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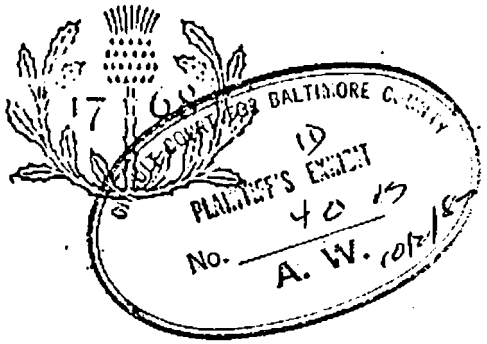

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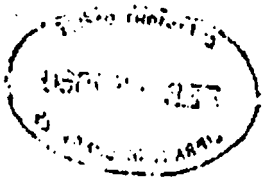

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pneumonias were different from the bacterial forms.

...ide interest in the subject followed the establishment of viral pneumonias as a separate entity by H. A. Reimann in 1938. Apparently there are a number of viral pneumonias caused by different agents, some occurring in epidemic form and others as sporadic cases. The epidemic forms of pneumonia apparently represent the severest cases of otherwise mild infections of the respiratory tract called colds, grip or pharyngitis. The sporadic cases resemble the pneumonias of psittacosis or ornithosis, but since the agents causing the latter can be seen microscopically and are influenced by antibiotics, their classification as viruses is questioned.

Confusion in nomenclature of this group of diseases is not surprising since up to 1951 the cause or causes of them had not been discovered. The term "atypical pneumonia" became widely used, but is undesirable since in Cole's view, any pneumonia not clinically the same as typical pneumococcal pneumonia is atypical, and furthermore pneumonia is absent in the majority of mild diseases of which most viral pneumonias represent the severest stage. The broad term "viral pneumonia" is preferable since all evidence, except the final proof of demonstrating a virus, indicates that viruses are the cause. The name "viroid" was proposed to include both pneumonic and nonpneumonic forms of the respiratory tract disease in question.

In the search for the cause of viral pneumonias, about 25 related or unrelated viruses had been encountered up to 1951, most of which proved to be natural residents of animals used in the experiments, so that the cause or causes still were unknown. However, all other facts pointed to a viral cause, especially the epidemiologic aspects of the disease, the absence of special bacteria in the sputum, the absence of any bacteria in the lungs at biopsy, and experiments in which volunteers inhaled filtered bacteria-free exudates from patients with the disease and developed respiratory tract disease several days later with or without pneumonia, identical with the natural form.

The pneumonic forms begin with symptoms of gradually increasing severity over several days. There is headache, dry paroxysmal cough, slight sore throat, sweating and fever which is irregular, but seldom high, a pulse rate often relatively slow, slight indefinite or absent physical signs of pneumonia which are strikingly less than the roentgenographic evidence of pulmonary invasion, a normal or slightly elevated leukocyte count and rate of sedimentation of erythrocytes, followed by recovery by lysis and few complications. Encephalitis occurs in a small number of patients. Pleural effusion is uncommon. The mortality rate is ... The few deaths which occur are caused by circulatory collapse, severe infections and by the presence of other disease or complications.

The incidence of viral pneumonias varies from year to year but great increases in number of cases come with epidemic periodicity. Epidemics occur at any season of the year, but most commonly in the winter months. During the years 1941 to 1944 viral pneumonias outnumbered all other forms of pneumonia in military groups and among civilians.

Diagnosis is made on the epidemiologic aspects, the clinical behaviour the leukocyte count and the absence of the usual bacterial causes of pneumonia in the sputum. Retrospective diagnosis may be aided by the ability of the serum to agglutinate erythrocytes at low temperature (the cold-agglutinin test) after the tenth day of disease in 50% of cases. Diagnosis of the pneumonia of influenza of Q fever, of psittacosis and of ornithosis, is made by isolation of the respective causative agents or the serologic demonstration of their activity by appropriate tests. The pneumonia of psittacosis, ornithosis, Q fever, coccidioidomycosis, tularaemia, tuberculosis and other known forms may closely resemble the viral pneumonias of unknown cause and must be differentiated since specific therapy for some of them is available. Penicillin and aureomycin are effective for psittacosis and ornithosis, and streptomycin for pneumonia caused by the influenza bacillus and for tularaemia and tuberculosis.

Pathologically, viral pneumonias are characterized by involvement of the structure of the lung and the larger air tubes, and an exudate composed chiefly of monocytic cells. The mucous membrane of all or of parts of the upper respiratory passage may be inflamed and invaded with mononuclear cells. No bacteria are present in the tissue unless secondary invasion occurs.

Treatment.—Penicillin, sulfonamide compounds and streptomycin have no effect on viral diseases. They are used if complications occur caused by pathogenic bacteria susceptible to their effects. Aureomycin, chloramphenicol and terramycin apparently shorten the course in many instances; in others, no effect is evident. Convalescent serum and roentgentherapy are of no value. Oxygen is helpful for cyanosis and dyspnea.

THE OTHER GROUPS OF PNEUMONIAS

Pneumonias of Systemic Diseases.—Pneumonia may occur as an integral part of many systemic diseases. It may dominate the clinical picture, or be a minor factor or not occur at all in tuberculosis, tularaemia, plague, undulant fever, glanders, syphilis, rheumatic fever, ascariasis, periarteritis nodosa, sarcoidosis and many others. The pneumonia is usually not characteristic clinically and is often obscured by symptoms of disease elsewhere. Pathologically it may assume features characteristic of the respective disease. The treatment is that of the disease itself.

Pneumonia of Mixed Infections.—The pneumonias brought about by injury to the lung or interference of their self-cleansing process by obstruction of the airways, foreign bodies, tumours, failing circulation, submersion in or aspiration of fluids, trauma after surgical operation, shock and many other causes, are usually caused by a mixture of various kinds of bacteria which reside habitually in the air passages and are enabled to become invasive by the presence of injured tissue. Pneumococci, influenza bacilli, haemolytic streptococci and staphylococci are most frequently implicated. The treatment is primarily directed at correcting or removing the primary cause, the use of antibiotic agents according to the sensitivity of the invading bacteria to the respective agents, and appropriate supportive measures.

Pneumonia Caused by Noninfective Agents.—The aspiration of oil over long periods or in large amounts often causes a form of chronic reaction commonly called lipoid pneumonia. Long exposure to roentgen rays may also give rise to a pulmonary reaction called irradiation pneumonitis which leads to fibrosis. The inhalation of certain irritating chemicals and certain gases used in warfare or for industrial uses, or fumes from certain molten metals may cause forms of pneumonia. Allergic pneumonitis or Loeffler's syndrome apparently represents the hyperergic inflammatory reaction of sensitized lung tissue to repeated exposure to the specific antigen. The treatment of each condition mentioned under this heading is the removal of the offending agent.

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PNEUMONOCONIOSIS, also called pneumoconiosis, a term denoting a variety of industrial diseases of the lung, embracing lung response to any type of respirable hazard. Precise definition of the term is a matter of legal consequence, because occupational chest diseases are among the most litigious of medicolegal issues. In several countries many of these conditions are obligatorily compensable. To more than 3,000 naturally occurring minerals presenting respiratory hazard are added yearly an ever-increasing number of synthetic chemical agents. The biological action of less than 1% of these substances is known, yet about 10% of working persons handle such agents. While pneumonoconiosis is a generic term, silicosis has for many years been its chief specific manifestation. Because of the increasing range of dangerous respirable agents, however, emphasis is shifting from silicosis to other forms of pneumonoconioses. The majority of the significant reactions represent insidiously progressive chronic responses, frequently with a distinct latent period during which the workman is symptom-free. Fulminating cases also occur when exposure has been unduly severe or adjuvant factors play a part. A simplified etiological classification of the main varieties of pneumonoconioses is given in the table.

PNEUMOTHORAX

Pneumonoconioses

Siliceous	Nonsiliceous
Free silica (SiO_2)	Carbonaceous
Crystalline	Anthracosis* } ..... coal miners
Silicosis proper	Emphysema* }
Mining*	Graphitosis ..... graphite
Ceramics*	Smog lung ..... carbon and chemicals
Glass industry*	
Abrasives*	Metallogenic
Sandblasting*	Siderosis ..... iron
Foundry*	Baritosis ..... barium
Etc.	Stannosis ..... tin
Aberrant silicosis	Cobaltosis ..... cobalt
Todimytosis*	Argyrosis ..... silver
Cristobalitosis*	Aluminosis† ..... aluminum
Amorphous	Berylliosis* ..... beryllium
Natural	Cadmiosis* ..... cadmium
Diatomaceous earth [raw / calcined]*	Manganosis ..... manganese
Synthetic	Vanadiosis† ..... vanadium
Submicron, 20 Å–4,000 Å	Thioconiosis ..... sulfur
	Plumboconiosis ..... lead
Silicates (SiO_4)	Welder's lung† ..... iron + ozone or nitrogen dioxide
Mineral	Brass foundry lung .. copper + zinc
Asbestosis*	
Talcosis*	Organic
Micatosis	Byssinosis* ..... cotton
Kaolinosis	Bagassosis* ..... sugar cane
Fuller's earth†	Ptilosis ..... feather
Artificial	Tabacosis ..... tobacco
Cementosis	Piperosis ..... pepper
Glass wool†	Suberosis ..... cork
	Hemp lung ..... hemp
Mixed forms	Flax lung ..... flax
Disease in Bauxite extractors*	Wood lung† ..... wood
Granite polishers*	Rubber lung† ..... rubber
Slate workers*	Plastics lung† ..... plastics
Boiler scalers*	Farmer's lung* ..... fungi
Kiln liners*	Silo filler's lung* ..... nitrogen dioxide
Basic slag workers	Wheat handler's lung† ..... ?
Hematite miners*	
Magnetite miners*	Miscellaneous
Kolar gold miners	Oleogranuloma ..... oil mists
Insulation workers*	Fume injury* ..... nitric acid, etc.
Transite pipe workers*	Gas injury* ..... hydrogen fluoride, etc.
	Chemical pneumonitis* ..... inorganic, organic

*Disabling and lethal. †Disputed entities.

History.—Man-made lung diseases have been known for the past 2,000 years and were discussed by, among others, Hippocrates and Pliny. "Potters' rot" of ceramic workers, "grinders' rot" of steel implement sharpeners, and "coal miners' asthma" evolved as by-products of the Industrial Revolution. But this group of disorders first assumed major importance when, at the turn of the 20th century, thousands of gold miners in South Africa began to die from "miners' phthisis." Since that time, research and the introduction of rigorous environmental hygiene measures have brought about control over these diseases.

In modern industries, dust hazards have been virtually eliminated by advance engineering planning. Employees in well-controlled plants can anticipate trouble-free working periods of 30 to 40 years in spite of processing highly dangerous substances. Pneumonoconiosis arises in such industries in exceptional cases only. The explanation usually is either extreme individual susceptibility, predisposing disease such as tuberculosis, accidental excessive exposures, hazardous technical innovations or the potentiating effects of exposure to several different substances. The pneumonoconioses have in a few instances affected nonworking persons. Police officers, housewives and school children have contracted berylliosis from the inhalation of smokestack effluvia, dust from soiled working clothes or fluorescent lamp phosphors. The lung injury caused by city smog results from the action of chemical agents adsorbed onto carbon particles emanating in industrial operations. The catastrophic deaths at Donora, Pa. (1946), the Meuse valley in Belgium (1949) and in London (1952) illustrate the disastrous potential of industrial waste products immoderately disseminated into the air of cities.

Causes.—Theories of the causation of pneumonoconiosis include tissue injury by mechanical trauma, protoplasmic poisoning, metabolic changes in cells through a disturbance of pH, protein denaturing through piezoelectric and surface molecular forces, immune and antibody reactions, polymerization and the nonspecific action of submicron particles. No single explanation is universally acceptable. The pathogenic propensity of a number of specific agents has been proved by experimental reproduction of pathognomonic lesions in animals.

Symptoms.—Pneumonoconioses range from innocuous through variably disabling to lethal syndromes. Symptoms are determined by the focus of major lung injury, combinations of lesions, severity and rate of exposure, and infective or other complications. Dominant states include bronchitis, bronchiolitis, bronchiectasis, emphysema, pulmonary fibrosis and pulmonary hypertension. The main complications, which also determine the mode of lethal outcome in most instances, are cor pulmonale with congestive cardiac failure, tuberculosis, pneumonia, pulmonary insufficiency, hemorrhage, lung gangrene and erosion of a blood vessel, a bronchus or the esophagus. Remote lesions may also occur; *e.g.*, portal artery obstruction, metastatic tuberculosis and liver, kidney or suprarenal failure. Respiratory tract cancer is closely associated with exposures to monochromates, asbestos, beryllium, nickel, arsenic and radioactive particles.

Diagnosis and Treatment.—Precise diagnosis depends largely on radiography, but so eclectic and diversified are the manifestations of the pneumonoconioses that interpretation of the X-rays requires great skill and experience. Advanced physiological studies have contributed greatly to understanding of the associated disability. Final diagnosis, however, often must await histopathological and petrographic confirmation.

Once established, the majority of the severe pneumonoconioses are irreversible. Some syndromes may become arrested on cessation of exposure but others, such as silicosis and asbestosis, progress inexorably. Therapy is directed at the alleviation of symptoms such as pain or bronchospasm, support of the failing heart and treatment of complicating infection. Apart from dust suppression, adequate ventilation and the use of exhaust equipment, the only effective prophylactic agent against silicosis is inhaled aluminum administered in the form of aerosols of aluminum oxide, hydroxide or hydroxychloride. Good results have been achieved in cases of berylliosis by means of the chelating agent aurin tricarboxylic acid or by cortisone. No specific therapy is available for other pneumonoconioses. *See* also DANGEROUS TRADES; DUST; INDUSTRIAL MEDICINE. (G. W. H. S.)

PNEUMOTHORAX. The chest is divided into two airtight compartments by the mediastinum. A thin membrane called the pleura (*see* COELOM AND SEROUS MEMBRANES) covers each lung and is reflected at the root of the lung to form the lining of the inner surface of the chest wall. Normally the visceral pleura (covering the lung) is in contact with the parietal pleura (covering the inner surface of the thorax), and the potential space between them is spoken of as the pleural space. A pneumothorax exists if the two layers of pleura are separated by air, and the lung collapses away from the chest wall. Air may be introduced into the pleural space as the result of trauma, by spontaneous rupture of the visceral pleura or by means of a needle inserted through the chest wall.

Traumatic Pneumothorax.—Since the lung is an elastic organ and tends to be pulled toward the lung root, the pressure in the pleural space is less than atmospheric, averaging minus five centimetres of water. This means that air under atmospheric pressure will be sucked into the pleural space if the visceral pleura is torn (*e.g.*, by a fractured rib) or if an open wound in the chest wall penetrates the parietal pleura. If a "sucking wound" of the chest wall exists, more air may be pulled into the pleural cavity via the wound than is pulled into the lung via the larynx, with the result that the collapsed lung and mediastinum are pushed toward the healthy side on inspiration and away from it on expiration. This paradoxical motion of the lung and mediastinum causes progressive asphyxia unless the wound is closed.

Spontaneous Pneumothorax.—The sudden collapse of the lung without previous trauma is termed spontaneous pneumothorax and is usually associated with severe chest pain and difficulty in breathing. Most cases occur in apparently healthy persons. Because spontaneous pneumothorax may occur as a complication of pulmonary tuberculosis, it was assumed for years that almost all cases were due to tuberculosis. This is not true. In 1932 H. Kiaergaard suggested that emphysematous blebs in the pleural surface of the lung were the common cause of spontaneous pneumothorax, and this concept was later confirmed. Superficial blebs on the pleural surface are weakened areas of