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INTRODUCTION TO LUNG DISEASES

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CHAPTER 4

PULMONARY EMPHYSEMA

Pulmonary emphysema is an anatomical change in the lungs characterized by enlargement of the air spaces beyond the terminal bronchioles with some destruction of the air sac walls. Functionally, emphysema is characterized by dyspnea (shortness of breath) due to difficulty in expelling air from the lungs and poor distribution of the inhaled air throughout them. The disease has also been called chronic obstructive emphysema, hypertrophic or large-lunged emphysema, and obstructive ventilatory disease of the lungs.

Two centuries ago chronic bronchitis, pulmonary emphysema, and bronchial asthma were all simply called "asthma." Advances in pathology, auscultation of the lungs, radiology, immunology, and physiology have differentiated them. In Great Britain, chronic bronchitis is the predominant diagnosis in cases with combinations of the three conditions.

The introduction of exact methods of measuring lung ventilation, volumes, and gas exchange has made it possible to determine degrees of functional impairment due to emphysema. A revival of interest in lung pathology, using new and more refined techniques, has provided a basis for differentiating the various types of emphysema and defining the disease in internationally accepted terms. Primary emphysema, which may follow years of chronic bronchitis, may start either in the center of the lung lobules (centrilobular type) or throughout (panlobular type). Localized emphysema produced by any condition causing scarring in the lung is called paracicatricial emphysema. Bullous emphysema, a

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type with large airspaces (over one inch in diameter), occurs to some degree as part of ordinary pulmonary emphysema. Such air spaces are called bullae or blebs. They may be the dominant feature of the changes found in the lungs.

A clear distinction must be made between chronic pulmonary emphysema and emphysema that is due to aging or is compensatory. Emphysema resulting from aging is of the panlobular type. Compensatory emphysema is a simple distention of the lung occurring when it expands to fill a space caused by surgical removal or by contraction of a lung segment or lobe. This is not true emphysema; it is non-obstructive overinflation. Overinflation without destructive changes in air cells can also be caused by a valve-like bronchial obstruction (foreign body, tumor, secretions).

CAUSE

There is no known single, primary cause of chronic pulmonary emphysema. The condition is more common in men than women, suggesting that smoking is a major factor, since there are more long-term heavy smokers among men than among women—a situation that is rapidly changing: As smoking among women increases, so does their share of emphysema.

Another factor—a hereditary lack of a blood protein, alpha$_1$ antitrypsin—accounts for a small number of emphysema cases. Individuals who inherited this protein deficiency have been seen to have more obstructive lung disease and at earlier ages than those with the normal supply of the protein.

The more immediate cause of emphysema is, in most instances, recurrent or chronic inflammation and functional obstruction of the bronchioles, the smaller branches of the bronchial tree that carry air to and from the primary units of the lung, the lobules. A natural tendency to trap air in the lobule exists because all the airways widen on inspiration and narrow on expiration. Inflammatory swelling of the bronchial walls, thick or excessive secretions, and external compression by surrounding distended air spaces may all produce varying degrees of bronchiolar obstruction during expiration.

Some air may still make its way into lobules, even when isolated by complete obstruction during the inspiratory phase of breathing, by way of connecting pores (collateral respiration); this will increase the volume of trapped air and the distention. Thus, constant and increasing stretching occurs around the central bronchiole until alveolar walls with their web of capillaries begin to break down, an irreversible change. A deficient blood supply for gas exchange eventually results, but whether an inadequate blood supply to the lung plays any part in causing emphysema is not known.

In England it is exceptional to find a patient with emphysema who has not also had chronic bronchitis. Data now being collected in this country indicate

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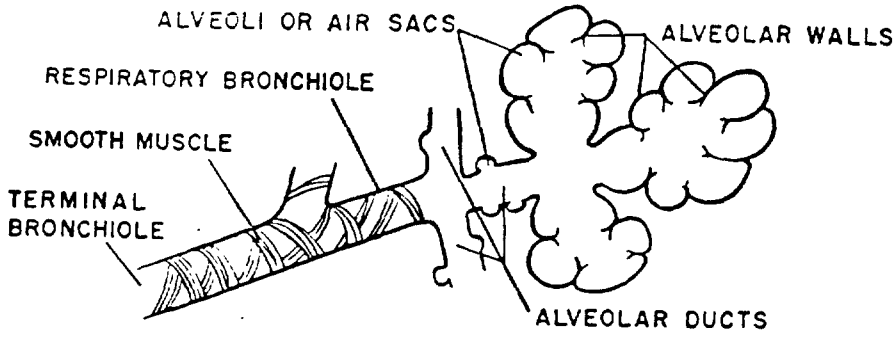
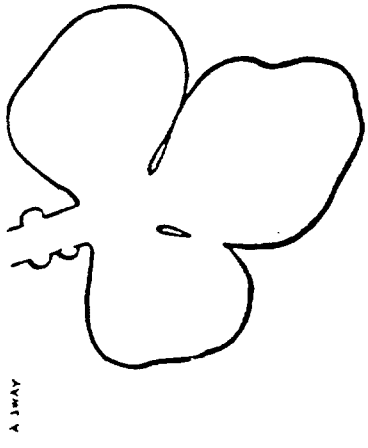
EMPHYSEMA

NORMAL PRIMARY LOBULE
(after Miller)

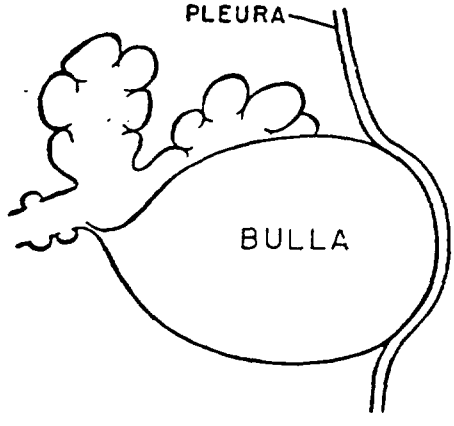


EMPHYSEMATOUS LOBULE

WITH BREAKDOWN OF ALVEOLAR WALLS



EMPHYSEMATOUS BULLA AT SURFACE OF LUNG



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that about two-thirds of the people with emphysema have previously had cough and expectoration and that another 18 to 20 percent develop these symptoms after emphysema is clinically apparent.

More than 90 percent of emphysematous patients in some studies are heavy smokers—that is, smoke more than 20 cigarettes a day—indicating that such smoking is a causative factor in emphysema. Air pollution also contributes to the incidence of emphysema. Conditions that produce extensive scarring in the lungs, such as silicosis, sarcoidosis, and tuberculosis, have an end stage of which pulmonary emphysema is a part. Localized scarring accounts for the uneven distention resulting in bullous emphysema.

PATHOLOGY

When the chest is opened at surgery or autopsy, the lungs in chronic pulmonary emphysema are grossly very much enlarged. They are whiter than normal and feel downy. Large, clear blebs (air blisters) may be seen along the margins. On close examination of the lung tissue, the individual air spaces are so enlarged as to be easily seen with the naked eye.

Microscopically, the walls of the alveoli are thinned out, stretched, and often broken down, with several such primary air spaces thrown together, thus diminishing the total surface area carrying capillaries in close contact with alveolar air. When this process is localized around the terminal bronchioles, sparing the alveoli in the periphery of the lobule, it is called centrilobular in type. When the whole lobule is involved it is panlobular. By the time that emphysema has progressed over many years to the point of causing death, the architecture of the lungs may be so destroyed that one cannot determine the original type. The pathologic changes characteristic of chronic bronchitis are commonly also present, and there may be scarring due to other chronic disease.

NATURAL HISTORY

Emphysema begins insidiously. The victim usually cannot date even the year of onset. Repeated winter colds with increasing duration of cough and expectoration are followed by the clinical picture of chronic bronchitis merging into emphysema. Less commonly, shortness of breath begins gradually without cough in a middle-aged man, who notices it first on exertion or on arising in the morning. This shortness of breath, unlike that due to heart failure, is relieved by lying down. It progresses relentlessly from an inconvenience to a steady struggle to ventilate the lungs during all the waking hours.

As the patient reaches the limits of capacity for excessive work in breathing and of endurance of lowered blood oxygen, he can no longer carry on any physical activity. In the final stage, the overloaded heart reaches its limit of

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muscular compensation and begins to fail (cor pulmonale). The destruction of a large part of the capillaries in the lungs, the effect of lowered blood oxygen in raising the pulmonary artery pressure, and the shunting of unaerated blood, all play a part in overworking the right ventricle of the heart. A large proportion of patients with advanced emphysema die of heart failure.

COMPLICATIONS

Chronic bronchitis, recurrent bouts of acute infection, and cor pulmonale can be regarded as part of the whole picture of chronic pulmonary emphysema. Pulmonary embolism is common. Peptic ulcers are so often associated with emphysema that some link of common causation is suggested.

PROGNOSIS

The prognosis is bad in all patients whose disease has progressed to the stage in which it can be diagnosed by the usual methods. However, modern treatment methods may improve lung function and prolong life in most cases. Occasionally a man in his forties of the "driving" type, who smokes a great deal, develops a rapidly progressive, dry type of emphysema and dies within a year or two. But most patients may carry on useful lives for a number of years in spite of emphysema.

OCCURRENCE

Survey data indicate that chronic pulmonary emphysema is increasing in incidence. The number of deaths caused primarily by emphysema doubled in a recent ten-year period. How much of the increase is due to better medical recognition and how much may be due to aging of the population is not clear. A considerable part must be accounted for by the widespread practice of smoking cigarettes and by environmental conditions.

It is hoped that more generally accepted definitions of chronic bronchitis and emphysema will make possible valid comparative epidemiologic studies and facilitate scientific communication. Emphysema is particularly a disease of white males between 50 and 70 years of age. Women are affected in a ratio of less than one to every ten men.

MANIFESTATIONS

The chief and only essential symptom is shortness of breath. Noticed only on exertion at first, this may be ascribed to smoking, lack of sufficient exercise, or a gain in body weight. When this dyspnea has progressed to the stage at which it interferes with work or play, a physician is finally consulted, generally because of fear of "heart trouble" or "asthma."

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Breathing may become more difficult when the patient is under strain in his business and, conversely, it may be easy on vacation or after a few days of rest in the hospital. Almost any new treatment seems to relieve it temporarily. Acute respiratory infections greatly increase the shortness of breath. Before heart failure develops, dyspnea is relieved by lying down flat: after the heart becomes unable to maintain adequate circulation, dyspnea is continuous and not relieved on reclining. Cough and expectoration vary with the season, the weather, and the recurrence of superimposed acute respiratory infections.

Some blueness of the lips, ear lobes, skin, and fingernail beds is one of the signs in established emphysema. There is often some clubbing of the fingertips. Characteristically, but not invariably, the thorax is deep and almost spherical in shape ("barrel chest"). The neck muscles are in use for even quiet respiration, and it can be observed that expiration calls for active effort.

The lungs give an unusually resonant note when struck in percussion. When a stethoscope is applied, the breath sounds are faint, with a prolonged expiratory phase. There are rales and wheezes in proportion to the amount of bronchitis present at the time of examination. Rather high degrees of bronchiolar obstruction to expiration do not produce the concert of musical sounds heard in asthmatic attacks. Swelling of the ankles appears with beginning heart failure.

LABORATORY FINDINGS

Emphysema cannot be diagnosed from a standard chest X-ray film (14" x 17") or by a 70 millimeter microfilm, showing the postero-anterior view, although when the diaphragm shadow is in a low position this may suggest emphysema. A lateral chest film may be more indicative, showing the depth of the chest and the flattened diaphragm. The difficulty in expiration characteristic of emphysema is revealed on fluoroscopy, which shows the lungs in action, better than on X-ray films.

Bullous emphysema can be diagnosed by plain postero-anterior or lateral chest films, but the bullae show up particularly well on planigrams, that is, body section X-ray films. A characteristic "tree in winter" effect is revealed on bronchograms, with the smaller branches and foliage not outlined, and the changes in the outlines of the bronchial tree characteristic of chronic bronchitis may be observed.

The red blood count increases to compensate for lowered oxygen saturation, just as it does at high altitudes. Therefore, polycythemia (a condition in which there is an excess of red corpuscles) may develop in well-established emphysema. Such a condition is the rule when cor pulmonale has appeared. The white cell count is normal except during episodes of acute infection. When respiratory failure has developed, analysis of the arterial blood shows some

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decrease in oxygen tension and an excess of carbon dioxide. As carbon dioxide accumulates, the acidity of the arterial blood is increased.

A battery of physiological tests and formulas has been developed to diagnose emphysema. These measurements are most useful in establishing the stages of the disease from the earliest impairment of ventilation through oxygen lack and carbon dioxide accumulation to heart failure. An increase in residual air trapped in the lungs, a decreased ability to empty the lungs rapidly as shown by forced expiratory volume in one second (FEV_1), and poor diffusion of gases in the lungs are basic abnormalities. It is important to know the oxygen and carbon dioxide content and the pH (acid-alkali balance) of the arterial blood. The skills and equipment needed for these and many of the other measurements should be available in all hospitals and in other facilities.

METHODS OF DETECTION

When symptoms cause a patient to go to a physician and the disease is diagnosed, it is in a well-developed stage. A careful history and physical examination, including a pulmonary function test, should enable a physician alert to the problem to detect emphysema in earlier stages. Obviously, such an examination is not suitable for mass surveys. Some of the tests for pulmonary function, however, may make possible the detection of those in the population who are beginning to develop emphysema.

METHODS OF CONTROL

The treatment of this condition is essentially palliative, because we do not know all its causes and because irreparable lung damage is its basis. Attempts to reverse the process by the use of hormones and steroids have been unsuccessful. In the patient who has chronic bronchitis or an acute respiratory condition, the first need is to combat infection. This is done by administering antimicrobial drugs by mouth or intramuscularly. There is no advantage in nebulization and inhalation as a means of administering these drugs. For treatment to succeed, cigarette smoking must be discontinued.

Obstruction to the smaller air passages must be relieved by: (a) treating the infection; (b) thinning the secretions by an increase of fluid intake, expectorants and humidifying or wetting agents; and (c) giving drugs that will dilate the bronchi. Such bronchodilators and liquifying agents are most effective when delivered directly into the bronchial tree by nebulization.

Positive pressure set up by expiration against pursed lips or by any other method tends to prolong expiration, thus preventing collapse of bronchioles. Exercise during oxygen administration by mask or nasal prongs may be very

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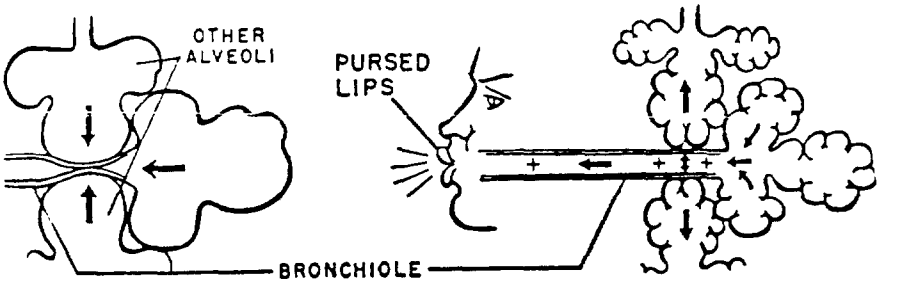
EFFECT OF POSITIVE PRESSURE BREATHING



helpful to the patient's efficiency and sense of well-being in the second stage of the disease. In the latter stages, the use of oxygen in high concentrations, if required, may be dangerous unless done in a hospital where close supervision is available and tests essential to safety can be made. Heart failure should be treated as would any other form of decompensation.

Above all, the patient with pulmonary emphysema needs a physician, skilled and understanding, to treat his symptoms, to help protect him from infection, to prolong his life, and to lend him moral support.

Surgical treatment is indicated for the excision of large, solitary bullae, particularly if these are compressing normal lung tissue adjacent to them. Resection of such non-functioning portions may improve the mechanics of breathing. Surgical treatment designed to improve the circulation of the lungs or to cut their nerve supply has not been successful in producing better function.

PREVENTION

Just as the known causes of emphysema should be removed in the individual case, so should the public be made aware of potentially dangerous habits and conditions, such as cigarette smoking, air pollution, exposure to occupational dust and fumes, and the neglect of bronchial infections.

Individuals suspected of having early emphysema should certainly stop smoking. Annual immunization against influenza is advised for those with any stage of emphysema. Every attack of bronchitis should be treated vigorously and for a sufficient length of time.

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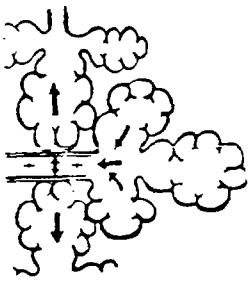

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SOCIAL AND ECONOMIC IMPLICATIONS

Chronic pulmonary emphysema not only is killing more people than ever before, but also is making respiratory cripples of many otherwise strong and hard-working men. Those in white-collar positions can, with good medical care, carry on quite successfully for some years, but the laborer or skilled worker is usually forced to become a charge upon health insurance, union funds, or the public treasury. The costs of hospitalization, medication, oxygen, and apparatus to aid breathing for a single person with pulmonary emphysema may reach a considerable total over a period of years.

APPROACHES BEING USED

Pulmonary emphysema is being vigorously studied as to possible causes, frequency, and best methods of early detection and treatment. Since a more exact definition of emphysema is now internationally available, it is to be hoped that its general use and correlation with accepted physiologic standards will promote better understanding of the early stages of the disease, better measures of prevention, and general agreement on the stages of emphysema and the degrees of disability they produce.

A promising approach at present is the rehabilitative one. The patient who is overborne and discouraged by his symptoms may be more disabled by them than he need be. Help in controlling the symptoms and training in minimizing their effects may be of great benefit in individual cases.

FUTURE APPROACHES

We need to know a great deal more about the causes of emphysema and about the early stages of the condition. Further studies on physical and vocational rehabilitation of those affected by emphysema are also badly needed.

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CHAPTER 5

TUMORS OF THE LUNG

Tumors of the bronchi and lungs are showing an alarming increase in frequency. Because their cure depends upon earlier recognition and education of the public, diagnosis is stressed in the following section.

New growths (neoplasms) may originate in the lung or arise there by extension (metastasis) through the blood or lymphatics from other parts of the body. Metastases to the lungs are frequent because malignant tumor cells entering the bloodstream in any organ must go to the capillaries of the lungs. They are usually scattered through both lungs. They cause fewer symptoms than do primary growths, and their origin, if not previously known, may be found by a thorough examination of the whole body.

Primary tumors of the lung are either *benign* or *malignant*.

BENIGN TUMORS, such as adenomas and hamartomas, crowding into and invading normal tissues, are important chiefly because they sometimes become malignant and because they cause difficulties in differential diagnosis. Adenomas of the bronchus can cause serious hemorrhage, recurring pneumonia or bronchiectasis due to chronic obstruction.

Benign tumors, discovered by routine chest X-rays, can be investigated by bronchoscopy and cytologic studies of the sputum, but usually require surgical exploration and microscopic study of a specimen for diagnosis. The incidence of such tumors is about one tenth that of carcinoma. They occur at a younger age and without any sex differential. Surgical removal is the treatment.

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MALIGNANT TUMORS of the lungs, invasive locally or into the lymphatic system or bloodstream, occur in two principal forms—bronchogenic carcinoma and alveolar cell carcinoma. Other forms, such as sarcoma arising from tissues of non-epithelial origin, are exceedingly rare. Bronchogenic cancer is by far the commonest type of lung tumor.

BRONCHOGENIC CARCINOMA

Bronchogenic carcinoma is a malignant new growth originating in the bronchial mucous membrane and progressing locally and by metastasis to a fatal termination unless discovered early and treated radically. It is the disease meant whenever reference is made to "lung cancer" and remains one of the health problems that most demands study and control.

CAUSE

The primary cause that starts a single cell multiplying in an uncontrolled fashion is unknown, for the lung as for other sites of origin and types of malignant tumors. Secondary causes that produce a chronic irritation of the bronchial mucosa have been under intensive epidemiologic and laboratory study for some time.

There is strong proof that smoking by males, when heavy (more than 20 cigarettes daily) and continued for 25 years or more, greatly increases the probability of developing this kind of cancer. Hereditary factors may be involved both in a tendency to develop cancer and in the type of personality prone to smoke cigarettes excessively rather than to smoke a pipe or not at all. Living and working in a place with constant air pollution adds to the probability of lung cancer in both smokers and nonsmokers. Less widespread causes are working at the mining of radioactive ores or asbestos and working with chromium.

PATHOLOGY

The three types of bronchogenic carcinoma are, in order of frequency: epidermoid or squamous cell carcinoma; undifferentiated or oat cell carcinoma; and adenocarcinoma. The first of these resembles a wild growth of epidermis with cornified epithelium in whorls. It is hard, dense, slow-growing, and slow-metastasizing. The undifferentiated type is the most primitive, with small, uniform, spindle-shaped cells or large cells with deeply staining nuclei. It is rapid in growth and early to metastasize. Adenocarcinomas, as the name implies (adeno means gland), show a cellular organization like bronchial glands that may contain mucus. They are usually peripheral (on the outer edge) in the

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lung, grow rapidly, but do not spread as fast as the undifferentiated type does. Although there is some correlation between the type and the prognosis, for practical purposes they may all be considered a single disease, because the type can be determined only by surgical removal of sections for microscopic examination.

The new growth starts with a cell in the bronchial mucosa and extends both into the lumen (open space) of the bronchus and outward into the lung tissue. There may be early extension into the lymphatics leading to the mediastinal lymph nodes and into the blood vessels. Cells reach the lymph nodes, brain, bones, liver, and adrenals in the course of metastasis. Obstruction of the bronchus by the growth leads to most of the symptoms felt by the patient and signs observed clinically. Invasion of the pleura by the cancer causes serious pain and effusions.

There is great variation in the natural duration of the disease. Three to five years from the earliest X-ray evidence to death is not uncommon. Without treatment death usually occurs within a year of the time of a conclusive diagnosis. This may have been nine months to a year after the first symptom, because of the patient's delay in seeking medical care and the physician's slowness in arriving at the diagnosis. In somewhat over half the cases, the disease imitates an acute respiratory infection, and the temporary improvement after antimicrobial treatment sometimes deludes both physician and patient as to the real nature of the trouble.

COMPLICATIONS

Bronchial obstruction, producing pneumonia or lung collapse beyond it, may cause complications. Invasion of the pleura by the tumor results in an effusion, which is usually bloody. The great vessels or nerves in the mediastinum may be compressed or invaded. Metastasis to distant organs such as the brain or the adrenal glands often occurs.

PROGNOSIS

Even with the earliest diagnosis and best treatment available today, the outlook remains poor, although no longer totally hopeless. Of all patients with lung cancer, little more than 5 percent survive five years after operation. The salvage rate has not been improving. Of those who go to the surgeon before there is evidence of metastasis, about 15 percent survive five years or more. Of those who have the tumor apparently completely resected, from 25 to 35 percent survive more than five years. Forms of treatment other than surgical resection are today able to do no more than relieve symptoms and to prolong life only a matter of months.

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TUMORS OF THE LUNG

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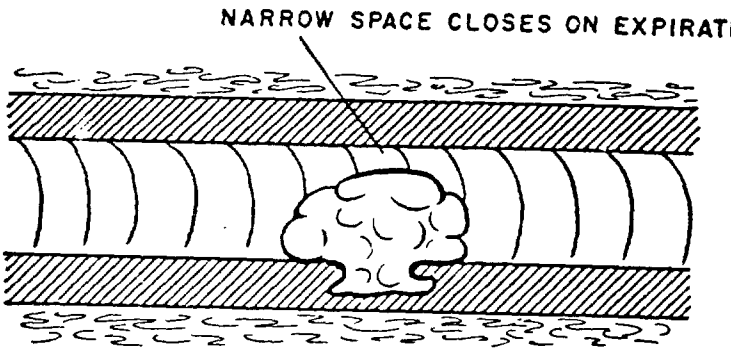
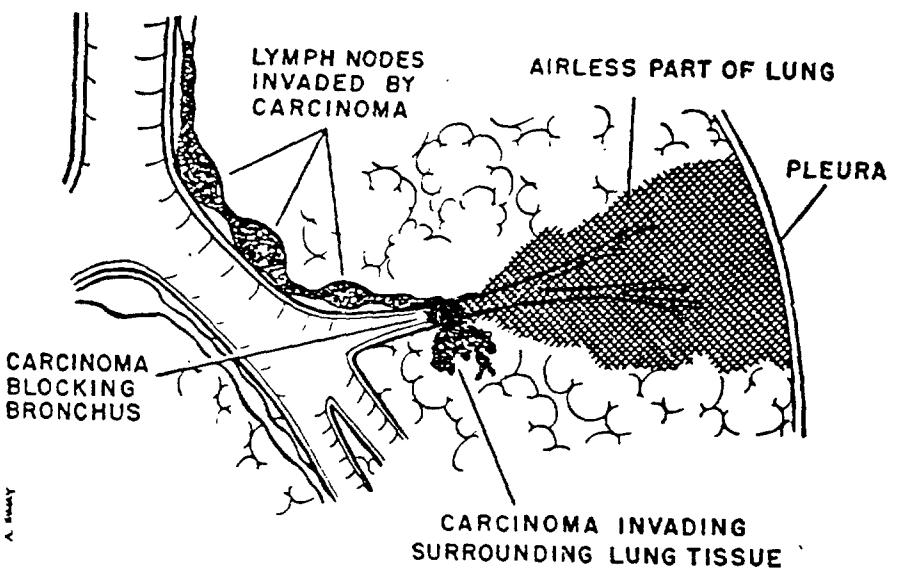


(DIAGRAMMATIC LONGITUDINAL SECTION OF BRONCHUS)

BRONCHIAL CARCINOMA



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OCCURRENCE

Considered a rare disease a generation ago, cancer of the lung has been increasing alarmingly in incidence. It is the most frequent type of fatal cancer among males. The sex ratio varies from place to place, lung cancer attacking from seven to eight males for every female. There is no known racial differential. The age distribution is from 35 to 75, with a lower rate on the mortality curve below 45 and a sharp rise after age 55.

Studies of the relation of cancer to air pollution and to smoking show a ratio of cigarette smokers to nonsmokers of more than twenty to one in rural areas and of three to one in such a city as Liverpool, England. Immigrants from industrial England to New Zealand with smoking habits presumably similar to the natives have a much higher incidence of lung cancer. Non-smoking Yemenite Jews in Israel have a much lower (1 to 6.5) incidence than do Jews from central Europe. There is also a direct statistical relationship between poverty and the incidence of lung cancer, suggesting that environmental factors other than cigarette smoking may contribute to the causation.

The social and economic implications of lung cancer are apparent. This disease produces a great loss of life among men still in their productive years. It causes extensive and severe suffering and a large total medical expense. This cost must be added to the cost of the tobacco habit and to the other costs of permitting air pollution in our cities.

MANIFESTATIONS

Cancer imitates most other pulmonary diseases and has no typical mode of onset. Often it masquerades as an acute pneumonitis that does not entirely clear up. Persistent cough or a change in a habitual cough is a frequent first symptom and is the most common one. Expectoration is absent at first, then varies with the amount of secondary infection and with bronchitis, but also with the amount of smoking and with the season.

Blood spitting, which occurs at some time in many cases, may be the first sign. Pain in the chest is such a regular symptom of cancer that persistent pain in any combination with other pulmonary symptoms and with an X-ray shadow strongly suggests cancer as the diagnosis. Progressive loss of weight is common. Wheezing may be an early complaint. "Arthritis" of the shoulder or of various other joints is not uncommonly an early symptom. Clubbing of the fingers may be present.

Such symptoms as loss of voice, continued chest pain with shortness of breath, and headaches with nausea are ominously linked with extensions of the disease beyond the curable stage.

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expiration. Aside from this relatively early clue, physical signs of lung cancer are those of pneumonia, atelectasis, or lung abscess resulting from the growth in the bronchus. The discovery of palpably enlarged, firm lymph nodes in the neck or armpit or of paralysis of a vocal cord or of the diaphragm on one side indicates presence of the disease. Signs of pleural effusion or of masses in other organs may be found.

LABORATORY FINDINGS

The chest X-ray film is in itself not diagnostic of primary cancer, a disease that takes many forms. A solitary nodule ("coin lesion") in the peripheral portion of the lung is the most typical early X-ray finding. Lobar collapse or pneumonia is a frequent appearance. A rounded mass adjacent to the hilum or in the apex is another. Sometimes, when the center of the tumor breaks down and is coughed out, the picture is that of lung abscess.

The opacity of pleural effusion may obscure tumor shadows in the lung. Bronchograms and planigrams may show the outline of a cancer obstructing or narrowing a large bronchus. Angiograms (X-ray films of the blood vessels) may show the circulatory changes characteristic of a malignant tumor, as may lung scans.

Bronchoscopy may reveal the tumor projecting directly into the bronchus, permitting removal of tissue for study, or it may show that the tumor has invaded and fixed the mediastinal structures. It may provide by aspiration a specimen of secretions from a peripheral tumor for study of the cells.

The most specific laboratory evidence is the demonstration of cancer cells in the sputum or bronchial secretions. Whether such cytologic studies are made by the Papanicolaou method or by some other, the examination of repeated smears by competent pathologists yields a high (up to 85 percent) positive correlation with operative findings and seldom a false positive report. Other findings, such as accelerated red-cell sedimentation and secondary anemia, are the rule but are not specific for cancer.

METHODS OF DETECTION

X-raying all who have a persistent cough (for more than four weeks) or who spit blood will yield a high incidence of advanced cancer. Cytologic smears of the sputum should be performed repeatedly on all those in the cancer age with symptoms, an X-ray film showing a new lesion, or both. Bronchoscopy without delay is indicated whenever lung cancer is suspected. Finally, the differential diagnosis between cancer and a benign tumor or granuloma cannot wait upon observation of the course of the disease or of the effects of a trial treatment. Exploratory chest surgery with direct biopsy and microscopic exami-

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nation of a frozen section must be undertaken even though many such operations may disclose nonmalignant lesions.

METHODS OF CONTROL

The treatment of lung cancer is still unsatisfactory, mainly because of delayed diagnosis. Since Dr. Evarts Graham performed the first successful cure in 1933 by removing one entire lung, surgical treatment has offered hope in this previously fatal condition. However, the percentage of all lung cancers attaining a five-year surgical cure has failed to show a steady increase in spite of all efforts at earlier diagnosis and treatment.

Radical pneumonectomy with dissection out of the mediastinal glands has also failed to improve the results. Lobectomy, with a lower operative mortality rate than pneumonectomy, has become the operation of choice because it is less disabling. Radiation therapy or chemotherapy will relieve symptoms and sometimes provide an interval of fair health for those with inoperable lung cancer, but "cure" by these methods is extremely rare. They may properly be considered as part of the palliative treatment of lung cancer. This also includes symptomatic medical care. Physicians and nurses who give these patients intelligent, undismayed, and kind treatment to the end perform an important duty.

PREVENTION

On the basis of present knowledge the only practical means of prevention are (1) not to smoke cigarettes and (2) not to live in an atmosphere of environmental or industrial pollution. Discontinuing the smoking of cigarettes is almost as good as never starting the habit. Certainly, heavy smokers should not live where air pollution adds to their risk. Measures should be taken to protect miners of radioactive ore and asbestos and chrome workers against occupational exposure, and particularly to help them discontinue their smoking habits.

APPROACHES TO THE PROBLEM

There is now an enormous worldwide effort to find the primary cause of malignant new growths. There is also a search for an effective treatment. Until a more basic preventive measure is found, the problem of cigarette smoking is central in the prevention of lung cancer. Education of the public, especially of young people, not to smoke must be pushed by precept and example. Studies of air pollution and its prevention may help, but certainly less than discontinuance of smoking.

The possibility of obtaining a somewhat better rate of cure by earlier diagnosis, improved surgical techniques or combinations of surgery, radiation, and chemotherapy has not yet been exhausted. The late appearance of symptoms

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and early invasion of the bloodstream in this form of cancer do not encourage great hope for a high cure rate once lung cancer is diagnosed.

Immunization against lung cancer and other forms may be developed. A synthetic chemical or antibiotic drug may be discovered capable of destroying cancer cells in early or late lesions. A simple, accurate test for the presence of cancer anywhere in the body may be found (although such a test may also cause great mental anguish if positive without a demonstrable lesion or if falsely positive). In the meantime, cigarette tobacco must be modified or filtered in such a way that the smoke is no longer carcinogenic, but better still abandoned completely.

ALVEOLAR CELL CARCINOMA

Alveolar cell carcinoma (also called pulmonary adenomatosis or bronchiolar carcinoma) is a malignant new growth believed to originate from the cells lining the alveoli or bronchioles, and progressing chiefly inside the lung to a fatal termination. It is much more rare than bronchogenic cancer. However, it is being recognized more frequently and is now found to constitute at least one percent of lung malignancies. It does not seem to have any geographic, racial, or sex differentials. Nor does it seem to bear a direct relationship to smoking or air pollution. It occurs chiefly in persons more than 40 years old.

PATHOLOGY

The microscopic picture is characteristic, consisting of alveoli lined with columnar epithelium containing large, heavily stained nuclei. Extension is by contiguity through the lung and possibly also by way of the bronchi, but slight or absent by way of the lymphatics and bloodstream.

MANIFESTATIONS

The onset is usually insidious, with gradually increasing cough. The cough may be productive of enormous amounts of sputum, a symptom peculiar to this condition, but one not invariably present. Blood spitting is unusual. Pain is not the rule, as it is with bronchial cancer. There is progressive loss of weight and increasing weakness accompanied by a low fever. The principal signs are similar to those characteristic of pneumonia. The clinical picture is often that of a chronic pulmonary infection rather than that of cancer.

LABORATORY FINDINGS

The chest X-ray film shows one or more poorly defined areas of consolida-

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tion without mediastinal gland enlargement, suggesting infection rather than new growth, but serial films show a steady progression. Cytologic studies of the sputum frequently may establish the diagnosis by demonstrating the characteristic columnar cells. The final diagnosis depends upon microscopic study of sections from the involved area in a lobe, removed by surgery.

PROGNOSIS

The prognosis is uniformly bad unless surgical removal of the diseased lobe is carried out early. Even some time after an apparently successful resection, new centers of alveolar cell cancer may develop.

METHODS OF DETECTION

Most cases of alveolar cell cancer are discovered by physicians familiar with the condition as a possible explanation for a puzzling, progressive disease. Persistent and increasing lung symptoms with no demonstrable cause, in the face of treatment, in a person in the cancer age, lead the physician to obtain chest films and cytologic studies of the sputum.

APPROACHES TO THE PROBLEM

This condition has not been recognized very long and is still under active study. The originally assumed relation to jaagziekte disease of sheep not having been proven, there is at present no good lead to its cause or control. Its similarity to a low-grade infection leads to some hope that further analysis of individual cases and the collection of epidemiologic material may disclose the cause.

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CHAPTER 6

PNEUMOCONIOSES

The term pneumoconiosis is a general one. It simply means "dust retained in the lungs" without implications of any reaction to the dust. While some dusts, notably silica and asbestos, cause a fibrous tissue reaction in the lungs, others are quite inert when they are retained in moderate concentrations.

With this broad definition, it is evident that everyone acquires some pneumoconiosis because varying amounts of particulate matter are retained in everyone's lungs. Only in the past few decades has the relative harmlessness of certain inert dust been recognized. For example, the coal smoke or soot pigmentation of the lungs of all city dwellers is by definition a pneumoconiosis, yet it is not usually recognized as such because this carbonaceous material is not sufficiently dense to cast shadows on X-ray films. If the retained carbon in our lungs were as dense as iron, radiologists would diagnose pneumoconiosis in most of us.

For this reason it is necessary to use specific terminology when speaking of various pneumoconioses due to occupational dust exposure. The two that most commonly may lead to impaired lung function are *silicosis* and *asbestosis*. They are entities about which there is general medical agreement. Less specific and more controversial is the so-called coal workers' pneumoconiosis, which will be discussed separately below. The so-called benign pneumoconioses, due to focal retention of inert dusts that have sufficient density to be seen on X-ray film as isolated shadows, also are discussed later.

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SILICOSIS

This pneumoconiosis is due to the occupational inhalation of dust containing crystalline free silica (SiO_2), resulting in fibrous nodules uniformly scattered in both lungs or in a more diffuse fibrosis, which may be progressive, with increasing impairment of lung function. Among the older names for silicosis and silicotuberculosis, all indicative of particular trades, are miner's phthisis, stonecutter's disease, potter's asthma, and grinder's rot.

Silicosis will occur in an industry in which workers are exposed over a long enough period of time to a sufficient concentration of dust containing free silica in crystalline form in particle sizes below five microns. All types of mining in which the ore is found in quartz rock have produced silicosis to an excessive degree, especially gold mines, copper mines, lead and zinc mines, iron mines, anthracite coal mines, and occasionally bituminous coal mines. Other industries in which silicosis has occurred in workers are foundries, sandstone grinding, sandblasting, concrete breaking, pottery and china manufacturing, and granite cutting and polishing.

In most of these industries in recent years preventive measures have markedly reduced the incidence of this completely preventable disease. In the foundry industry, for example, numerous plants have brought dust under such adequate control that silicosis is no longer a hazard. This may make it permissible for workers with minimal nodular silicosis to remain at their regular jobs.

CAUSE

From Roman times it was recognized that miners and workers in stone had a high prevalence of lung disease. The earliest theory of the cause of silicosis was that the hard, sharp particles cause mechanical injury to the lungs, but animal experimentation some years ago disproved this. The most commonly accepted theory is that the reaction to silica dust is physicochemical due to the solubility of free silica in the tissues. A new immunological antigen-antibody approach to explaining the disease is now being explored, but has not yet had wide acceptance.

The factors determining the development of silicosis are: (1) the percentage of free silica in the dust inhaled; (2) the concentration of dust at the breathing level on the job; (3) the size of the dust particles; (4) the admixture of other dusts; (5) the time, usually in years, spent in this atmosphere; and (6) individual differences in susceptibility. Silicosis occurs in direct proportion to the percentage and concentration of silica and to the duration of exposure. Generally it takes at least five years of exposure to concentrations of more than

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PNEUMOCONIOSIS

ROUTE OF DUST PARTICLES

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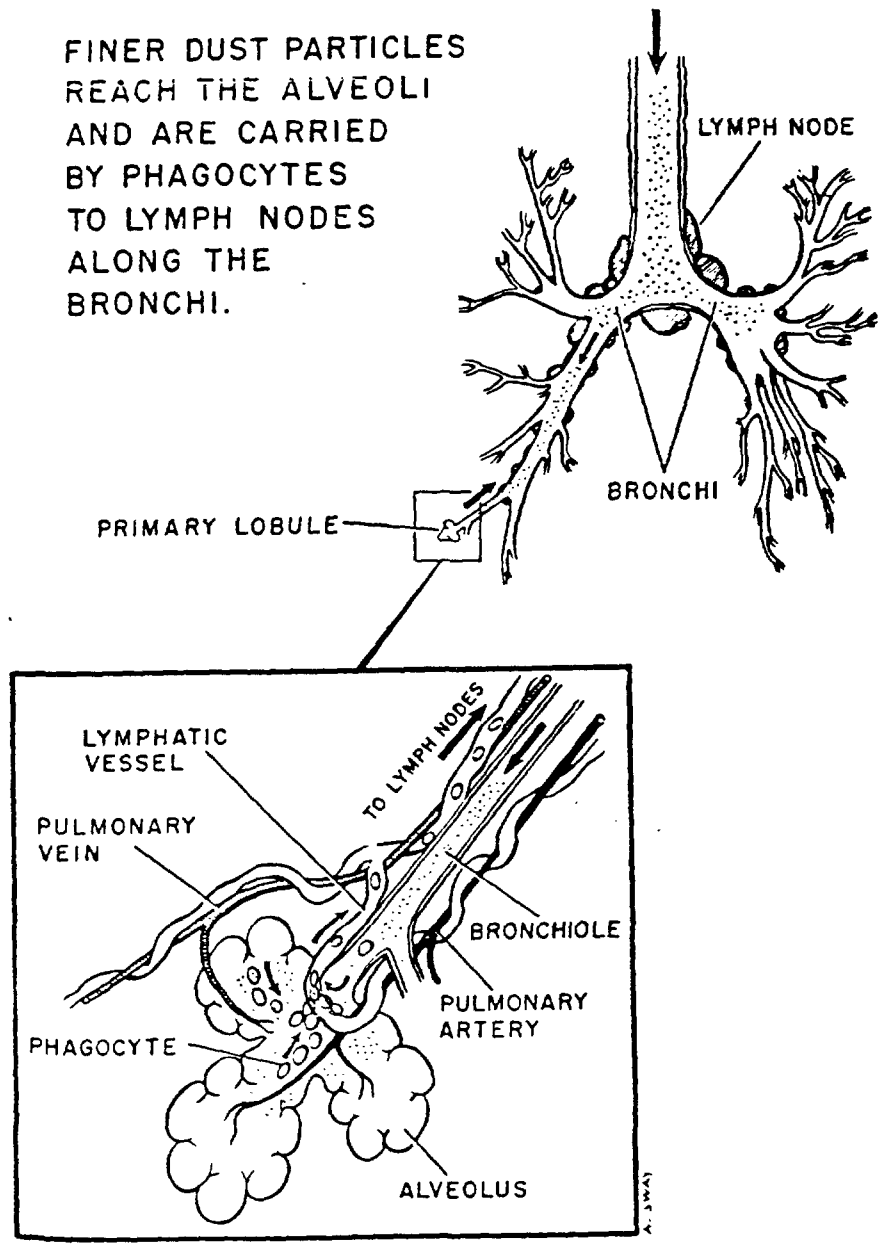

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five million particles per cubic foot of dust with at least 50 percent silica content to produce clinical silicosis.

All particles more than 10 microns in size are stopped by the protective mechanisms of the upper respiratory tract and bronchi. When the silica dust is accompanied by iron, aluminum or carbon dust, the development of silicosis usually is modified and the resulting fibrosis develops more slowly.

No two workers develop silicosis at the same rate. The differences may be due to mouth breathing in some people, inadequate filtration in the nasal passages in others, chronic bronchial infection, or to individual variations in tissue reaction. Silica particles that remain in the tissues long after the period of exposure to dust may continue to produce reaction and scarring of the lung, but usually for a few years only, unless tuberculous infection is associated with the silicosis.

PATHOGENESIS

Dust particles less than five microns in size are carried by inhaled air to the alveolar ducts and sometimes into the air sacs themselves. Only about half get as far as the alveoli. There, like any other particles, they are engulfed by phagocytes and carried by the lymph stream to the pulmonary lymph nodes. The lymphatics may become blocked with the dust-filled phagocytes that lodge in the lymph nodes at the junction points.

Silica, slowly dissolving in the phagocytes, causes an excessive proliferation of fibroblasts (scar tissue cells), which eventually results in the typical silicotic nodules. When silica dust has been excessive, individual silicotic nodules may become so large and numerous as to result in massive fibrosis. This, when extensive, may contract to cause overdistension of the surrounding lung parts.

PATHOLOGY

The changes in the structure of the lung are divided into three phases. In the initial phase, there are nodules of collagen (fibrous connective tissue), still microscopic in size along the lung lymphatics and in the hilar lymph nodes.

In the second phase, which is the earliest phase that can be diagnosed by X-ray, there are multiple, discrete nodules from two to five millimeters in diameter throughout the lungs. These often have sharply defined borders and consist uniformly of collagen fibers arranged either concentrically or in basket-weave patterns. With rapidly evolving silicosis caused by ultra-fine particles, all of these nodules may have necrotic centers.

The third phase is that of massive conglomerate fibrosis. Viewed at autopsy, the lungs show extensive scarring of the upper and central portions, often

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bilaterally symmetrical. All normal lung structures are replaced in these areas and there may be irregular cavities due to lack of blood supply. Peripherally the lung is overdistended, with many bullae on its surface separated by dark retracted scars. Microscopically the large areas of fibrosis consist of massed nodules in scar tissue.

NATURAL HISTORY

The onset of silicosis is gradual and not recognized. Often it is years before a diagnosis can be made. One man may work a lifetime as a miner or foundry worker, suffer little or no disability with moderately advanced silicosis, and finally die of old age. Another person, exposed to high concentration of very fine dust with a high silica content, may become disabled within a year or two after first exposure and die within another year; but this is rare.

The course of the disease is modified by the fact that few workers are constantly and uniformly exposed to silica over a long time. They change jobs or protective measures are taken in the plant or mine. Infections, most notably tuberculosis, often change the course of the disease. *Tuberculosilicosis* is a term used for conglomerate silicosis in which previous infection with tuberculosis plays a part.

COMPLICATIONS

Pulmonary tuberculosis is a possible complication of silicosis. The cellular toxicity of silica tends to stimulate multiplication of tubercle bacilli. Fibrosis may retard the extension of the disease and prevent symptoms due to absorption of tuberculoproteins.

Although pneumonia seems no more frequent in silicotics than in the general population, its prognosis is not as good in silicotics. Chronic bronchitis occurs in a high proportion of advanced silicotics but generally not with simple nodular silicosis. Emphysema is a fairly frequent end stage of those with advanced silicosis. The occurrence of heart failure with cor pulmonale seems to depend more upon the degree of emphysema than on the silicosis itself. Spontaneous pneumothorax may occur when the silicosis is accompanied by subpleural blebs.

PROGNOSIS

With simple nodular silicosis acquired over a period of 15 years or more, the prognosis is good, provided that further silica dust exposure is reduced to a safe level or is stopped altogether and also provided that tuberculosis is prevented. The ultimate prognosis with conglomerate silicosis is not good, but

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often the patient can continue to work for a long time at his trade. When silicotuberculosis is active, with positive sputum, chemotherapy must be started promptly.

MANIFESTATIONS

Simple nodular silicosis causes no symptoms until the disease is advanced. Then shortness of breath on exertion may long precede other symptoms. Cough and wheezing appear next. Expectoration of dark grey or black sputum occurs with respiratory infections. When the third stage develops, there may be chest pains. Hemoptysis, or blood spitting, usually means a complicating tuberculosis. Fever and loss of weight do not occur with silicosis alone.

There are no particular signs of silicosis in the beginning, and the appearance of good health remains. In the late stages, there may be some blueness of the lips and ear lobes and some clubbing of the fingertips. The chest may become characteristically barrel-shaped when accompanied by emphysema. Surprisingly few abnormal physical signs are heard through the doctor's stethoscope over lungs that are full of silicotic nodules or conglomerate masses, unless there is a complicating bronchitis.

LABORATORY FINDINGS

The chest X-ray film affords the basis for diagnosis. Early silicosis can best be detected if there is a high degree of contrast in the chest films. The nodules of simple silicosis are first revealed on the X-ray film when they are about two millimeters in diameter. In later stages, the larger nodules throughout both lungs and the conglomerate masses are striking. The patterns of silicosis as seen on the films are sometimes characteristic of different trades and types of exposure.

Although there is some correlation between the degree of shortness of breath and the extent of X-ray shadows, in many instances people with extensive infiltrations have little disability. Others whose chest films show relatively few shadows may have difficulty in breathing because of associated emphysema. The diagnosis cannot be made on the basis of silica in the sputum because those who have been exposed to it will have it in the sputum for years whether diseased or well. Examination of the sputum for tubercle bacilli, however, is necessary to rule out tuberculosis with conglomerate silicosis.

Theoretically, the degree of disability can be determined in the pulmonary physiology laboratory by testing the maximum breathing capacity or the maximal expiratory flow rate of the patient. But frequently the resulting figures are not valid because the full cooperation of the patient is not obtainable. More objective ventilatory tests of lung function are urgently needed.

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METHODS OF DETECTION

A comprehensive occupational history, carefully taken, is the first step to the diagnosis of all pneumoconioses. Chest X-ray surveys are the best method of mass detection but photofluorograms may not show early silicosis. For those in dusty occupations, chest films should be scheduled at least every year or two.

METHODS OF CONTROL

Silicosis is a permanent condition, irreversible by any treatment. The daily inhalation of aluminum powder by exposed individuals has been tried without much success. Antimicrobial treatment of intercurrent infections and supplementary treatment with bronchodilator drugs and intermittent positive pressure breathing (IPPB) apparatus may be helpful in some cases.

PREVENTION

Men discovered to have silicosis in its early stages should be protected against further harmful exposure. A change of job may be indicated for a young man with relatively short exposure and X-ray evidence of early silicosis. Older men, and those with advanced silicosis who have only a few more years before being eligible for retirement benefits, may well continue on the job. Dust concentrations should be reduced below the danger level by provision of exhaust ventilation at the point where dust is produced, by air piped into a closed hood over the worker's head, or by changing from a dry to a wet operation. Or a change in techniques may be needed, such as the use in a foundry of a parting compound that does not contain free silica.

Each industry and each job must be studied on the spot to determine the best means of protection. Many workers will not use masks on the job because they are either too hard to breathe through or are poorly fitted and uncomfortable. The general reduction in tuberculous infection is reducing the frequency of silicotuberculosis.

APPROACHES TO THE PROBLEM

State and federal authorities, including the National Institute of Occupational Safety and Health and the occupational safety division in the Department of Labor, as well as compensation insurance companies, are actively engaged in studying the conditions under which silicosis occurs and in setting up protective measures. Samples of air are taken at face level in the place of employment and analyzed for total dust count particle size, and silica content. Job applicants are required to have pulmonary function tests and to fill out health questionnaires before employment in dusty trades and at intervals thereafter.

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New industries are watched for possible new dust hazards. Industry itself has found it necessary to protect its workers from developing silicosis of any degree and to give them medical supervision.

The individual factors by which one worker is more prone than another to develop silicosis need further study. Better methods to detect the earliest stages of silicosis are needed as well as more objective tests of pulmonary disability. The possibilities of modifying silicosis by the concomitant inhalation of other substances or of discovering some effective treatment have not been exhausted.

COAL WORKERS' PNEUMOCONIOSIS

Coal workers' pneumoconiosis (CWP) is the accumulation of coal dust in the lungs and the body's reaction to this dust. "Black lung" and "miner's asthma" can refer to CWP but are also sometimes stretched to include any chronic respiratory symptom or disease that may afflict a miner.

For many years it was assumed that the silica often present at the mine face was the only substance harmful to miner's lungs. Coal dust was dismissed as a pathogenic agent. Only recently has CWP been accepted as an entity distinct from silicosis, although a coal worker may have both.

PATHOGENESIS

Particles of coal dust five microns or less in diameter inhaled into the lung eventually build up into macules (spots), which may be seen on X-ray. Macrophages surround the dust particles and wisps of fibrous tissue appear. Often, focal emphysema or abnormal dilatation of small airways or bronchioles occurs. CWP develops very slowly, over years of exposure. Continued exposure is related to advancing degrees of simple pneumoconiosis.

With simple CWP that is advancing, increasing amounts of fibrotic tissue may join the macules together into an ever denser and larger mass or masses, usually in the upper lung. The surrounding lung tissue may become shrunken and distorted. This mass formation is known as progressive massive fibrosis (PMF). When PMF develops, the exchange of gases between the lung and blood is hampered and cor pulmonale can appear. In severe cases, the lung's blood vessels can be largely destroyed and heart failure occur.

MANIFESTATIONS

Although dyspnea, coughing, and sputum may be present among workers with

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X-ray signs of simple CWP, no correlation has been shown between these symptoms and the radiographic CWP categories. Only in classification 3 (see below) has a physiologic correlation been demonstrated—and then the impairment of ventilatory function is slight.

Once PMF develops, however, symptoms do appear. Dyspnea is most common, usually accompanied by coughing. Sometimes there are chest pains. Gas exchange is increasingly impaired. Both symptoms and X-ray evidence tend to increase with the number of years of underground coal mine work.

LABORATORY FINDINGS

The diagnosis of CWP is by X-ray evidence, according to standards established by the International Labor Organization (ILO). According to this classification, CWP is diagnosed as follows: Simple CWP, stages 1, 2, and 3 designate from very few and small to ever larger and more profuse, but still disconnected, macules. Complicated or advanced CWP, otherwise known as PFM, stages A, B, and C designate from small to large masses of dense fibrous tissue.

METHODS OF CONTROL

Preventive measures can probably effectively control CWP. On an individual basis, removal of the worker from a dusty job before the disease progresses beyond stage 2 seems to prevent its further development. Although cigarette smoking seems unrelated to CWP, symptoms of CWP may be aggravated by smoking. On a larger scale, reduction of the coal dust to amounts too small to affect the miner and periodic chest X-rays with transfer to a less dusty job when indicated should effectively control CWP.

ASBESTOSIS

Asbestosis is a diffuse fibrosis of the lungs quite unlike nodular silicosis and due to many years of inhalation of asbestos fibers (magnesium silicate). Asbestos is used to insulate against heat and cold. Exposure may occur in plants making asbestos products, and in installation of insulating materials.

PATHOGENESIS

The longer fibers, from 10 to 50 microns in length, and not the shorter ones, appear to cause the characteristic pathological changes. They become lodged in the bronchioles where they set up a chronic irritation. Too large to be picked up by phagocytes, they become surrounded by a colloidal membrane to produce the characteristic "asbestos bodies" or they worm their way into the peribronchiolar spaces where they cause fibrosis and thickening of alveolar walls. As a result of the latter, there is a relatively early interference

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with the exchange of oxygen and carbon dioxide across the alveolar membrane. This diffusion difficulty usually occurs before there is any real ventilatory impairment.

While asbestos is chemically a silicate, it is the fibrous nature of the material rather than its chemical nature that is responsible for the characteristic pathology. The same pathological changes occur with talc, which contains tremolite (also magnesium silicate) fibers. Other silicates such as mica, feldspar, and soapstone generally cause little lung disease unless they contain quartz or other crystalline silica in a free state.

LABORATORY FINDINGS

The diagnosis of early asbestosis by X-ray is seldom conclusive because there are no nodular shadows as with silicosis. The earliest X-ray evidence is increased prominence of the finer lung markings, often referred to as "groundglass appearance," which sometimes is also seen in persons who have not had any dust exposure. By the time minimal changes due to asbestosis become apparent on X-ray film, there already may be some diffuse fibrosis and thickened alveolar walls in the lungs.

The diagnosis of such cases often must depend on history plus lung function studies or lung biopsy. With more advanced cases, there frequently is thickening of the pleura and pericardium, with increasing diffuse fibrosis and emphysema.

OCCURRENCE

Occupations that expose workers to fine asbestos fibers are the mining, crushing, and grinding of asbestos. Asbestos textile mills may have considerable fiber exposure. The processing and handling of insulating materials containing asbestos also constitute a hazard when there is inadequate dust control.

PROGNOSIS

There is no inherent predisposition to tuberculosis as there is with silicosis. Lung cancer of the bronchogenic type, however, especially cancer of the *mesothelium* (certain lining surfaces, such as that of the pleura), seems to be somewhat more prevalent with asbestosis than in the general population. As uncomplicated asbestosis progresses, shortness of breath increases and cyanosis may develop, due to decreasing oxygen saturation of the arterial blood. Right side heart failure (cor pulmonale) may cause death in advanced cases.

BERYLLIOSIS

Occupational inhalation of beryllium dust may result in chronic granuloma-

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tous disease in the lungs and elsewhere in some persons so exposed. Beryllium is a metal that is inert, except in individuals who have become sensitized to it. The onset sometimes is acute, with chills and fever, and marked shortness of breath due to edema of the alveolar walls. The X-ray appearance at first may be one of generalized haziness or an irregular fine nodulation. There may be years between the last exposure and onset of symptoms.

Industries that have been involved with this condition are the beryllium extraction plants where beryllium compounds are refined from beryl ore and plants making use of beryllium oxide in the manufacture of numerous materials and alloys. The earliest cases found about 30 years ago were in plants making fluorescent lamps in which the phosphorescent coating was zinc beryllium silicate. By 1949, a substitute coating material had been found and this source of the disease was eliminated. Beryllium extraction plants still have occasional cases but rigid dust control has much reduced the incidence of new cases.

The so-called "neighborhood cases" among persons living near plants found before 1950 have disappeared because beryllium oxide no longer is allowed to escape from the stacks. The acute pneumonitis and the dermatitis cases due to the acid compounds, beryllium sulfate, and beryllium fluoride have largely disappeared due to the better control measures in the extraction plants. Beryl ore itself never has been a skin or lung problem.

Beryllium is having a marked increase in use as an alloy with various other metals. With all of these newer uses, elaborate dust control methods have been worked out that are successfully preventing the disease.

The clinical picture of chronic, disabling berylliosis, however, should be recognized even though this condition is becoming increasingly rare. The main symptom is marked shortness of breath, at first only with exertion but later even at rest. With this there may be mild to marked cyanosis of the lips and nailbeds due to insufficient oxygen in the arterial blood. Treatment with cortisone or one of its derivatives will relieve the shortness of breath and cyanosis, but it must be given fairly early in the disease before much fibrosis of the alveolar walls develops. It must be continued on a maintenance basis, too, almost indefinitely, to prevent the return of the symptoms.

Chronic cases discovered 10 to 15 years ago are being maintained on an able-to-work basis with such treatment. Others have developed progressive fibrosis and emphysema in spite of the treatment when the cortisone was started after there was much irreversible fibrosis of the alveolar walls.

OTHER PNEUMOCONIOSES

Some occupational pneumoconioses due to vegetable (organic) dusts are:

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1. *Byssinosis*, a respiratory disease found among cotton mill workers. Its manifestation of periodic dyspnea, plus tightness of the chest and cough, is believed to be due to a pharmacologic agent in cotton fibers, called bract, that is borne in the air in cotton dust. This eventually may lead to chronic bronchial irritation and emphysema, with impaired lung function. Byssinosis cannot be diagnosed by X-ray.

2. *Bagassosis*, a lung disease found among workers who have had exposure to dust from sugar cane fibers that have been baled and stored for months. The reaction in the lungs is a granulomatous one somewhat similar to beryllium disease and may be a sensitivity response to moldy dusts.

3. ***Farmer's Lung***, thought to be a sensitivity response to fungal spores in moldy hay, or to a chemical change in the hay itself, quite similar to bagassosis. No definite fungus has been identified, however. *Silo-filler's disease* is not a pneumoconiosis but a chemical pneumonitis due to nitrous gases.

4. *Maple Bark Disease*, first described by Towey in 1928, also is believed to be a sensitivity reaction to fungal spores.

Some benign pneumoconioses are due to the retention in the lungs of inert dusts that do not cause fibrosis, impaired lung function or increased susceptibility to tuberculosis, yet can be visualized on X-ray films as nodulation when the dust is sufficiently dense. Dusts of low atomic weight, such as carbon (coal or soot), aluminum, limestone, marble, and gypsum dusts, may cause deposits in the lungs that are not seen on chest films.

However, where the atomic weight of the dust is higher, such as iron ore dust or iron oxide fumes, retention in focal areas at junction points of blood vessels may produce fine nodular shadows. On X-ray film these look like nodular silicosis. *Siderosis* is the proper term for this benign pneumoconiosis. Welders of iron or steel in confined areas and flame cutters or grinders of iron and steel may develop iron retention. With such an X-ray pattern, careful differential diagnosis is needed to avoid a misdiagnosis of silicosis. Those with simple iron retention may stay on their jobs with safety.

Other benign pneumoconioses that can be seen on chest films are *stannosis* (tin) and *baritosis* (barium). Because of the high atomic weights of these materials, their focal nodular collections cast very dense shadows quite dramatic in appearance on X-ray film. Yet there are no resulting symptoms and no impaired lung function, as shown by function tests.

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CHAPTER 7

PULMONARY MANIFESTATIONS OF GENERALIZED DISEASE

Many of the pulmonary diseases already considered are, like pulmonary tuberculosis, the chief manifestation of a generalized disease. Other lung conditions are medically important as part of generalized diseases. Some of these, such as bronchopneumonia with chickenpox and metastasis of cancer to the lungs, have been mentioned under other headings. Others, such as syphilis of the lungs or rheumatic fever pneumonitis, are omitted because they are very rare or because they are overshadowed by the importance of the general condition.

Several of the more important diseases with pulmonary manifestations will be described here. Sarcoidosis not only is common but was often mistaken for pulmonary tuberculosis in chest X-ray surveys. Cystic fibrosis and embolism of the lung are being increasingly observed at their respective extremes of life. (Cystic fibrosis is covered in Chapter 8, Lung Diseases of Childhood.)

SARCOIDOSIS

Sarcoidosis is a chronic systemic disease or group of diseases due to unknown causes, characterized by granulomatous masses of epithelioid cell tubercles. Lungs, lymph nodes, eyes, skin, liver and spleen are most often involved, but any tissue or organ may be affected. This disease was first observed in various

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parts of the body and much later assembled as an entity. Therefore, it has a large number of other names—lupus pernio of Besnier, Boeck's sarcoid, lymphogranulomatosis of Schaumann, Heerfordt's disease or uveoparotid fever, Mikulicz's syndrome, Jungling's disease, Besnier-Boeck-Schaumann-Heerfordt disease.

CAUSE

For many years, sarcoidosis was considered a form of tuberculosis. However, failure to demonstrate tubercle bacilli in the lesions has finally converted almost all of those who were proponents of the theory that sarcoidosis is a non-caseating tuberculosis to the conviction that the disease is nontuberculous. No other viral, bacteriologic, or fungal causative organism has been isolated.

The peculiar geographic distribution of most, but not all, cases has led to theories that sarcoidosis may be due to the inhalation of pine pollen or dust from peanut hulls, but these relationships have been disproven. The strong similarity to chronic berylliosis has suggested that beryllium or some other trace element in the soil may be involved.

The clinical syndrome of sarcoidosis may be several different but similar diseases, but more likely it is the result of different agents acting upon the tissues of individuals with a peculiar reactivity to irritation and an abnormal immune response.

PATHOLOGY

The microscopic study of sarcoid tissue is the basis for recognition of sarcoidosis as a disease. It resembles the tubercle of tuberculosis without central caseation, necrosis (tissue death) or evidence of surrounding allergic reaction. There are "cold tubercles," that is, discrete, rounded, concentrically arranged masses of epithelioid cells around giant cells that sometimes contain peculiar star-shaped or round inclusion bodies. Healing is by scar formation without cavitation. If there is any nonliving tissue in the center of lesions, it is hyaline (clear, glassy) rather than caseous (cheese-like). Healing may proceed slowly to a point at which microscopic study of this tissue shows only scars without sign of any of the original sarcoid granulomas.

NATURAL HISTORY

Sarcoidosis commonly starts with the appearance of skin lesions, enlarged lymph glands, or a miliary seeding of the lungs. Such subacute manifestations proceed slowly to healing. Remissions may occur during pregnancy. A minority of cases may proceed by further exacerbations to death from lung insufficiency or heart failure.

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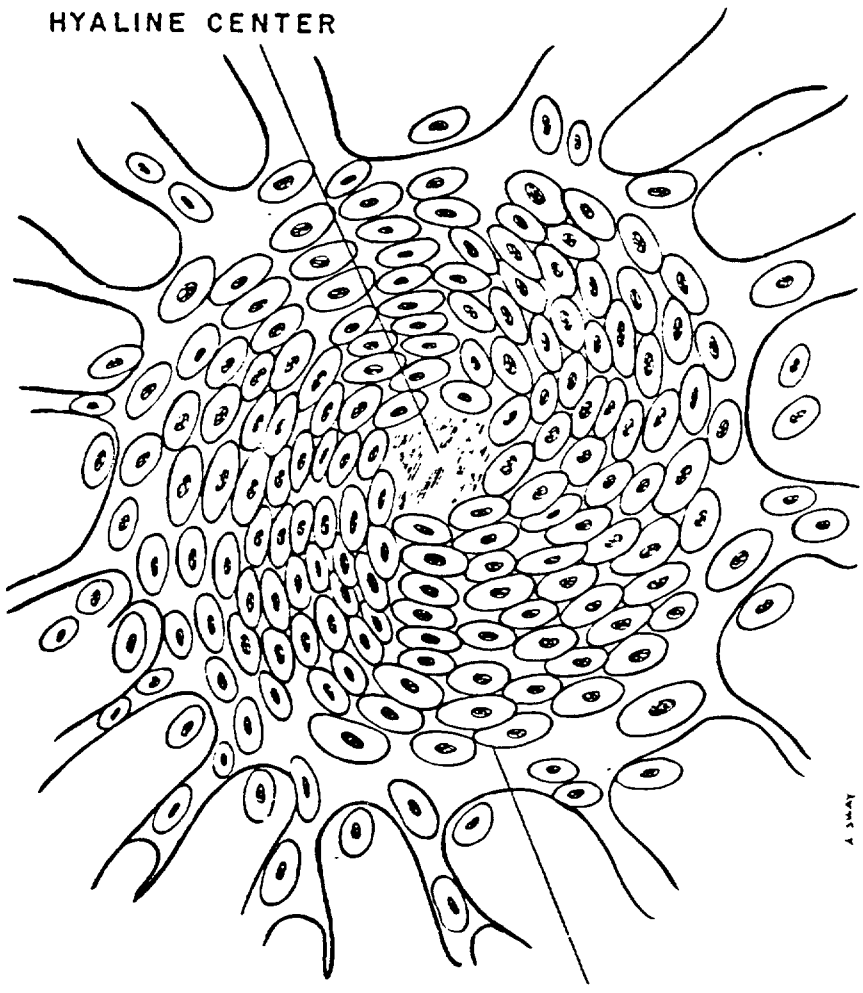

TUBERCLE FORMATION IN SARCOID
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In the past, tuberculosis developed as a complication in 10 to 15 percent of such cases. There is considerable controversy as to whether this TB is to be considered primary or secondary in the disease process. Pleurisy, hemoptysis, and spontaneous pneumothorax are rare. The involvement of heart muscle and of other organs cannot be considered as a complication: it is a regular part of the disease. Healed or "burned out" sarcoidosis may leave extensive pulmonary fibrosis, bronchiectasis, and emphysema.

OCCURRENCE

Sarcoidosis occurs at all ages and in both sexes but is most common between the ages of 20 and 40 in women and in those raised in rural areas. In this country, from eight to 15 black people are involved to one white person. The geographic concentration is along the Piedmont area of southeastern United States, where the attack rate is about 2 per 100,000 population, and in Norway and Sweden in Europe.

The social and economic implications of sarcoidosis are not apparent in its early and active stages because it is not contagious or disabling. Even with extensive involvement, patients usually continue their work. In the end stages, lung insufficiency or heart failure are chronically disabling and may represent a large total cost as well as prolonged suffering.

MANIFESTATIONS

Until sarcoidosis becomes advanced or complicated there are surprisingly few symptoms. At onset there may be fatigability that gradually increases, with loss of weight and vague pains. There may be low-grade fever. Often a mild cough with clear, scanty sputum develops with lung involvement. Later, with more extensive lung infiltration or scarring, shortness of breath becomes the principal symptom.

Old cases with lung scarring and distortion may present the clinical picture of chronic bronchitis or of emphysema. Signs of sarcoidosis found on examination include generalized, uninflamed lymph node enlargement, enlargement of the parotid and lachrymal glands, skin lesions (especially on the shins), and enlargement of the liver and spleen. The lungs usually show no abnormal signs even when grossly involved.

LABORATORY FINDINGS

These findings are useful in differential diagnosis. Extensive changes seen in the chest X-ray, with few or no symptoms, are suggestive of sarcoidosis. Four typical radiologic patterns are: (1) bilaterally enlarged mediastinal lymph nodes; (2) fine uniform seeding like miliary tuberculosis; (3) fan-like infiltra-

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tions, with or without enlarged nodes; and (4) soft, patchy shadows throughout both lungs. X-rays of the fingers and toes may show characteristic punched-out areas in the bones.

Examination of the blood serum shows increased globulin. There may be an increase of calcium in the blood and often an eosinophilia. Physiologic tests show an abnormal oxygen and carbon dioxide diffusion capacity and later decreased lung capacity due to scarring. The tuberculin test is negative in from 65 to 80 percent of cases.

The Kveim test, done by the implantation of sarcoid material into the skin, may show a nodule after weeks or months. This should be biopsied; it will show the microscopic picture of sarcoidosis in up to 80 percent of cases. But the test may give false positives and is therefore not believed to be entirely specific for sarcoidosis. It is not very useful as compared with an immediate tissue biopsy.

The diagnosis ultimately depends upon the microscopic demonstration of the characteristic pathologic pattern in association with a characteristic X-ray pattern and absence of other disease. This is made possible by a minor operation to secure a lymph node, skin lesion, liver, bone marrow, or lung specimen.

METHODS OF DETECTION

Because the lungs are involved in 80 percent of cases of sarcoidosis, routine chest X-rays or mass surveys discover most of them. Tuberculin testing surveys will miss most of them. Various specialists—the ophthalmologist, the dermatologist, the hematologist, the internist, and sometimes the abdominal surgeon—encounter sarcoidosis in their own spheres. The resemblance of sarcoidosis to tuberculosis without fever and with a negative tuberculin test should lead to the obtaining of a biopsy and thereby to a diagnosis.

METHODS OF CONTROL

There is no specific treatment for sarcoidosis. It runs its course whether treated or not. The corticosteroids can alleviate symptoms and produce regression of lesions. Administration must be prolonged and flare-ups may follow its cessation. Cortisone or one of its improved derivatives is indicated when there is lung involvement sufficient to produce dyspnea or cyanosis or when vision is threatened. Danger of lighting up a latent tuberculous infection in a patient with sarcoid being treated by steroids can be avoided by also giving preventive treatment with isoniazid daily.

PREVENTION

Sarcoidosis cannot be prevented without knowledge of its causes. Even

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secondary prevention of progression or complications arising from its early stage is limited to the prevention of tuberculosis by the use of isoniazid or of eye or kidney damage by the use of corticosteroid therapy.

APPROACHES TO THE PROBLEM

The search for a viral or bacterial causative agent has so far been unsuccessful. Recent studies have been based upon the peculiar epidemiology of sarcoidosis. Further analysis of these data and a follow-up of the leads uncovered, such as vegetable dusts or pollens and soil character, may be productive of more knowledge as to causes. There is also valuable information being accumulated by the longitudinal study of sarcoid cases over the years as to the natural history of the disease and its prognosis. As new methods of immunologic, biochemical, and other forms of scientific research are developed, many of them can be applied to these known cases under prolonged observation.

Possible future investigation of sarcoidosis may produce a better understanding of the special granulomatous defense reaction of the tissue in these patients. This subject leads into the area of intracellular immunity, so important also in tuberculosis, and into the nature of the tuberculo-allergy reaction and immunity. More immediately, the discovery of the cause or causes of this disease may lead to simple, positive measures capable of preventing it.

LUNG EMBOLISM AND INFARCTION

Embolism (from the Greek "to throw in") is the plugging of an artery by matter foreign to the bloodstream, that is, an *embolus.* (A *thrombus* is a blood clot that is stationary; an embolus, which may be a blood clot or other matter, moves through the blood vessels.) *Infarction* (from the Latin "to stuff into") is the process of tissue damage and death resulting from the cutting off of the blood supply to a region of the body. Either an embolism or a *thrombosis* (local clotting) in the arteries can produce infarction.

CAUSE

The lung is the organ most subject to embolism because all of the blood from every part of the body must come to it on each circuit. But the lung, having a blood supply through both the pulmonary artery and the bronchial arteries, is not particularly vulnerable to infarction, unless previously damaged by pulmonary or cardiac disease.

Thrombosis in the lung arteries is not uncommon. The commonest source of emboli to the lung is the heart: clots forming on the wall of the right auricle become detached and go into lungs that are already congested. The next most



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EMBOLISM AND INFARCTION

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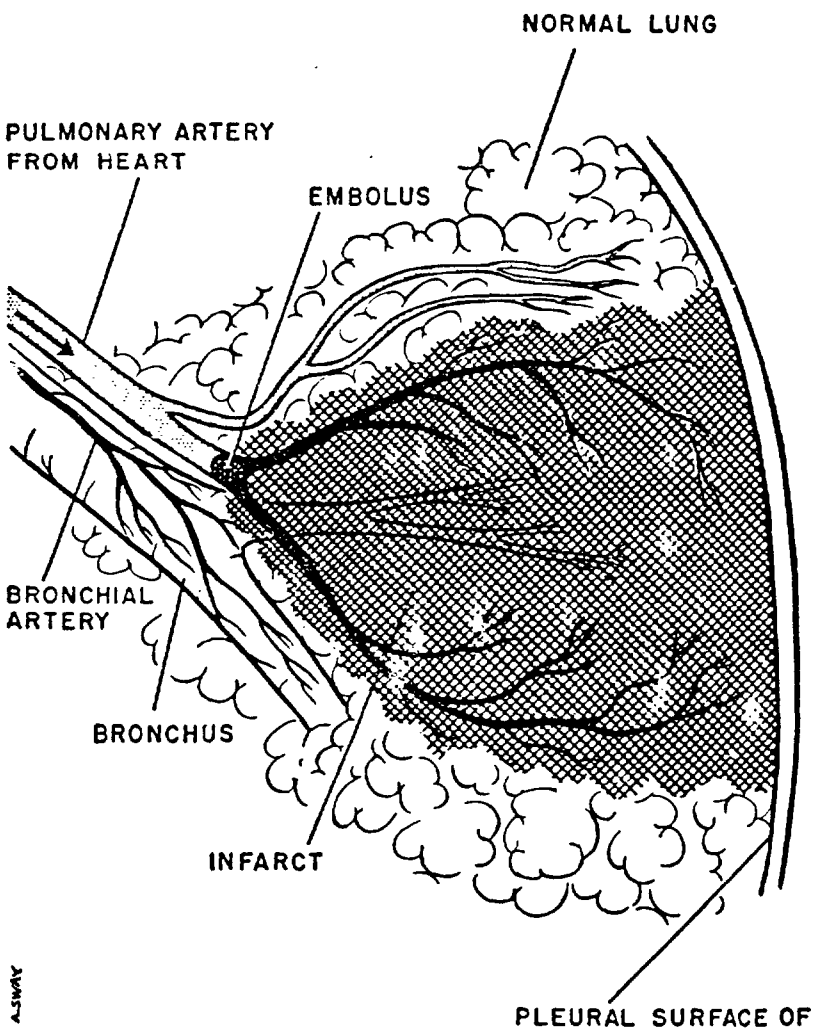

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common source are the veins of the lower extremities. Thrombosis may occur there after an operation, after a prolonged stay in bed, or following an injury. Embolism may then occur with a clot broken off from the thrombus that is large enough to block the whole circulation to the lungs, causing sudden death.

Clots from the upper extremities seldom cause lung embolism. The thrombophlebitis known as "milk leg" (phlebitis means inflammation of a vein), which may occur following normal obstetrical delivery, and clots in varicose veins also seldom cause embolism. Infected pelvic veins after poorly performed abortion may become the source of repeated showers of emboli that produce infarcts with multiple abscess formation in the lungs.

Lung embolism can also be caused by any other material that gets into the venous circulation. Fat droplets from the bone marrow also produce severe embolism in cases of extensive fractures. Air or any other substance injected for diagnosis or treatment can do the same.

PATHOLOGY

In cases of sudden death due to massive lung embolism, the pathology found at autopsy may consist entirely of a large clot obstructing one or both pulmonary arteries. Pulmonary infarcts are firm, red, wedge-shaped areas extending out from the point of complete artery obstruction to the surface of the lobe. The pleura over them is covered with fibrin. Microscopically the air spaces are seen to be filled with red blood cells and coagulated serum.

If the blockage is extensive and the collateral circulation is inadequate, there may be dry necrosis (tissue death) of the whole center of an infarct. Otherwise, the area becomes invaded by leukocytes (white blood cells) that clear up the exudate cells and restore normal function in the course of several weeks, very much as in pneumonia. Extensive thrombosis of the pulmonary arteries may occur without infarction as the intact bronchial arteries supply the tissues.

NATURAL HISTORY

The history of a lung embolism depends upon the source, size, and nature of the embolus. A small percentage of all major surgical operations are followed by the sudden death of the patient from embolism, perhaps just as he is ready to go home from the hospital. These are usually from an unrecognized leg vein thrombosis. More commonly, a patient under treatment for congestive heart failure or some other medical condition undergoes a sudden change for the worse that may imitate pneumonia or a coronary occlusion, but that proves to be a lung embolism. If not fatal or repeated, there is a gradual recovery.

PROGNOSIS

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embolism has been mild, because the source of the clots may set free more of them to lodge in the lungs. Lung embolism accounts for at least 2.5 percent of deaths among hospital patients and 5 to 6 percent of postoperative deaths. The infected multiple type of embolism arising from pelvic veins has a high fatality rate.

COMPLICATIONS

The principal complication is the regular occurrence of fibrinous pleurisy and the formation of pleural fluid or pus. Aside from leaving some pleural thickening, lung infarcts create surprisingly few residual defects.

OCCURRENCE

Lung embolism is far more frequent than is usually realized. Unfortunately, the incidence seems to be rising. Some of this increased incidence may be due to better diagnosis.

Lung embolism is more frequent in women than in men and in both sexes over age 45. One-third of lung embolisms accompany heart disease, with a 50 percent case fatality rate. One-fifth are postoperative, with a 5 percent case fatality rate. The others follow trauma, infections, pregnancies, hemiplegia (one-side paralysis, usually from stroke), cancer, and varicose veins, in that order of frequency. It is, therefore, predominantly a disease of older people and a complication of other chronic conditions, hence social and economic effects are usually not separable. The sudden death of a young parent after surgical operation or delivery is, of course, both a family tragedy and an economic catastrophe. The prolongation of hospital stay by older patients due to embolisms adds to the total cost of medical care already so heavy for this segment of the population.

MANIFESTATIONS

The onset of the massive fatal embolism is also its end. The victim falls unconscious and dies quickly. In the postoperative or bedridden patient, the onset of lung infarction is with a sharp chest pain, cough, and expectoration of blood-tinged sputum. There is some degree of shock, as evidenced by pallor, sweating, and rapid pulse with low blood pressure. Shortness of breath, chest pain, and cough continue. Fever is not so high as with a corresponding lung involvement with pneumonia.

The signs are often indistinguishable from those of pneumonia—that is, consolidation of lung tissue and pleurisy or fluid. There may be cyanosis from the inadequate oxygenation of the blood and, later, jaundice from the reabsorption of large amounts of blood pigment from the infarct. Clinically, pulmonary

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infarction has been called "the great deceiver" because it is so difficult to differentiate from other heart or lung conditions.

LABORATORY FINDINGS

Good chest X-ray films are difficult to obtain when most needed because of the patient's serious condition. Lateral or oblique films are desirable to demonstrate the infarct with its base against the lung surface or a fissure. The lung lesion is often obscured by fluid in the pleural space. The red-cell sedimentation rate is elevated and there is a mild increase in white blood cells. Examination of the blood serum may show an increase in the enzyme transaminase.

METHODS OF DETECTION

Lung embolism must be suspected to be detected. In the course of certain other well-known diseases, any sudden worsening of the patient's general condition, with accelerated pulse and chest symptoms, should lead to physical examination, which should include the extremities, to chest X-ray examination, and to laboratory tests that will detect the presence of lung embolism as a complication.

METHODS OF CONTROL

The treatment of lung infarction is both surgical and medical. Under exceptional conditions in which a hospital has a system of immediate mobilization of operating room, anesthetist, and full thoracic surgery team, it has been possible to perform the heroic operation of removing the clot or clots from the pulmonary artery to save life. This, naturally, is a rare procedure.

Open cardiac surgery to clear mural thrombi (those attached to the membrane lining the heart) has also been performed. In cases with repeated infected emboli coming to the lungs from the pelvic veins, the main vein (vena cava) from below the heart can be tied off. To prevent further embolism from the leg veins, the physician may perform femoral vein ligation; that is, one or both femoral veins may be tied off by a relatively simple operation under local anesthesia.

The medical treatment is primarily symptomatic, plus the administration of anticoagulants to prevent more clots. This is actually a modern version of the age-old method of applying leeches, the anticoagulants heparin and dicumarol now used resembling in effect the hirudin of the leech. In the prevention of recurrent embolism, both anticoagulant treatment and ligation of the femoral veins are valuable. Chest pain must be relieved, cough controlled, nourishment maintained, and good nursing care provided. Administration of oxygen is often necessary.

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Prevention of lung embolism has consisted of earlier ambulation than was formerly practiced after all surgical, obstetrical, or medical procedures. Daily examination of the patient for early signs of thrombophlebitis in the extremities is needed, plus frequent changes in position to avoid stoppage of the flow of blood in the leg veins. Ligation above any thrombosis found and the administration of anticoagulant drugs should not wait on an embolism. Poorly performed abortion, the commonest cause of infected lung emboli, is a problem involving medico-legal and social factors in the community. Air embolism and fat embolism can often be prevented by better medical care.

APPROACHES TO THE PROBLEM

There is considerable progress being made in developing better tests for lung infarction. Such tests are important because of the difficulties of promptly diagnosing the condition. There is also progress in improving the awareness of the medical profession of this problem and of the importance of taking measures to prevent it. Injectable drugs to dissolve clots in the blood vessels are being used, with excellent results.

The basic problem is that of blood clotting in the veins. Fundamental understanding of this phenomenon is needed. It is possible that means will be found to prevent clot formation in heart disease and in other conditions often complicated by phlebitis and lung emboli.

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