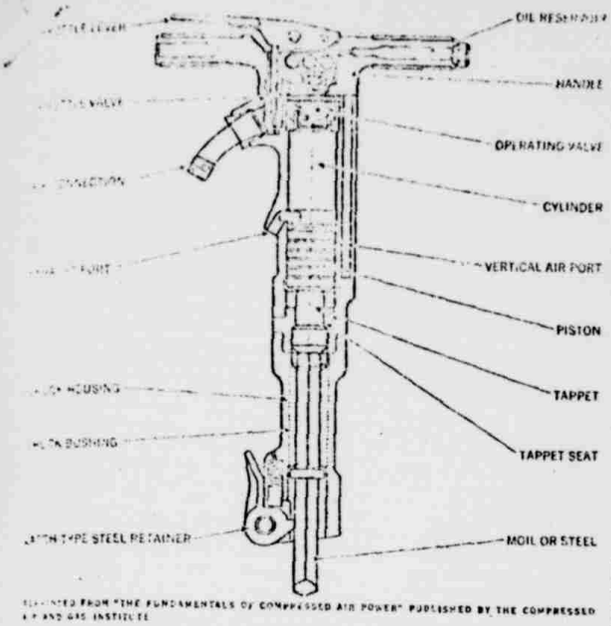


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PORTABLE PNEUMATIC PAVING BREAKER

good for horizontal holes in mining operations and tunnel driving. The usual air-cylinder bore diameters are 3 in., 3½ in. and 4 in. The drill is mounted on some type of rig or frame. The usual drifter is mechanically fed into the work. Stoper drills are used primarily on up-hole or overhead drilling. The usual stoper is a hammer drill with a self-rotating drill and an automatic feed by means of an air piston.

Large air-operated earth drills, mounted on motor trucks or trailers, are used for putting down water wells and blast holes for quarry operations. A high-capacity compressor provides air not only to power the drilling tool but also to raise and lower the tools in the hole and remove drill cuttings from the hole. Such machines are used to advantage in areas where surface water supplies are insufficient to provide the drilling fluid needed for standard rotary and cable-tool well drilling machines.

Paving Breakers.—Hand-operated pneumatic paving breakers usually use solid drill steels and are not equipped with automatic rotation. One type of tool is valve actuated; the other is valveless. Heavy machines, weighing about 80 lb., are used to break concrete pavement, foundations and boulders. Medium breakers, weighing 50 to 70 lb., are used to break light concrete floors, macadam and frozen ground. The light tools, weighing less than 50 lb., are used for breaking floors, paving and masonry walls. Heavy- and medium-weight breakers can be adapted for use in driving spikes. (R. C. Br.; X.)

PNEUMATISM. The theory of pneumatism in medicine, based on the premise that *life is associated with a subtle vapour* called the pneuma, was expounded by the Alexandrian anatomist and physiologist Erasistratus, who flourished about 300 B.C. The concept of *pneumatism* had been previously suggested by other commentators. Unlike Herophilus, who accepted the old theory of humoral pathology, Erasistratus held that health and disease and, in fact, the nature of life were intimately connected with the pneuma, which had affinities with the *air man breathes*. Erasistratus drew a distinction between two kinds of pneuma: one was a "vital spirit" formed in the heart from air; the second type was formed in the *brain from the first kind*. The former was transported by arteries to the parts of the body and the latter, called the "animal spirit," by the nerves, being the prime cause of movement. Although Erasistratus held that hindrance of the flow of the pneuma, or an excess of blood, was the essential cause of certain diseases, he did not follow the contemporary practice of blood-letting, preferring to attempt to control the blood supply by diet and other less violent measures.

PNEUMOCONIOSIS, also called pneumonoconiosis, a term denoting a variety of industrial diseases of the lung caused by breathing dust particles. 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 pneumoconiosis 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 pneumoconioses. 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 pneumoconioses is given in the table.

Pneumoconioses

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
Tridymitosis*	Argyrosis ..... silver
Cristobalitosis*	Aluminosis† ..... aluminum
Amorphous	Beryllosis* ..... beryllium
Natural	Cadmosis* ..... cadmium
Diatomatosis (raw / calcined)*	Manganosis ..... manganese
Synthetic	Vanadosis† ..... vanadium
Submicron, 20 Å–4,000 Å	Thiosmosis ..... 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	Papricosis ..... 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. Pneumoconiosis arises in such industries in exceptional cases only.

Ency. Brit. (1960 ed.) Gerrit Schepers

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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 pneumoconioses 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 workers' 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 pneumoconiosis include tissue injury by mechanical trauma, proteplastic 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 characteristic lesions in animals.

Symptoms.—Pneumoconioses 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 (pulmonary heart disease) 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 pneumoconioses 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 pneumoconioses 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 beryllosis by means of the chelating agent aurin tricarboxylic acid or by cortisone. No specific therapy is available for other pneumoconioses.

See also DANGEROUS OCCUPATIONS; DUST; INDUSTRIAL MEDICINE; RESPIRATORY SYSTEM, DISEASES OF; SILICOSIS.

(G. W. H. S.)

PNEUMONIA, an inflammation of the lung. Pneumonia was recognized as a disease by the ancients, but no progress was made in the knowledge of it until the time of Leopold Auenbrugger (1761) and René Laënnec (1819), when order was brought into its classification. From then until the bacteriologic era opened, classification depended on the anatomic or clinical features, and adjectives such as lobar, lobular, broncho-, interstitial, pleural, double, central, croupous and catarrhal were used. Other pneumonias were called aspiration, contusion, hypostatic or terminal pneumonias, depending upon the cause. Although after 1880 various bacteria and viruses were known to cause pneumonia, the modern etiologic classification did not become popular until about 1930. Precise knowledge of the bacterial or other cause permits the use of specific therapeutic or preventive measures and other rational management in the control of the disease. More than 50 different infectious, physical or chemical causes of pneumonia were known by the second half of the 20th century, but of these, relatively few were of common occurrence. Pneumonias may be grouped as follows: (1) primary, *i.e.*, those caused by pathogenic cocci, bacilli, viruses and fungi that attack the lung primarily; (2) pneumonias that occur in more or less specific forms as part of systemic infections such as tularemia, psittacosis, tuberculosis, rheumatic fever, rickettsial and protozoal diseases, lupus erythematosus and others; (3) pneumonias caused by mixed infection induced by physical or mechanical injury of the lung such as shock, debility, passive congestion, aspiration of foreign bodies of fluid, obstruction to airway, atelectasis, trauma and others; and (4) pneumonias caused by the aspiration of oil, chemicals or dusts, by exposure to roentgen rays, by allergic reaction and other causes.

Pneumococcal Pneumonia.—The modern era of this disease began with the discovery of the pneumococcus in 1880 by G. M. Sternberg and its association with pneumonia by A. Fraenkel and A. Weichselbaum in 1884. F. Neufeld first pointed out serologic differences among strains of pneumococci in 1910, and shortly afterward F. S. Lister and A. R. Dochez independently classified them into types. Much fundamental work on the bacteriology, etiology, pathogenesis, clinical aspects and treatment of pneumococcal pneumonia was done after 1911 at the Hospital of the Rockefeller Institute under the leadership of Rufus Cole and Oswald Avery. Extensive bacteriologic, clinical and epidemiologic contributions on the subject were also made, notably by Russell Cecil, O. H. Robertson and Maxwell Finland in the United States and by H. Loeschke and Max Gundel in Germany. Specific immune serum for treatment reached its greatest effectiveness and popularity as the refined, concentrated form, only to be displaced by the sulfonamide compounds after 1935 and by penicillin in 1942.

Etiology.—There are about 75 types and subtypes of pneumococci, of which only a few are of clinical importance. Types 1, 2, 3, 4, 5, 7, 8 and 14 account for about 80% of all pneumococcal lobar pneumonias. The rest are caused by pneumococci of other types that, in contrast with those mentioned, often are present in the throats of healthy persons.

Pneumococci rarely are primary invaders of the lung. In almost all instances there is evidence of some preceding condition that injures the lung and favours invasion, especially minor infections of the respiratory tract commonly called colds; chilling of the body, and debilitation. The pneumococci that become invasive are acquired by inhalation from outside sources or are already present in the pharynx; the first circumstance indicates that pneumonia may be a contagious disease contracted from other patients or carriers of pneumococci, and the second that it may arise as an autogenous infection. Under certain conditions when the general resistance is lowered by serious conditions, and in old age, the pneumonia is apt to be atypical or lobular (bronchopneumonia); in other words, it does not behave like the lobar form. The onset is gradual, pulmonic solidification may not occur and the course is irregular. The pneumococci causing this form are usually of the higher numbered types, indicating that they are part of the normal flora of the respiratory tract and have become invasive.

Pathology.—Pneumococci reach the lung by way of the air passages, settle in an area where they find circumstances suitable for growth and give rise to an area of inflammation conditioned by complex factors, among which partial immunity, allergy and nervous influences seem to play an important role. The inflammation spreads rapidly and soon involves part or most of a lobe, first as congestion and then with an outpouring of fibrin and polymorphonuclear leukocytes into the alveolar spaces. The exudate becomes rather solid and grayish over a period of several days, and finally, when recovery occurs, it softens and is resorbed. Pneumococci often enter the blood stream and may localize on the valves of the heart, the meninges or elsewhere. The pleura may be invaded, resulting in empyema.

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