused by diseased lung, usually small cysts or blebs, and is not the result of trauma. A number of cases in which the patient had a spontaneous pneumothorax and alleged this to be the result either of working or of minor trauma sustained during the course of employment have been encountered. Even though the onset of the tear in the pleura may have occurred during working hours, during the act of performing some specific

task, there is no evidence that the work caused, aggravated, or precipitated the lung collapse, since spontaneous pneumothorax commonly occurs while the subject is at rest and has no known precipitating or aggravating factors.

PULMONARY EMBOLIZATION

Formation of blood clots (thrombi) in the vascular system which break off and lodge in the lung (embolism) is a common medical problem. It is not usually related to employment or to minor trauma. The immobilization necessary for the treatment of traumatic fractures of the extremities may result in venous stasis and thrombosis. If pulmonary embolus subsequently occurs, there would be at least an indirect causal relationship between the original trauma and the pulmonary embolus. Trauma resulting in large bone fractures may permit release of bone marrow fat into the venous system and fat embolization of the lungs.

DOUBTFUL DISEASE ENTITIES

Disease entities based upon one or only a few observations, but not confirmed by others, create many problems. It was alleged that the inhalation of hair sprays produced abnormalities of lung structure. The term "hair spray thesaurosis" was introduced to describe this entity.[56] Subesequent studies of particle size of the hair sprays indicated there was minimal alveolar penetration. Epidemiological studies on people employed in the beauty industry who used hair sprays extensively failed to reveal any increased incidence of respiratory disease, nor was it possible to reproduce the entity in animal experiments.[57] "Hair spray thesaurosis" is not now considered a disease, but rather plumonary sarcoidosis which is coincidental to use of hair spray.[58]

BLUNT TRAUMA & CARDIAC DISEASE

Blunt or nonpenetrating trauma to the chest can result in com-

56. Bergman, Flance & Blumenthal, *Thesaurosis Following Inhalation of Hair Spray—A Clinical and Experimental Study*, 258 NEW ENG. J. MED. 471 (1958).

57. Herrero, Fiegelson & Becker, *Sarcoidosis in a Beautician*, 92 AM. REV. RESP. DIS. 280 (1965).

58. Schepers, *Thesaurosis Versus Sarcoidosis*, 181 J.A.M.A. 635 (1962).

pression of the heart between the sternum and the vertebral bodies.[59] This occurs in steering wheel and similar type injuries with the force applied directly to the heart muscle. If there is myocardial damage, signs thereof will be apparent either immediately or after several hours, including electrocardiographic changes and elevation of enzymes from heart muscle. Blunt trauma may produce valve rupture, injury to a coronary artery, or rupture of the great vessels.

MYOCARDIAL INFARCTION

The patient who develops a myocardial infarction secondary to a coronary artery thrombosis while at work represents a difficult problem because he often resorts to litigation alleging a causal relationship between his work and the cardiac disease. Myocardial infarction can be readily diagnosed and treated, although not always successfully. The usual case is arteriosclerotic narrowing of a coronary artery, which subsequently becomes thrombosed. The cause of the coronary arterial disease is unknown but is now the subject of numerous epidemiological studies. The risk of coronary thrombosis is increased if there is: serum cholesterol elevation; blood pressure elevation; obesity; cigarette smoking; elevated serum triglycerides; abnormal carbohydrate metabolism; or sedentary habits. Physical activity is associated with fewer episodes of myocardial infarction as has been shown by the incidence in letter carriers versus postal workers, bus conductors versus bus drivers, working farmers versus non-working farmers, and railroad section hands versus railroad clerks.[60]

Ever since Kohn described "Angina Pectoris Traumaticum" in 1929, the possibility has existed of a patient with myocardial infarction recovering damages from his employer. In a careful analysis of the factors involved, Boas[61] concluded that an etiological relationship existed only if the patient was previously normal, developed the characteristic chest pain while engaged in unusually strenuous work, and developed signs of cardiac disease immediately or after a short interval during which symptoms persisted. In a

59. WARBURG, SUBACUTE & CHRONIC PERICARDIAL AND MYOCARDIAL LESIONS DUE TO NON-PENETRATING MYOCARDIAL INJURIES (1938).

60. Epstein, *The Epidemiology of Coronary Heart Disease*, 18 J. OF CHRONIC DIS. 735 (1965).

61. Boas, *Angina Pectoris & Cardiac Infarction from Trauma or Unusual Effort*, 112 J.A.M.A. 1887 (1939).

careful analysis, Masters, *et al.*,[62] concluded that work seldom precipitated myocardial infarction, but cardiac arrythmias and shock immediately following trauma may precipitate myocardial infarction.[63]

Social and psychological factors have been implicated as possible precipitating factors for coronary artery disease, but it is questionable if these are significantly independent of the risk factors mentioned above. Aggravation of pre-existing congestive heart failure commonly results from overexertion or excessive physical activity whether related to employment or not. This is partly responsible for the common but erroneous view that hard work causes heart trouble.

ESOPHAGEAL DISEASE

Lower esophageal disease may resemble cardiac disease because of the similar location of the pain that is produced. Regurgitation of gastric contents and sometimes even herniation of portions of the stomach up into the thorax (hiatus hernia) may be considered as the basis for legal action, since the symptoms frequently occur when lifting or stooping. It is frequently associated with obesity and is probably better related to excessive food intake than to excessive physical exertion. "Spontaneous rupture" of the esophagus occurs with violent vomiting, usually among alcoholics. A tear occurs in the esophageal mucosa with disastrous results. Spasm of the lower end of the esophagus may result in the accumulation of food and secretions in the esophagus which can spill over into the lungs and cause a chronic scar tissue accumulation. Out pouchings (diverticuli) of the esophagus may also account for accumulation of ingested food and sometimes may be associated with development of abnormal communication between the trachea and the esophagus (tracheal-esophageal fistula).

GREAT VESSEL DISEASE

Blunt or penetrating wounds of the chest may result in damage to the great vessels with either immediate rupture or subsequent

62. Masters, Dack & Jaffe, *Factors & Events Associated with the Onset of Coronary Artery Thrombosis*, 109 J.A.M.A. 546 (1937).

63. Muritz, *Trauma, Stress & Coronary Artery Thrombosis*, 156 J.A.M.A. 1306 (1954).

weakening and dilitation of the vessel. It is important to be sure that the offending trauma actually produced the abnormalities noted. An example of this is a young girl who was stabbed in the chest with an ice pick and was found to have an enlargement adjacent to the aorta just beneath the stab wound. Surgery to correct this supposed traumatic aortic lesion revealed a benign congenital cyst located in the area in question and no evidence of aortic damage.

CONCLUSION

Trauma and/or working conditions may be the cause or may aggravate or precipitate pre-existing thoracic disease. More frequently, neither trauma nor working conditions are related to disease entities existing as a result of pathological processes. In order to determine if any relationship exists in a specific case, it is necessary to survey the working conditions, review the alleged trauma or precipitating factors, and then thoroughly evaluate the disease entity in question. Only after these steps have been taken can the practicing attorney reasonably evaluate the advisability of litigation.