Pathology and Pathogenesis.—Pulmonary infarcts are found usually in the lower lobe of the right lung when no previous disease, such as pleurisy, previous infarction, etc., has weakened the strength of the stream in the right pulmonary artery. The gross appearances of pulmonary infarcts are generally characteristic. While they vary somewhat in shape and position, yet it may be said that they are chiefly wedge-shaped and peripherally situated. The apex of the wedge is embedded in practically normal pulmonary tissue, while the base is at the pleural surface of the lung, presenting an area of dark red or purplish color, somewhat elevated above the surrounding parts. Some infarcts are conical and many of these are found deep in the lung substance.

When recently formed, infarcts are hard and swollen and sharply defined, and on section the surface is smooth or slightly granular and of a deep, dark red or purple color, with an appearance often of a dense blood clot. It is rare to find but a single infarction of the lung. They are usually multiple. In size they vary within wide limits, although it may be said that they are generally from one to four cubic inches in measurement; they have, however, been described as occupying the greater part or the whole of one lobe.

When examined microscopically the tissues within the area of infarction are found filled or stuffed with blood, the capillaries engorged, and the air cells distended with red corpuscles; some of the vessels, however, may be quite empty. The bronchi are often seen more or less filled with a flaky substance in which blood and epithelial cells in various stages of disintegration may be found. When subjected to a staining process, the signs of necrosis are observed in the failure of cells and nuclei to stain normally. This is often first seen about the centre of the infarct. In the older infarcts the hyaline thrombi of von Recklinghausen, by whom they were regarded as causative factors, are seen in greater or less numbers, while fibrin is present in abundance. The vessels of the pleura are also greatly congested in many specimens, and in the pleura itself signs of thickening with inflammatory exudate are common.

After Laënnec, Cruveilhier explained infarction in the lung by attributing it to a primary inflammation of the vessels, chiefly the venous capillaries, and accounted for all clots or coagula within the vessels upon this ground. It remained for Virchow, several years later, to refer these changes, included under the term "infarction," to disturbances of circulation induced by embolism or thrombosis. At the same time it appears that Virchow left the question of the relationship between embolus and infarction an open one, although it would appear from certain comparisons made that, in the great majority of cases at least, he regarded this relationship as one of cause and effect.

From the time when these views were set forth (*i.e.*, about 1856) until even the present, hemorrhagic pulmonary infarction, so far as its relation to embolism and thrombosis is concerned, has been under discussion and many views have been advanced.

The following are the most important of these: Cohnheim, alone and with Litten, came to the conclusion, on experimental grounds, that the pulmonary artery belonged to that group of arteries in different organs known as *end arteries*, and that occlusion of such arteries was especially favorable to the formation of infarcts of the hemorrhagic variety. The vessel wall, thus robbed of its nourishment, permitted the blood which came as a venous reflux to pass through it and out into the tissues. Hence venous reflux and occlusion of an end artery constituted the main points in this view.

In an article published in 1891, P. Grawitz maintained that in order to induce or to have induced in the lung the condition of infarction, an abnormal condition of the pulmonary tissue must be present, such as the brown induration of heart failure, inflammation of the lungs, chronic bronchitis, etc. Grawitz found the blood-vessels which he regarded as the source of the hemorrhage, in the newly organized and vascularized tissue incident to chronic bronchitis, that is, in peribronchial, subpleural, and interlobular fibrous tissue. Given, then, such a state of lung tissue with a disease of the heart, such as mitral regurgitation or stenosis, and all the conditions were fulfilled for a hemorrhagic infarct. And should such an infarct be found with an embolus in the artery leading thereto, he would say, "an infarct in spite of the embolus."

Gsell, in his work published in 1895, maintains that typical hemorrhagic infarcts may result from emboli in some branch of the pulmonary artery, but that they are much more likely to occur, and to occur in greater numbers, when there is an abnormal condition in the pulmonary tissue, as hyperæmia, atelectasis, etc.

Some of the above-mentioned observers, together with several others, attempted a solution of this question from the experimental side with various and hence confusing results. Fujinami, in 1898, published the results of his researches, which included experiments upon eighteen dogs and five cats. In addition to these he carefully examined thirteen cases of infarct of the lungs found in the human subject. This very careful study resulted in establishing beyond doubt that a pulmonary infarct is a consequence of circulatory disturbances in the capillaries in certain areas. This occurs in many instances as a result of emboli occluding branches of the pulmonary artery, even in a healthy lung. There are certain conditions, however, which aid in bringing about these circulatory disturbances.

It has been seen from the above cursory review of this part of the subject that much interest has centred about embolism as a causative factor; and that both experimentally and clinically embolism is established as a cause of pulmonary infarction. The evidence is not so clear, however, when one turns for proof of those other causes of infarction, viz., thrombosis of the pulmonary artery and the occlusion of a terminal bronchus, bringing about atelectasis.

It must be admitted that thrombosis of the pulmonary artery may occur apart from embolic processes; but, according to the present consensus of opinion, this is not a common finding. Thrombosis of this vessel, when found under such conditions, may be said to fall under one of the following classes:

1. Hemorrhage between the adventitia and the media, occurring as a result of pulmonary disease to which the arterial change is secondary; or a primary hemorrhage in this vessel, resulting in an occlusion of its lumen through pressure upon the media.
2. Endarteritis, or fatty degeneration of the artery.
3. Chronic lymphadenitis involving the vessel walls.

Atelectasis acts but rarely as a cause of infarction. When it does so it is doubtless through the absorption of air and the resulting alterations in blood pressure in the affected part, whereby some blood is extravasated into the tissues. There is reason to question this mode of production, or, even accepting this as a cause of hemorrhage, to disregard such hemorrhages as those of infarction.

Before leaving this part of the subject, we must consider the source of the blood in pulmonary infarction, as well as the mode by which it gets into the tissues. We have already referred to the view expressed by Grawitz, who maintains that the newly vascularized tissue formed as a result of pulmonary disease gives rise to the hemorrhage. The vessels in this tissue rupture under increased pressure.

Hamilton, of Aberdeen, scornfully dealing with the embolic theory of infarction, states that the blood gushes out of the overdistended pulmonary capillaries in heart disease and takes the characteristic wedge shape because it is effused into a certain space corresponding, not to the distribution of a terminal artery at all, but to a group of alveoli about a terminal bronchus.

Earlier writers upon this subject would account for the hemorrhagic character of the infarct by a reflux of blood in the veins of the affected part; but, in more recent times, this view has been set aside by careful experiments and close observation. Regurgitation does not take place through the veins. The capillaries, along with any arterial anastomoses, supply the blood and the red cells pass out by diapedesis which, as Welch has pointed out, is greatly favored by slowing and stagnation of the blood current and a high intracapillary and intravenous pressure.

Classification.—Pulmonary infarcts are usually red or hemorrhagic. It is rare that an opportunity is afforded one of studying an anæmic or white infarct of the lung, although it does occur. Freyberger's report, published in 1898, presents a good example of this variety. It is his view, as the lung in his case contained in all twenty-two infarcts, and but one of them was white or nearly so, that this was the last one formed; and, as the patient died a lingering death, the circulation had become so weak that anastomoses could not be established. The writer suggests, partly as a result of a study of this observation, that, at the first instant or so of formation, all pulmonary infarcts are anæmic.

Changes in Infarcts.—One rarely finds in the lungs signs which one must regard as those marking the site of a former infarct. However, such are occasionally discovered, a small fibrous pigmented scar being the only evidence of former circulatory disturbances. There seems to be little doubt but that the smallest may undergo resolution, yet the larger ones rarely do. They may become organized in part or again gangrene may result or an abscess may form in the area of infarction and subsequent infection. In a lung taken from the body of a person dead from cardiac disease, and examined while this article was in preparation, a deep red, wedge-shaped area, fluctuating and very soft to the touch, was discovered near the apex of the right upper lobe. When this portion of the lung was cut into it was found to contain a dark brownish fluid—the liquefied infarction clot. Leading up to it a branch of the pulmonary artery was occluded by a small embolus. The right auricle contained a thrombus.

Conditions Favoring Infarction.—From what has been already stated it may be concluded that cardiac disease, in which the pulmonary circulation is abnormally altered, favors infarction. There is a heightened venous and capillary pressure under such circumstances. Then again, thrombi not infrequently are found in the right auricle. Chronic pulmonary disease may also be induced by the cardiac state. Thrombosis in the systemic veins, from any cause, may give rise to the pulmonary affection. Diseased conditions in the pulmonary artery itself, rapidly inducing thrombosis, favor infarction.

It must be borne in mind that embolism and thrombosis may each take place in the branches of the pulmonary artery without infarction occurring. In one case infarction does not occur, as the circulation is completely established, while in the other death may quickly supervene, affording no occasion for such changes.

Symptoms and Signs.—In many cases there are no clinical features whatever to denote the pulmonary changes incident to infarction. Intense thoracic pain with pronounced dyspnœa may usher in the closing scene of a case of chronic cardiac disease or one of venous thrombosis. Such cases terminate with signs of asphyxia or syncope. In other cases partial recovery may follow the alarming and distressing onset and in a short time—the space of a few hours—dark red blood may be expectorated. Of thirty-seven patients dying with pulmonary infarction in the Royal Victoria Hospital, only fifteen expectorated blood. The temperature usually remains undisturbed, but later it may become febrile as a result of the process of gangrene or abscess formation. Pain in the side is often complained of, while a pleural friction sound may mark more definitely the site of infarct. Dulness on percussion and blowing breathing are often present. Fine moist râles may be heard. The breath sounds may be absent.

Diagnosis.—The greater number of pulmonary infarcts are latent. Of thirty-seven cases showing this condition in the lungs at autopsy in the Royal Victoria Hospital, only four were recognized definitely before death. Sudden and severe dyspnœa was noticed in four, while pain over the region of the infarct was felt in two cases.

One must exclude asthma, coronary-artery disease, and pneumonia. As a rule this is not difficult. The history and the course, and, in the case of asthma especially, the physical signs, are characteristic.

Prognosis.—Much depends upon the extent of the infarct, the condition of the heart and lungs, and the possibility of recurrence in other parts.

Treatment.—A word in this connection may be said regarding prophylaxis. There can be but little doubt that early or violent movement of a limb whose vein is thrombosed, or massage over such an obstruction, may occasion embolism. Hence the necessity of an intelligent and careful treatment of such cases. Well advanced involution of the parturient uterus should be secured before the patient is allowed to get out of bed and move around. One must seek to maintain the compensation of the heart in cases of endocarditis. Since so little can be done by active treatment of infarction, the object should be to prevent its occurrence.

In addition to these measures for prevention, treatment consists in but little more than the relief of symptoms and chiefly that of pain.

William Fawcett Hamilton.

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LUNGS, DISEASES OF: LOBAR PNEUMONIA. See *Pneumonia, Lobar.*

LUNGS, DISEASES OF: ŒDEMA.—Etiology.—This occurs in practically the same diseases as does passive congestion. The two conditions have much in common. When occurring in the neighborhood of inflammations, tubercles, new growths, infarcts, etc., it is termed collateral œdema. It is a common complication of chronic Bright's disease, chronic heart disease, very severe anæmias, cachexias, and cerebral affections. The pre-agonal form is common and does not cause the patient's death, but occurs because he is dying.

Pathology.—There is usually an hydræmic state of the blood, with increased intrapulmonary pressure. Welch thinks that the essential factor lies in a disproportionate weakness of the left ventricle, so that the blood accumulates in the lung capillaries till transudation takes place.

The lung is heavy, pits on pressure, and the involved parts are filled with serum; if the œdema is associated with congestion the serum has a reddish color. The lung may have a gelatinous appearance.

Diagnosis.—The symptoms are very indefinite and cannot be dissociated from those of the primary disease; there is perhaps an increase in the severity of the cough and dyspnœa. Large quantities of sero-mucoid fluid are expectorated.

The physical signs are not remarkably characteristic. The presence of numerous fine, moist, uniform râles in the dependent portions of the lungs, in association with œdema in other parts of the body and secondary to heart or kidney disease, is very suggestive of pulmonary œdema. The breath sounds are usually weak. Percussion may be negative, unless hydrothorax or hypostatic pneumonia is present, when dulness will be elicited.

Pulmonary œdema may occur suddenly, with a rapidly fatal issue.

Treatment.—It is that of the primary disease.

James Rae Arneill.

LUNGS, DISEASES OF: PNEUMONOKONIOSIS.—Definition.—Etymologically, pneumonokoniosis is a general term indicating deposit of dust within the pulmonary parenchyma. Owing, however, to the intimate etiological relations which such deposit bears to subsequent pathological changes, the term is commonly used as including the earlier stages of the diseases thus arising.

Synonym.—The specific term anthracosis is often and, indeed, usually employed for the generic and more cumbersome one originally proposed by Zenker.

Classification.—As the pathological processes and anatomical changes do not differ in character, whatever the nature of the foreign matters, the classification is based upon the form of the inspired dust, and may be extended almost indefinitely.

The more common forms are anthracosis (ἄνθραξ, coal), due to a deposit of coal or other carbonaceous dust; siderosis (σίδηρος, iron), applied to all metallic dusts as well as iron; chalicosis (χάλιξ, gravel), including the various forms of mineral dust; byssinosis (βίσσος, cotton), due to cotton or other vegetable fibre; tabacosis, inhalation of snuff or tobacco dust. Deposits of any other form of dust may receive similarly appropriate names.

On account of the evident relations which certain diseases bear to dusty occupations they have received such characteristically descriptive names as miners' or stonecutters' phthisis, masons' or millers' lung, potters' asthma, buffers' consumption (among metal polishers), Sheffield grinders' rot, and elevator disease, or "scoopers'" pneumonia.

History.—Both physiological experiment and postmortem examinations have conclusively proved that foreign matters inhaled in fine subdivision, not only pass to the bronchial surfaces, but also reach the alveolar cavities, enter the pulmonary parenchyma, and are finally lodged in the bronchial glands. The relations of such dust deposits to various forms of chronic lung disease have long been recognized. Hilaire (Paris, 1845) and Vernois (Paris, 1858), among earlier writers, and more recently Michel (Bonn, 1872), Kuntzen (Berlin, 1873) ("Handb. d. spec. Path.," Ziemssen, Leipzig, 1874), Roy (Bordeaux, 1884), Perissé (Bull. Soc. de Méd., Paris, 1894), and others have described both the pathological changes and the clinical manifestations of pneumonokoniosis in its chronic form. We are indebted to Rochester (Buffalo, N. Y.) for much of our knowledge of the more severe acute conditions. The changes caused by infective elements of dust do not receive consideration in the present article.

Pathology and Morbid Anatomy.—It is not possible to suppose that any dust can pass by direct inhalation beyond the second or third bifurcation of the bronchial tubes, or that it remains suspended in the residual air. The first point of deposition must be, then, upon the bronchial mucous membrane, at some considerable distance from the alveoli. The larger portion is here taken up by the mucous corpuscles, or becomes entangled in the bronchial secretion and is thrown off in the expectoration. More or less, however, makes its way along the bronchial tubes, notwithstanding the opposing action of the cilia, and although it is gradually lessened in amount by expectoration a small residue eventually reaches the alveolar cavity, where the particles may be found closely adherent to the epithelial surface.

Occasionally this distribution occurs quite evenly throughout the lung, but more commonly the apices receive the larger portion, which by gradual increase becomes in some cases sufficient to fill the smaller tubes and alveoli and cause consolidation.

Of those particles which are finally lodged on the alveolar walls some become incorporated with the epithelial cells, to remain there permanently or to be transferred to the subjacent lymph spaces. Others reach the lymphatic channels by insinuating themselves between the epithelial cells, which are loosened and elevated by the rapid growth of new cells that is excited by the irritating presence of the foreign bodies, and from here they are carried in by cellular elements which are probably migrated white blood corpuscles.

From this point the distribution follows the course of the lymph channels, more especially in the sheaths of the bronchial tubes and smaller branches of the pulmonary artery, and in the interlobular septa. Many of the pigment granules are arrested along the course of the lymphatic vessels. They either become clogged in the lumen of the vessel or some sharp point pierces the thin wall and they are then embedded there or pass into the connective-tissue spaces. At many points they become aggregated in minute nodules which completely block the vessels and arrest the lymph current. When this condition is very extensive, nutrition of the pulmonary tissue may be seriously affected.

Notwithstanding the continual permanent deposition of the dust particles along the lymphatics, proportionately large amounts pass through these vessels and finally become deposited in the bronchial glands. Only in rare instances do particles find their way to the cervical or abdominal lymphatics.

All the pulmonary tissues thus become infiltrated and stained by processes which are physiological, or at least conservative, since they are directed to the removal of the irritating foreign particles from the delicate alveolar walls, where even small amounts are productive of serious inflammatory changes, to the lymphatic glands, in which considerable quantities can be stored without special detriment to the system.

The above are the processes involved in all forms of pneumonokoniosis. The morbid anatomical appearances will vary with the amount and nature of the material deposited.

Post-mortem examinations show a moderate amount of carbonaceous and other extraneous pigment deposits in the lungs of all adults, more especially of such as have resided in cities. In the lighter grades the surface of the lung is uniformly mottled and striated in black or deep brown, the striæ marking out the interlobular septa and the pigmented spots indicating concretions in the lymph vessels or areas of lymphatic plexuses.

As the pigment deposits increase the color deepens, until in the higher grades of anthracosis the lung is of a uniform coal-black color, while the pleura presents a bluish-black and semi-transparent appearance, owing to the implication of only the deeper layers in the pigmentation. On passing the fingers over the surface distinct hard nodules may be detected, either causing slight elevations or lying more deeply embedded in the substance of the lung. The lungs are increased in size, often markedly so; they have everywhere a firm resistance, which in some portions amounts to an almost stony hardness. They crepitate but little, and their specific gravity is in many cases raised above 1.000.

On section the cut surface presents the same variations in color, from a fine outlining of the lymph courses to a uniform black. Here the nodules and concretions become more apparent, varying in size from the most minute appreciable point to others the size of a pea. On pressing the lung a more or less deeply stained fluid exudes, from which the pigment matter may be obtained and its nature determined. The concretions when isolated resemble minute bits of coal.

Upon microscopic examination, in the earlier stages, the lines of pigmentation are seen to follow very closely the distribution of the lymphatic vessels. Later, the pigment granules may be detected in the alveolar epithelium and free among the connective-tissue fibres. In many instances the nature of the pigment matter can thus be recognized.

The changes in the bronchial glands are equally varied in extent. As increasing amounts of inorganic matter become arrested in their meshes, gradual absorption of the glandular substance takes place, while the glands themselves become enlarged and indurated, until in extreme cases they may reach the size of walnuts, and on section present the appearance of encapsulated, compact masses of fine coal. When other pigments than carbon

are deposited within the lung, the only variation in the anatomical appearances will be in the color. The oxide of iron gives a brown or reddish color, and the metals generally give a lighter tint. Silica and the various clays cause gray tints, which are often darkened, however, by admixture with carbon elements. The ease with which the various forms of dust penetrate the tissues will determine largely the proportionate distribution of the pigmentation. In anthracosis the bronchial glands are quickly affected. The same is true of some of the metallic dusts and silica in some forms. The various clays pass but slowly into the tissues, and the pigmentation will therefore be more strongly marked in the interlobular septa near the alveoli.

It is to be remembered in this connection that high grades of pulmonary pigmentation, with quite decided enlargement, induration, and staining of the bronchial glands, may be due to processes entirely independent of inhaled matters, and that in some instances it is not possible to distinguish anthracotic from melanotic pigment derived from the blood.

An exception must be made to the above description as applied to byssinosis and allied forms of dust. When inorganic matters are mixed with the inhaled organic fibres, as happens with dirty cotton, they become separated by mixture with the bronchial secretions and afterward pass into the lung tissue as already described.

The organic fibres, however, cannot pass through the alveolar wall, and, indeed, they seldom are carried thus deeply into the lung, but are gradually softened in the mucous secretions, become rolled into slate-colored gelatinous masses, and are thrown off in the expectoration.

The above constitute those changes which can strictly be called pneumonokoniosis. They are seldom present alone, however, and in the higher grades they always induce secondary changes.

Whatever the nature of the dust inhaled, the secondary processes excited by reason of its chemical or mechanical irritating qualities are identical in character; they vary only in intensity and in the order and proportion of their development.

Dust deposits occur in the lung in a large proportion of cases intermittently and with extreme slowness, few artisans working over ten, and miners only eight, hours out of the twenty-four. The consequent diseases are, with equal frequency, chronic inflammations and degenerations.

The one most constantly and earliest developed is bronchitis. It presents no peculiarities beyond a tendency to the production of an exceedingly viscid mucus. The mucous membrane at first is thickened; later, it is atrophied, and may be ulcerated or contain ecchymoses.

Closely following the bronchial changes, and coincident with the passage of the dust elements into the interlobular tissue, there occurs a low grade of productive inflammation, characterized by cellular infiltration and connective-tissue hyperplasia.

These fibroid changes at first produce thickening of the interlobular and alveolar septa, but as the new tissue becomes organized and begins to contract, pulmonary nutrition is decreased, the septa atrophy and finally are absorbed, and the lung tissue gives place at various points to firm, tough bands and masses of the new growth. Adjacent lobules, which have escaped, in part, the fibroid processes, become distended, thus developing a compensatory emphysema. Similar fibroid changes about the tubes exert traction, which, in connection with softening and ulceration of the tubes, causes bronchial dilatations or bronchiectasic cavities. These bands of new tissue may be several inches in length, and are at times an inch or more in thickness. They have no definite outline, but merge gradually into the surrounding tissues. Small fibrous bands pass from the pulmonary tissue to the deeper layers of the pleura, where a similar fibroid condition exists. Such changes are best marked along the anterior borders of the lungs, and over such areas the pleura may be thickened by organization of surface exudation as well as by the subpleural changes.

The contracting fibroid growth not only induces atrophy and absorption by compressing the capillaries, but causes similar obstruction to the circulation in the larger pulmonary vessels and lymphatics, a condition which in the lymph vessels is augmented by pigment concretions and glandular infiltration.

As a result, local congestion, exudation, œdema, or even extravasation may occur, and in extreme cases infarctions, abscess, and gangrene are present. These, by rupture or sloughing, form large ragged cavities, whose walls continue to secrete offensive pus, which appears in the expectoration mingled with gangrenous shreds of pulmonary tissue.

Chronic bronchitis and fibroid phthisis are thus seen to be the necessary complications of chronic pneumonokoniosis, to which lobular pneumonia and compensatory or atrophic emphysema are often added. Such a lung would seem to furnish a fertile soil for the growth of tubercle bacilli, and it is a noteworthy fact that tuberculous processes are developed late if at all.

It has been questioned whether acute inflammatory processes, with exudation and cellular proliferation, are ever excited within the alveolar cavities by the inhalation of dust. Recently such a condition has been described as affecting grain-shovellers, in which the etiological element was unquestionable. The lungs are never seen until the process is well advanced. Then the pleura is found to be adherent, deeply congested, red, thickened, and covered with a false membrane of plastic exudation. Serous effusion into the pleural cavity is rare. The lung itself is dark red, with occasional points of extravasation just beneath the pleura.

Consolidation is most marked posteriorly, and is due to both vascular engorgement with serous exudation, and inflammatory products within the alveoli and smaller bronchioles. The consolidated portion is soft and pulpy, breaking down easily under pressure. On section it presents a deep red or gray color, according to the stage, and from the cut surface there flows a frothy, bloody, or purulent fluid. Small infarctions and abscesses may be present in the later stages.

Under the microscope the alveoli are seen to be filled with exudative products and granular or broken-down cellular elements. Rarely, a bit of the beard from the grain may be recognized.

The pathological processes, which affect both lungs, appear to be a mixture, in varying proportions, of hypostatic and broncho-pneumonia, accompanied by circumscribed areas of plastic pleurisy. Changes in the liver and kidney are functional rather than organic.

Etiology.—Predisposing conditions: There is no condition which strictly can be considered as predisposing to inhalation of dust, beyond the anatomical conformation of the nasal and respiratory passages, except the habit of mouth-breathing.

Workmen who habitually inhale through the mouth, or whose occupation compels them to take sudden, deep inspirations often suffer more in a dusty atmosphere than those who, though working under the same conditions, breathe through the nostrils and inhale more gently.

Very many conditions, however, under which artisans labor exert a strong influence in increasing the extent and severity of the diseases consequent upon dust inhalations.

Imperfect ventilation of mines or workshops and overcrowding of operatives result in a vitiated air which of itself tends to pulmonary congestion and inflammation. Under such circumstances not only is the bronchial mucous membrane more susceptible to irritation, but the amount of dust deposited is relatively larger.

Again, constrained positions, as in mining, or occupations requiring but little muscular effort, as in metal-polishing, not only tend to favor the rapid accumulation of inhaled matters and lessen the ease with which they are expectorated, but they seriously interfere with pulmonary nutrition, and so decrease the power of resisting deleterious influences.

All inherited vices of constitution, more especially the

lymphatic diathesis, enervating habits of life, the use of alcohol, and excesses of all kinds, lower vitality and predispose to pulmonary disease when pneumonokoniosis or any other irritant is the exciting cause.

Exciting Causes.—Any form of inorganic dust, and very many organic products, when persistently inhaled will produce various degrees of pneumonokoniosis, which in turn may be the direct cause of any of the secondary diseases.

It were superfluous, then, to attempt to mention all the exciting causes. Among the more common avocations, however, in which laborers are exposed for prolonged periods to a dusty atmosphere are mining of the various minerals, and the handling of anthracite or bituminous coal in transit to its point of consumption; charcoal-grinders and carriers, moulders and those who clean castings, metal- and glass-polishers, stone-masons and plasterers, chimney-sweeps and laborers who tear down old buildings, potters and grinders on various forms of stone, bakers and pastry cooks, gilders and gold or tin-foil beaters, workers in mother-of-pearl and lead, jewel- and glass-cutters, file-cutters, millers, tobacco-workers, factory operatives, grain-shovellers, etc., through a still longer list, all suffer from inhaling the peculiar dust produced by the nature of their avocations, and develop varying grades of pneumonokoniosis.

Recent years have seen very great improvements, however, in the measures taken to protect operatives, particularly polishers and grinders, and a corresponding decrease in this form of disease.

The extent and character of the inflammatory changes, together with the order and rapidity of their development, will depend upon several factors.

1. The amount and character of the exposure. Other things being equal, the secondary conditions will stand in a direct ratio, as to their extent, with the amount of dust deposited. The rapidity with which this deposition takes place affects very decidedly the nature and severity of the subsequent disease. When artisans breathe a dusty air for only a few hours each day, as is almost invariably the case, the lungs soon accommodate themselves to the new conditions, and the usual processes of absorption are sufficient practically to clear the alveoli of foreign matters during the hours of non-exposure. There will be an acute bronchitis for a short time, but it soon subsides and passes into a chronic form, which is unimportant and causes little trouble to the patient. In such cases the principal changes will be fibroid in character and may not become prominent for years, the rapidity of their development depending upon conditions yet to be considered.

The results are very different when dust is inhaled continuously for a long period.

In handling grain the shovellers not only labor in confined places, as the holds of canal boats, where there is absolutely no ventilation, but they work without intermission for days. The gang bosses admit that the labor is sometimes continuous for thirty-six hours, while the workmen claim that they are often employed for five and six days, with intermissions of only a few moments for food and rest. Taking an average as the truth, it gives three and four days as the probable length of time during which every respiration bears to the lung large quantities of an exceedingly irritating dust. Under such conditions the absorptive processes are inadequate for its removal, and the tubes become filled with the irritant. The resulting inflammations are acute exudative processes. As before, bronchitis appears first, usually following the first exposure, but later, similar exposure induces the pseudo-pneumonic changes already described.

2. The nature of the inhaled dust, as regards its penetrating power and chemical qualities.

The most penetrating, as well as the most irritating, forms of dust are the siliceous, as the particles have exceedingly sharp edges and fine points. Similarly, mineral coal passes into the tissues more easily than charcoal, but both are only slightly irritating, owing to their chemical properties, as compared with other forms of dust. True anthracosis often reaches a condition of almost complete solidification without inducing any extensive fibroid change. Although pulmonary diseases are much more frequent among miners than in the community at large, the percentage of phthisis cases to the total number of sick, among this class, is lower than in any other class of dust workers (Hirt's statistics).

Various clay dusts pass into the lung but slowly, being deposited more thickly about the alveoli; yet they possess specially irritating properties and speedily cause severe disease. Metallic dusts also stand high in the list of irritating matters.

Tobacco dust passes into the lungs quite freely, but the resulting diseases are due to its constitutional effects rather than its locally irritant properties.

The frequency with which pneumonokoniosis is an etiological factor in pulmonary phthisis among the laboring classes in large cities may be fairly determined by the following list, taken from one thousand consecutive cases of pulmonary tuberculosis entered upon the dispensary records of the University Medical College and Bellevue Hospital, only those being given here in which dust inhalation was a possible factor. It is but fair to state, however, that probably only a small percentage of the 311 cases classed as laborers were engaged in handling coal. The list includes only males.

Printers	48	Machinists	14
Carpenters	45	Bakers	12
Masons	39	Moulders	10
Painters	28	Hatters	7
Tobacco-workers	24	Wood-turners	6
Factory hands	23	Glass-workers	4
Stonecutters	23	Millers	4
Iron-workers	20	Weavers	3
Blacksmiths	18	Gold-beater	1
Brass-workers	15	Dyer	1
Total			345

The above cases, together with those which should be taken from the class of laborers, form nearly forty per cent. in which inhalation of dust can fairly be regarded as having predisposed to the phthisical processes.

Symptoms.—Chronic pneumonokoniosis presents but few symptoms. So long as the patient continues his occupation the bronchial secretions will contain pigment matters. Cough is an early and persistent symptom. It may be due to either bronchitis or pressure of an enlarged bronchial gland. Dyspnœa is often a prominent symptom even when no appreciable inflammatory conditions are present, and appears to depend upon deficient oxygenation caused by abundant pigment deposit. Other symptoms will depend upon the secondary diseases. Subacute and chronic bronchitis will afford the usual subjective and physical signs. In some cases the prominent symptoms will be those of fibroid phthisis, with compensatory or atrophic emphysema. In others the asthmatic element is prominent, and in all the physical signs of pleurisy will be present at an early stage of the disease. Inflammation or enlargement of the bronchial glands (*q. v.*) will cause characteristic pressure symptoms. If tuberculous infection occurs it will soon be indicated by rise of temperature, hectic, rapid exhaustion, and hæmoptysis.

The phthisis of anthracosis, however, is seldom tuberculous or rapidly progressive, but tends to abatement or even recovery when the exciting cause is removed.

The acute processes which ensue upon the prolonged inhalation of specially irritating dusts are the most severe in the so-called elevator disease.[1]

The earliest attacks are in the form of acute bronchitis with profuse muco-purulent expectoration, unattended by fever or other constitutional symptoms. Within a year or two, however, when from repeated attacks of bronchitis the lungs are more susceptible to irritation, some especially prolonged period of exposure excites an inflammatory process resembling acute broncho-pneumonia.

The more decided symptoms are preceded for a day or two by some bronchial irritation, cough, and expectoration. Distinct onset of the disease is marked by a light chill, and a rapid rise of temperature to 101° F. in mild, or 105° to 106° F. in severe cases, with an average of 103° F. The pulse is frequent and feeble, and the heart's action tumultuous. The face is flushed, but the skin remains moist. Delirium is frequent in both the sthenic and the asthenic cases. The cough is increased, and the sputa become thick, tenacious, rusty, or hemorrhagic; later they are purulent, with an exceedingly offensive odor.

On physical examination both lungs are found to be affected. There is partial consolidation in the posterior and lower portions of the lungs, with evidences of alveolar and bronchial exudation such as are usually present in hypostatic pneumonia. The physical signs of a plastic pleurisy are often present over the consolidated portion.

The disease runs a prolonged course of from ten days to two weeks with sthenic symptoms, and convalescence may not be complete for two or three months.

Many cases pass into a condition presenting all the rational signs of phthisis, with hectic, night sweats, and rapid emaciation, but without the physical evidences of tuberculous infection.

In connection with the pulmonary processes there may be renal and hepatic complications, and general disturbances in the digestive functions.

Diagnosis.—The recognition of pneumonokoniosis, either as a condition *per se* or as a causative factor in other pulmonary diseases, depends entirely upon the history of the case and the detection of pigment in the sputa.

Prognosis.—The prognosis depends primarily upon the possibility of removing the exciting cause, and the extent and character of the secondary changes.

When the subject cannot or will not give up his occupation, the duration of the disease will depend upon the general habits and constitution of the patient, and upon those factors which are more fully discussed under Etiology.

Treatment.—Prophylaxis is the only practical line of treatment. Since men must work at dusty avocations, means should be devised for preventing the inhalation of the dust particles. In those trades in which dust is formed at a single point, as in polishing, glass-cutting, wood-turning, etc., some form of blower or aspirator which will entirely remove the dust is the most effective protection. When, however, the dust is evenly diffused, as in tobacco factories and iron foundries, any amount of ventilation which would successfully remove the dust will become a source of danger from cold and draughts. In such cases respirators, although more or less clumsy and disagreeable, are exceedingly valuable. In all cases they possess the advantages of being under the control of the operative and always available, while ventilators, blowers, and aspirators must often be wrung from soulless corporations. A cheap respirator, and one which not only has the advantage of being easily cleaned, but is also very efficacious, may be made from a fine flat sponge.

Treatment of the secondary complicating chronic diseases presents no peculiarities.

For the acute broncho-pneumonia it is generally tonic, stimulant, and symptomatic. *Charles E. Quimby.*

[1] Rochester: Buffalo Medical and Surgical Journal, 1879.

LUNGS, DISEASES OF: PNEUMOTHORAX.—Definition.—By pneumothorax is meant the presence of air in the pleural cavity. In the great majority of cases a collection of fluid rapidly follows, either serous or purulent, and termed hydropneumothorax and pyopneumothorax respectively.

Etiology.—According to West and other writers, about ninety per cent. of cases of pneumothorax are due to the rupture of a tuberculous focus on the surface of the lung, allowing of the entry of air to the pleural cavity.

The disease usually occurs on the side in which the tuberculous lesions are most advanced; it occurs with greater relative frequency in acute cases of tuberculosis, and when occurring in chronic cases febrile symptoms are often present, indicating an exacerbation of the disease. Weil found that twenty-two out of forty-six cases were in subjects of acute tuberculosis, although it is well recognized that this type of the disease is rare compared with the chronic form. The period at which it is most common is during the first year, thirty of Weil's forty-six cases occurring within this time.

Both Douglas Powell and West agree that the condition occurs in about five per cent. of all cases of pulmonary tuberculosis.

It is more particularly in cases of rapidly forming small superficial cavities, lined by friable caseous tissue, that rupture is apt to occur. In more chronic conditions there is less danger of rupture taking place, owing not only to the less friable structure, but also to the presence of pleural adhesions.

Pneumothorax may occur early in the course of tuberculosis and even be the first sign of disease, but it is more common in the well-developed or advanced cases. In Weil's series only eleven of the forty-six cases occurred in the early stages of the malady.

Of late years a considerable number of cases of pneumothorax, occurring in apparently healthy persons, have been reported. It is highly probable that most of these cases are really due to a latent tuberculous focus. This view is borne out by cases in which a subsequent post-mortem examination has been made, and, as West points out, in many of these cases the pneumothorax has come on during rest or sleep, a circumstance which renders it highly improbable that the lung was in a healthy condition.

Of the ten per cent. due to other causes than tuberculosis, empyema rupturing into a bronchus is the most important. It is, however, only a small proportion of such cases that are followed by pneumothorax. Considering the fact that there is a negative pressure in the pleural cavity, it is remarkable how seldom pneumothorax is observed in such cases or when the pleura is torn by a fractured rib. West regards the absence of pneumothorax as being due to the cohesion between the two layers of the pleura, and supports his view by some very ingenious experiments. By making a double membrane of a piece of stomach, placing the serous surfaces in apposition, and then attaching the double layer to a bell-jar, connected by its other end to an air-pump, it was found that the two layers of membrane remained in apposition, unless there was a considerable diminution of air in the bell-jar. Gangrenous areas in the lung resulting from inhalation-pneumonia, putrid bronchitis, or other causes, occasionally terminate in pneumothorax.

Traumatism is an occasional cause. Penetrating wounds of the chest wall, such as stabs or gunshot injuries, severe crushing injuries, fracturing ribs and tearing the lung, or even rupture of the lung without fracture of ribs, are all recognized causes of this condition. Owing, however, to the force of cohesion, already referred to, it is only in a minority of such cases that pneumothorax actually occurs.

Rupture of an emphysematous bleb has been noted in a few instances, and a series of cases has been recently reported by Zahn (*Virchow's Archiv*, cxxiii., p. 197). Chauffard has also recorded a case presumably due to this cause, tuberculosis being excluded by the tuberculin test.

Holmes records a case of pneumothorax setting in some days after injury, and regards the condition as first of all an interstitial emphysema, and thence extending to the surface of the lung and rupturing into the pleura.

Hemorrhagic infarcts may break down and give rise to pneumothorax; Hale White attributes the pneumothorax observed in rare cases of typhoid to this mechanism.

Wilks and Moxon noted the occurrence of two cases of pneumothorax after tracheotomy; Money also found two instances in twenty-eight cases of tracheotomy in which it was specially looked for (Med.-Chir. Soc., lxvii., 1884).

It seems probable that air passes beneath the deep fascia of the neck to the mediastinum, and thence ruptures into the pleural cavity. This view is strongly borne out by Champney's experiments on dead infants, in whom tracheotomy and artificial respiration had been performed, air