

is always unfavourable. In the treatment, ammonia may be administered.

The Treatment of the foregoing diseases—the second group according to the classification on p. 115—may now be considered together, as they are all treated upon the same lines. In acute bronchitis caused by cold, the initial symptoms, or the very mild cases affecting only the larger tubes, are treated in the same way as ordinary coryza. In the severe cases, in adults, a turpentine stupe should be placed for half an hour or longer around the chest, which should afterwards be covered with a layer of cotton wadding. This is preferable to poulticing, in many cases, as there is less liability to chill. Sometimes, however, it is too severe a measure, and then poultices must be used, with, or without mustard, care being taken to have the second poultice ready to place upon the chest the moment the first is removed. In children, poultices or liniments (Bowe's, or compound camphor liniment) may be used, the latter having the same advantage as the turpentine stupe in adults, as wadding may be kept around the chest continuously, and the nurse's hand passed underneath when the liniments are used. The temperature of the room should never be allowed to fall below 63° Fahr.; and in the severe cases with obstruction to the breathing, the atmosphere around the patient's head should be kept moist and warm by means of the bronchitis kettle. Great care should be taken when steam is used, that the lamp is never allowed to go out, as there is then danger to the patient from the cold damp hangings surrounding him. If the patient be robust enough, the early stages may be treated by depressants. Small doses of tartar emetic (one-sixteenth of a grain every three hours) or antimonial wine (twenty minims every three hours), or tincture of aconite (in drop doses every ten minutes for two hours) may be used during the first day. Carbonate of ammonia is the most useful remedy in bronchitis, and it may be prescribed upon the second day, or from the first, if it be undesirable to use depressants. R 18 is a useful combination. Five to ten minims of solution of morphine hydrochlorate may be added to each dose when the cough is very troublesome. Sometimes a gargle of chlorate of potash may relieve the cough; and sprays are useful with children. Bartholow highly recommends a combination of carbonate and iodide of ammonia—five grains of each, every four hours—for bronchitis, catarrhal pneumonia, and croupous pneumonia. When the breathing is much obstructed by the tenacious mucus an emetic should be given. Half a teaspoonful or more of ipecacuanha wine (repeating in ten minutes if necessary) is the best for children. Apomorphia, hypodermically, may occasionally be required for adults when the mucus obstruction is very great. Quinine or antipyrin—fifteen grains of either—should be given when the temperature is high. The dose may be repeated three or four times at intervals of four or six hours. Chloral, fifteen grains, and bromide of potassium, forty grains, are the best remedies for sleeplessness. The dose may be repeated in two hours if necessary. Opiates should not be given

for this purpose, as in the larger doses they tend to dry up the pulmonary secretions. Morphine may be given hypodermically for pain or sleeplessness. When the case is improving, and the fever has passed away, an expectorant mixture (R 19) may be used, or the carbonate and iodide of ammonia may be continued. Cod-liver oil and iron tonics are indicated during convalescence. Alcohol may be necessary at times, or throughout the case, and the diet should be light and digestible, consisting chiefly of beef tea, milk, chicken broth, wine-whey, and egg flip, &c., given in small quantities, frequently repeated. The causes of bronchitis must be considered with the treatment; hence when due to valvular disease of the heart, digitalis will be indicated. In Bright's disease, also, the treatment will be modified. Fibrinous bronchitis may be benefited by a spray of lime water, which is a solvent for fibrinous matter.

In acute catarrhal pneumonia the treatment is the same as in acute bronchitis. In the sub-acute or chronic forms, turpentine, eucalyptus, and copaiba are useful remedies. Externally, iodine liniment or tincture is used—several coats being painted on the chest.

In active congestions of the lung, cupping or leeching at the bases, is useful in the earliest stage. Large mustard plasters may be placed upon the chest. Stimulating expectorants and digitalis are prescribed. In croupous pneumonia the treatment may commence with a large dose of quinine (twenty grains), or other antipyretic. Depressants may be used the first day if the patient be robust; but usually R 20 is given from the beginning. The external agents used are the same as in bronchitis. Digitalis is considered by some authors to increase the embarrassment of the heart. If a heart tonic be necessary, five to eight minims of tincture of strophanthus would do away with this objection, as the strophanthus does not raise the blood pressure by acting upon the capillaries, like digitalis. Strychnine, however, is the most reliable cardiac stimulant. The patient should be allowed water to drink, and the body should be kept cool by frequent sponging with cold water. Ice may be placed in the arm-pits, &c., when the temperature is high. Bromide is given for sleeplessness. Alcohol is very urgently needed in croupous pneumonia; and especially is it necessary with alcoholic patients. It should be continued for some days after the crisis, and the doses should be gradually diminished, if it has been necessary to order large quantities during the illness.

Chronic Bronchitis.—**Pathology.**—In chronic bronchitis the air-tubes are filled with muco-purulent matter, while the mucous membrane is seen to be much congested, thrown into folds, and of a dark red colour and glossy appearance. The smaller bronchi are often dilated. Minute depressions on the surface of the mucous membrane may be seen, by the aid of a small lens, to be dilated mucous glands. Microscopically, the whole of the bronchial wall is seen to be much thickened, and infiltrated with cellular structures. The epithelial lining of the tubes is irregular, the cells being in part cast off, and presenting the appear-

ance of a germinating membrane of round and pyriform-shaped cells. The capillary blood-vessels are dilated, and the cartilages and muscular fibres atrophied. The cellular structures, confined by the elastic basement membrane, make their way along the lymphatics, and in very chronic cases they are the cause of chronic interstitial pneumonia (cirrhosis); and especially is this the case when the cause of the bronchitis has been the inhalation of irritating dusts, as with coal miners, needle grinders, and stone masons. When the bronchitis is due to chronic valvular disease of the heart, the state of *brown induration* of the lung is induced. Punctiform hæmorrhages are then seen under the pleura. The whole lung is intensely congested, and throughout its substance there are indurated semi-solid patches, which, microscopically, are found to consist of blood effusions and epithelial cells containing brown pigment. The alveolar capillaries are dilated from mechanical causes. The congestions of the different organs, and the pathological conditions described in the section dealing with heart disease, will also be present. For the further pathological changes due to secondary complications, see *Emphysema*, *Interstitial Pneumonia*, and *Bronchiectasis*.

Chronic bronchitis may result from frequent acute attacks; but often it begins insidiously in those who are much exposed to cold and wet. It may be one of the results of chronic valvular disease of the heart; or it may arise in connection with the scrofulous diathesis, rickets, Bright's disease, infectious fevers, and parasitic diseases* (actinomycosis, aspergillosis), &c.; or it may be due to the constant inhalation of irritating dust, as already mentioned.

The symptoms begin with a tickling cough and increased expectoration, as in the acute form. The cough and expectoration is worse in the morning. There is not much dyspnoea at first, but this gradually increases. There is no fever unless an acute attack supervene. There is emaciation in the later stages. The physical signs are the same as in the acute form, to which are added the symptoms and physical signs of the causal affections and secondary complications. These are discussed later as separate and distinct diseases. It should be noted that while chronic valvular disease of the heart may be the cause of bronchitis, bronchitis in its turn may increase the embarrassment of the heart; and it may—without valvular disease being present—so dilate and weaken that organ, that eventually all the secondary train of symptoms due to obstructed circulation (described under heart disease) may set in.

Recovery from chronic bronchitis may sometimes take place if not too far advanced; and children suffering from chronic bronchial catarrh frequently "outgrow" their attacks of "winter cough." If the patient be placed in favourable circumstances, his condition

* *Actinomycosis*, a chronic infective disease, occurring chiefly in cattle, but sometimes in man, due to a micro-parasite setting up chronic inflammation, with the formation of granulation tumours, which often suppurate.

Pulmonary aspergillosis, runs a course either like bronchitis, or acute pulmonary tuberculosis, and is due to infection by a fungus. Both diseases occur in those whose occupations bring them in contact with infected grain.

may be ameliorated, and life may be prolonged; but complete cure is a rare event. Death may result from an intercurrent acute attack, or from slowly developed emphysema, and dropsy, as in the valvular diseases of the heart.

Bronchorrhœa serosa is a form of chronic bronchitis accompanied by the expectoration of large quantities of thin, watery fluid. *Fetid bronchitis* I have preferred to place in a separate group with bronchiectasis and gangrene of the lungs.

The diagnosis and differential diagnosis are considered in a summary at the end of the section on diseases of the respiratory system.

In the treatment of chronic bronchitis the diet should be light, but generous and nourishing. Over-loading the stomach predisposes to asthmatical attacks. The clothing should be suitable, and flannel should always be worn next the skin. Damp atmospheres are to be avoided. The medicines of most value are turpentine, eucalyptus, copaiba, benzoic acid, and cubebs; and these may be administered in capsules, in their proper doses. Easton's syrup combined with arsenic is a useful remedy. Cod-liver oil is often indicated. Sometimes the carbonate along with the iodide of ammonia, relieves; and even, according to Bartholow, may cure a case of chronic bronchitis. An expectorant mixture is often required—R 19, or 18—which, although apt to disorder the stomach when long continued, yet often seems to give most relief to the patient. Codeia—in syrup or pill—is the best remedy for the cough. Mentone and San Remo are *stimulating* climates, while Torquay, Penzance, and Madeira are examples of the *sedative*. Bournemouth has the advantage of a pine forest.

Subacute or chronic catarrhal pneumonia is frequently associated with chronic bronchitis. It is mentioned in this place for the sake of the classification and the reasons given for the same, on p. 114; but the disease is considered with acute catarrhal pneumonia, in part, and the remainder with phthisis.

Chronic Interstitial Pneumonia. Cirrhosis of the Lungs. Fibroid Phthisis.—This disease is to be regarded more as a secondary lesion rather than a primary one, as it always follows some irritation in other parts. Some authors, however, consider the pathology of cirrhosis as still unsatisfactory, and it may be that it occasionally arises as a disease *per se*, and then it is spoken of as a *chronic pneumonia*. In phthisis, cirrhosis is often described as a part of the pathological condition. Chronic bronchitis, by extension of the cellular structures, along the peribronchial lymphatics; catarrhal pneumonia, for the same reason; *pneumoconiosis* (coal, steel, and stone dust); and phthisis; are the primary lesions which give rise to cirrhosis of the lungs. Thickening occurs in the fibrous tissue along the line of the lymphatic vessels. The bronchi become dilated either from the expiratory force of the cough, or from the contraction of the thickened interlobular septa (bronchiectasis). In very chronic cases tubercles and caseous material are found in the lung-tissue, while there is

more or less compensatory emphysema present. The pleura is thickened, and the cavity, in extreme cases, may be obliterated, or the parietal and visceral layers may be firmly bound by numerous adhesions. The pulmonary vessels are dilated and thickened. Cirrhosis is commonly bilateral but often it is unilateral, especially when the primary irritating cause (as in phthisis) is limited to one lung. In *anthracosis*, the lung, besides being pigmented, has jet-black nodules throughout its substance, which are hard, and may be picked out. In *stone mason's* lung the nodules are grey, and the lung is more markedly cirrhotic.

The symptoms of chronic interstitial pneumonia are mixed up with the symptoms of the initial disease by which it is caused, and its presence may only, in many cases, be surmised. Inspection reveals the flattening, and in extreme cases, the indrawing of the chest-walls, due to the adhesions and their contraction. The heart is frequently drawn from its normal position. The measurement of the chest with the cyrtometer reveals great shrinking of the affected side as compared with the sound side—*i.e.*, in unilateral cases. The percussion-tone is impaired, and dulness is most frequent at the apices which are much shrunken. *Auscultation* gives the breath-sounds, accompaniments, and vocal resonance, according to the nature of the primary disease. In the phthisical—whether phthisis be the primary cause of the cirrhosis or *vice versa*—the physical signs of phthisis are present. The history of the previous health, and the course of the disease, are, therefore, important points in the diagnosis.

Pleurisy with contraction may simulate cirrhosis; and so may malignant growths. When the cirrhotic contractions affect the heart, the case may terminate fatally, by ascites and dropsy.

The treatment is to remove the patient from the cause, if possible; but generally it can only be symptomatic. Cirrhosis, most frequently, can only be viewed as incidental to bronchitis or phthisis.

Emphysema of the Lungs.—(1) *Vesicular* emphysema is sometimes classified—(a) Large-lunged emphysema; (b) small-lunged or senile atrophic emphysema; (c) local or compensatory forms. (2) Interstitial emphysema. In the *vesicular* forms there is an over distension of the pulmonary alveoli, while in the interstitial there is infiltration of air within the interstices of the lung-tissues. The two forms may co-exist when a rupture of the lung takes place. The air may, in extreme cases, make its way under the superficial fasciæ of the neck and chest, and it can there be felt to crepitate.

Vesicular emphysema is commonly a consequence of chronic bronchitis; but it may occur as a primary disease. The lungs are anæmic and much increased in volume. They overlap the heart in front, sometimes entirely. The lungs do not crepitate, and they feel like “thin bags loosely filled with feathers.” The parts most affected are the anterior borders and apices. When the air vesicles rupture, large bullæ are often formed. The circulation through the air vesicles is hindered, and hence there is congestion towards the central parts, and the heart becomes dilated. This is followed by the train of symptoms leading to dropsy, described under the valvular diseases of the heart.

Microscopically, the alveoli are seen to be enormously dilated, while their walls and the interlobular septa are much thinned or wasted away. Fatty granules are seen amidst the remains of the alveolar epithelium. It has been suggested, in order to explain these cases which arise without any history of antecedent causal disease, that emphysema may be produced by changes within the pulmonary tissue itself. It appears sometimes to be hereditary.

The cause of vesicular emphysema has been shown by Jenner and Mendelssohn to be forced expiratory and muscular efforts, as in coughing, &c. “The chosen seats of emphysema are those parts of the lung which are the least compressed during expiration” (*Jenner*).

The symptoms of emphysema are generally superadded to those of chronic bronchitis. The dyspnoea gradually increases, and at last it becomes very great. The patient complains of a “feeling of inflation” about the chest, and any intercurrent attack of bronchitis or asthma is always dangerous, and may prove fatal. There is only a cough when bronchitis is present. Very rarely is there hæmoptysis in emphysema. Inspection reveals the “barrel-shaped” chest in the advanced cases. The sternum and upper ribs are arched, and the arching of the dorsal vertebræ is increased. The circular shape of the chest is well seen when the cyrtometer is used, and a tracing is made. When the patient is made to cough, there is marked bulging at the root of the neck and in the upper intercostal spaces. There is distension of the veins at the root of the neck. Sometimes a transverse groove is formed when the thorax is markedly arched in its whole extent. In marked emphysema, with a barrel-shaped chest, there is little or no movement of the ribs in respiration, the diaphragm being the chief muscle used in the act.

The *percussion* yields important information, especially over the region of the heart. In the vertical diameter the dulness may not commence until the fifth or even the sixth rib is reached. In extreme cases the lungs cover the heart entirely, and there is no dulness on percussion, while palpation cannot detect the apex beat. There may then be pulsation in the epigastric region. The liver, in advanced cases, is also depressed—dulness (in the mammary line) beginning only when the seventh or eighth rib is reached. Percussion of the bases of the lungs reveals a clear note an inch or two lower than the normal, the character of the tone being almost tympanitic. *Auscultation* in an uncomplicated case of vesicular emphysema, reveals an absence or enfeeblement of the vesicular murmur. The normal bronchial breathing heard under the right clavicle and at the level of the fourth dorsal vertebra appears distant or greatly enfeebled. These physical signs are, however, in most cases masked altogether by the râles and rhonchi of the associated bronchitis, and *prolonged expiration*, rather than an absence of the vesicular murmur, is apparently present. Jenner has pointed out that sometimes the pulse after coughing becomes full and tense, and afterwards it is lost for a little time. A tricuspid murmur may be detected. Death

may result, like heart disease, by the obstruction of the circulation leading to dilatation of the heart, stasis, and dropsy.

The prognosis is grave in advanced emphysema. If there be much bronchitis, or if the patient be subject to severe attacks of asthma, there is great danger. A certain amount of emphysema is not incompatible with long life if the patient be placed in favourable circumstances.

The treatment of vesicular emphysema can only be palliative, and it consists in the treatment of the asthma and bronchitis, or in assisting the heart as in cardiac dilatation (see p. 34).

Fœtid or Putrid Bronchitis.—This condition occurs in cases of chronic bronchitis with dilatation of the bronchi. It is more apt to attack those in middle life. Small friable masses (Dittrich's plugs) accumulate within the bronchial tubes. The expectorated matter is highly offensive and of a dirty grey-yellow colour. Microscopically, numerous organisms, crystals, and fat globules are found. The offensive odour is due to the presence of volatile fatty acids. When the sputum is allowed to settle in the jar, three layers are formed—the upper opaque green-yellow and frothy, the middle an albuminous liquid like serum, and the lower stratum opaque dirty-yellow, and made up of pus cells and detritus.

The symptoms in severe cases consist of the sudden development of the *gangrenous odour*, with increased dyspnoea, followed soon by typhoid symptoms leading to collapse, coma, and death. The *post-mortem* examination reveals the sloughing and inflamed bronchial tubes full of the offensive matter; while here and there are patches of gangrene in the lung-tissue, with œdema. Slight cases sometimes recover; and some others run on for months.

Bronchiectasis.—This affection is closely allied, *clinically*, to the preceding. There is an offensive expectoration, and a dilatation of the bronchial tube, but as the latter is more defined than in the simple dilatations (bronchiolectasis) associated with bronchial catarrh and putrid bronchitis, it gives rise to different physical signs, and to a different *manner* of expectorating the offensive liquid.

There are three forms—the cylindrical or fusiform, the saccular, and cavernous. The cavities contain muco-purulent matter, and the walls are smooth. Sometimes there are fibrous bands crossing the cavities. The walls are never granular. The secretion consists of small casts, mucus, recent and putrid, with micro-organisms of putrefaction, and sometimes crystals of fatty acids and cholesterine. Numerous hypotheses have been advanced as to the causation of bronchiectasis. Grainger Stewart and Gibson believe it to be a "primary atrophy of the bronchial wall"; while Ewart considers that "the faulty distribution of space between the air-tubes and the pulmonary tissue" affords the best basis for explanation of the varied pulmonary conditions found.

The symptoms inseparable from bronchitis—as cough, dyspnoea, lividity, &c.—are present; but the one symptom peculiar to the disease is the absence of all expectoration for a time, and then the

sudden coughing up of a large quantity of offensive liquid. The physical signs are only marked when the cavity or cavities are large, and repeated examinations discover variations in the breath-sounds and percussion-tones, depending on the size of the cavity and whether it be full or empty. When large and full, there may be found dulness on percussion, and an absence of the breath-sounds over the part; when empty there is no dulness, but bronchial breathing and bronchophony may be made out. The râles too are coarser than those usually heard in the normal-sized tubes. The *constitutional* symptoms ultimately developed are due to septicæmia, and pyæmic affections may develop in other organs besides the lungs.

Gangrene of the Lung.—Gangrene is always the result of severe injury to, or intense inflammation of, a tissue. In the case of a lung being the seat of gangrene, the characteristic appearances of pneumonia are generally seen, but the gangrenous part is very soft and of a dirty green or black colour. The tissues emit a horrible odour, unless the inflammation has been so severe as to destroy life early, without giving time for putrefactive changes.

The causes are numerous. Croupous pneumonia may lead to gangrene in drunkards, and in the old and feeble, or in those suffering from diabetes or other chronic wasting disease. Gangrene seldom follows croupous pneumonia, in the otherwise healthy and robust individual. More commonly septic matter, by direct extension, as in ulcerating cancer of the œsophagus, suppurating hydatids or abscess of the liver, ulcers of the stomach, and even purulent empyema—sets up gangrene of the lung. Septic emboli, derived from a cerebral sinus (as in ear disease), or from an ulcerative endocarditis, may also produce gangrene. From the bronchial tubes, foreign bodies or food—especially when vomiting has occurred while using anæsthetics—and putrid matter derived from putrid bronchitis and bronchiectasis, may light up gangrene of the lungs.

The characteristic symptom is the horrible fœtor, only absent in the rare and very rapid forms. The sputum is like that which occurs in cases of fœtid bronchitis and bronchiectasis, but it differs in not separating into layers in the jar. It is dirty-grey or green in colour, sometimes black. Hæmoptysis is not common. If the gangrene be superficial, it may affect the pleura and a septic pleurisy may result. The pulse is feeble, small, and rapid. There is pyrexia, and the patient soon passes into the typhoid state if the disease be severe and about to terminate fatally. The stethoscope does not reveal any characteristic sign peculiar to gangrene. Sometimes fine râles and the signs of a cavity—as cavernous breathing and loud bronchophony—are made out, and percussion dulness may be observed. If phthisis be present these signs prove nothing, unless they have developed at the same time, or after the fœtor and expectoration.

The treatment of the members of the fœtid group is the same, inasmuch as they all require inhalations of turpentine, carbolic acid, iodine, creasote, or eucalyptus. Internally, carbonate of ammonia, camphor, quinine, and especially *iron*, should be administered.

The diet should be very generous, and alcohol should be allowed. *Fetid* bronchitis requires also the same stimulating treatment as the ordinary forms. Bronchiectasis may be treated by intra-tracheal injections (menthol and olive oil); or by Chaplin's creasote method. Dr. Poore recommends the use of garlic, which may be taken in capsules. Gangrene of the lungs does not allow of much time for treatment, if acute; but turpentine and eucalyptus—five drops every two hours—are recommended.

Phthisis Pulmonalis, and Miliary Tuberculosis.—The special works on pathology must be consulted upon this subject, as a mere outline of the morbid anatomy is all that can conveniently be included in a practical work of this kind. Tubercular disease of the pulmonary organs is the result of the invasion of the lungs by the tubercle bacilli. They make their entrance usually either by the blood-vessels, and then the miliary nodules are disseminated; or by the bronchial tubes, when the specific cell-growth is excited in the bronchioles and alveoli (peribronchitis and broncho-pneumonia). The disease, at first local, may invade more distant organs, and the constitutional effects are believed to be due to a chemical poison, produced by the bacilli, circulating in the blood.

A simple sub-acute or chronic catarrhal pneumonia, when the bacilli have gained access to the alveoli, becomes a phthisis. The inflammatory products form dense homogeneous masses, which interfere with the circulation of the blood, and so affect the nourishment of the tissues that a condition favourable to caseous or cheesy transformation is produced. There is absorption of the fluids, fatty degeneration of the cell elements, and disintegration of fibrinous matter—the result being the formation of a soft cheesy solid. Softening of these masses begins in the centre, and cavities (vomices) are thus formed, which may coalesce by the breaking down of the surrounding tissues. These cavities are at first irregular, but ultimately they become smooth. Fibrous bands are sometimes seen traversing the cavities. When putrefactive changes take place, the affected parts become foul and brown-green in colour. The sputum is then offensive. The examination of the expectoration with the microscope reveals elastic fibres, with mucus and purulent cells and debris, and the bacilli (when stained). Hæmorrhage may result from erosion of blood-vessels. The apex of the lung is the usual seat of the disease, but a whole lung may be affected by disseminated soft yellow-white or grey tuberculous nodules (phthisis florida). In the later stages of the forms of phthisis which commence with catarrhal pneumonia, tubercles are superadded to these conditions. The form just described is often called *caseous phthisis*.

Tubercular phthisis is the form which begins by the deposit of tubercles in the lung, chiefly at the apex, and these leading to desquamative pneumonia produce the same changes as in the caseous form. The source of infection is frequently a caseous gland, but it may be derived from disease in the lung itself. The tubercles are grey-white, translucent, semi-solid nodules about the

size of a millet seed. Microscopically, they are seen to be composed of a reticulum enclosing numerous cells within its meshes. In or about the centre, *giant cells* with numerous nuclei are found. The tubercles become caseous, and then they soften and break down along with the surrounding caseous pneumonic patches. There is, in short, a necrosis of the pulmonary elements. The mucous membranes are ulcerated by the tubercles.

In the form sometimes known as *fibroid phthisis*, there is slow development of cirrhotic tissue, causing atrophy and degeneration of the true lung-tissues. Tubercles ultimately develop. (See Chronic Interstitial Pneumonia, p. 95).

The pleura (in all forms) is usually affected with chronic inflammation, and ultimately the tubercles invade the inflamed parts. A cavity often ruptures into the pleural sac, and pyo-pneumothorax is the result. If pleural adhesions have formed it may discharge through the thoracic wall (fistula). The bronchial glands enlarge and undergo caseation, and they may sometimes suppurate and burst into the trachea, bronchi, or œsophagus. The enlarged glands may also give rise to suffocative attacks, especially in children, by pressure upon the trachea or vagi. The peritoneum, bowel, urinary tract, and the larynx are often affected by tubercular disease. In advanced and chronic cases the liver is often fatty; but perhaps this is greatly due to the cod-liver oil given in the treatment. The kidneys suffer from waxy disease, and hence there is albuminuria. Secondary pyæmic abscesses sometimes affect other parts, as the brain, liver, kidneys, &c. In the cases of phthisis which recover, there is either extrusion or absorption of the caseous matter, with formation of connective tissue and subsequent contraction; or the caseous matter may become calcified.

Miliary tuberculosis is the sudden and general infiltration of the pulmonary, as well as other tissues, with an enormous number of tubercles brought by the blood current. The infective source may be at a distance, or may itself be in the lung. A case of phthisis may terminate by the acute dissemination of tubercles. Owing to the greater susceptibility of the apices to tubercular infiltration, a case beginning as an acute miliary tuberculosis may, on the other hand, terminate as a case of ordinary phthisis.

For clinical purposes we may classify phthisis thus—the *caseous* form, or acute pulmonary tuberculosis; *tubercular phthisis*, or chronic pulmonary tuberculosis; *fibroid phthisis*; and *miliary tuberculosis*. The forms are often *mixed* more or less; but the antecedent disease, history, mode of origin, and frequently the symptoms often enable such a differentiation to be made.

The caseous form occurs in the weakly or strumous. The predisposition may be acquired by bad hygienic conditions and surroundings. Measles and whooping-cough are often associated with catarrhal pneumonia, and many cases terminate in caseous phthisis. A severe neglected cold is the commonest cause of all. The predisposition to acquire the tubercular form of phthisis is often hereditary; and some consider that the germ of the disease is com-

municated from the parent to the embryo. A bad shape of chest favours the development of tubercular phthisis. Damp climates and bad hygiene are also unfavourable. Phthisis is believed to be communicable to those predisposed to the disease; but the exposure requires to be somewhat prolonged under ordinary circumstances.

The symptoms of phthisis vary according to the form which attacks the patient. In the early stages of the acute or sub-acute cases (*caseous phthisis*), the usual complaint is that of a severe cold, with cough and expectoration of mucus, and later, of muco-purulent matter. The "cold" does not get better, or there is apparently improvement; but he soon fancies he has caught another cold, and these being repeated, he ultimately complains of continuous pains in the chest, feverish attacks, debility, and general *malaise*. The appetite fails and the patient feels unable for his work. The cough begins to be very troublesome, and perhaps the expectoration may be tinged with blood. A smart attack of hæmoptysis frequently occurs at this stage. The patient becomes anæmic and he loses flesh rapidly. The frequent chills, not unlike ague, with hectic flush upon the cheeks and hot hands, give place to undoubted pyrexia, accompanied by severe perspirations. The temperature varies from 100° in the morning to 102° or 103° Fahr. in the evening, but it is reduced after sweating. The pulse is feeble and quick, and after exertion of any kind it becomes still more so. The pain in the side is not acute unless pleurisy, at the lower part of the chest, is present. The coincident pleurisy at the apex does not cause acute pain, as the expansion of the chest is not great in this region. The hæmoptysis may be the first symptom calling attention to the case. Sometimes, in this form of phthisis, the symptoms begin suddenly with acute rigors, and for the first week or fortnight the disease runs a course similar to lobar pneumonia; but oftener the disease is insidious in its progress, and develops as a broncho-pneumonia, which, when wide-spread, may take the form of a *galloping consumption* (*P. florida*). This latter form frequently follows influenza.

When a patient begins to recover from an attack of hæmoptysis, the expectoration becomes less and less sanguineous, and its brown-red colour becomes clearer, until only the ordinary muco-purulent matter is coughed up. The expectorated matter is frequently "nummular." Microscopically, elastic fibres may be seen on examination of the sputum. (Some of the expectorated matter should be boiled for three minutes in an equal part of a solution of caustic soda (20 grs. to ʒi); next the resulting liquid is put into a conical glass with pure water. Some of the deposit which follows is then put upon the microscopic slide and covered with a cover-glass.) For the detection of the bacillus tuberculosis, the following preparation is necessary (*Heneage Gibbs' method*):—

"Take of rosaniline hydrochloride two grammes, methyl-blue one gramme; rub them up in a glass mortar. Then dissolve aniline oil, 3 c.c., in rectified spirit, 15 c.c.; add the spirit slowly to the stains until all is dissolved, then slowly add distilled water, 15 c.c.; keep in a stoppered bottle. The sputum having been dried on the cover glass in

the usual manner, a few drops of the stain are poured into a test-tube and warmed; as soon as steam rises pour into a watch-glass and place the cover-glass upon the stain. Allow it to remain four or five minutes, then wash in methylated spirit until no more colour comes away; drain thoroughly and dry. Mount in Canada balsam. The bacilli are stained red."*

Before proceeding to the physical signs, the symptoms of the other forms—so far as they differ from the "caseous form"—may first be described. There are two types of constitution associated with tubercular disease—the delicate strumous, and the lymphatic strumous. In the first, the figure is tall and thin, with long neck and prominent larynx; the shoulders slope, and the thorax is narrow or flat, and it has little expansile mobility; the hair is fine and generally fair, sometimes "lanky"; the eyes are large and bright, and the skin is delicate and flushes easily; the fingers are long and often "clubbed" at the extremities. They are bright and nervous individuals, fond of active work, but easily fatigued. Those of the *lymphatic* type are heavier in make. They are slow in their movements; and their skins are coarser and have a "bad colour." The hair is more frequently dark. Epistaxis—and, in women, disorders of the menstrual functions—are common in tubercular subjects. In rare cases the menstruation is *vicarious*.

The chronic tubercular form of phthisis usually begins very insidiously. The appetite is variable, and there is loss of weight without apparent cause. The patient is easily fatigued, feverish attacks begin (*hectic fever*), and a short dry cough, pains in the chest, and nocturnal perspirations may be among the earliest symptoms. Hæmoptysis may occur now, or it may be the first symptom. The pulse is quick and feeble, and there is breathlessness especially upon making any exertion. The respiratory movements—always shallow in tubercular patients—become very rapid; but actual dyspnoea can only be said to exist in the very advanced cases. Sometimes this form of pulmonary tuberculosis begins with symptoms of bronchial catarrh, or pleurisy, or by the early development of laryngeal disease.

Fibroid phthisis is the most chronic of all. The early symptoms are those of a chronic bronchial catarrh. The cough is at first dry and the expectoration consists of simple mucus. In course of time his condition becomes worse; but there is little emaciation in the early stages. After a few years, feverish attacks—in the evening generally—nocturnal sweats, hæmoptysis, and the other symptoms as in the tubercular form, begin to manifest themselves. Ultimately, the case is indistinguishable from the tubercular form in its further progress, except that the history of its origin, and the results of contraction of the fibroid tissue enable a diagnosis of this variety of phthisis to be made. There is increase of the connective tissue and contraction in *all* the forms of advanced phthisis, so that the name "fibroid" expresses the most striking pathological factor in the

* For Ziehl's modification of Ehrlich's method see Appendix.

disease rather than a special variety. Fibroid phthisis, clinically, is therefore chronic interstitial pneumonia (cirrhosis of the lung) *plus* tubercles and formation of cavities; or slowly developed tubercular disease *plus* great increase of fibrous tissue.

Miliary tuberculosis is a general disease affecting many organs. As there is always an infective source (sometimes in the lung itself), and from the liability of the apices of the lung to be affected and the case ultimately to become one of tubercular phthisis—it must be included in the phthisis group.

The most prominent symptoms are the pyrexia and the dyspnoea. The onset is generally sudden. The temperature ranges from 102° to 105° Fahr., and often it is peculiar in being higher in the morning than in the evening (but sometimes there is little elevation of the temperature during the course of the disease). The skin is covered with sweat during the remissions of the temperature. The breathing is very hurried, and often there is a lilac or purple appearance of the lips, cheeks, fingers, and nails—the latter appearance suggesting tuberculosis, if no heart disease or emphysema be present to account for the venous obstruction. The cough is short, dry, and troublesome, and the expectoration is sometimes streaked with blood. The tubercle bacilli are rarely found in the sputum—unless there are cavities in the lung. The physical signs are generally vague; but sometimes slight dulness is made out at the apices. A soft friction rub has been described, which is supposed to arise when the tubercles are superficial. “Consonating” râles are sometimes heard. The physical signs of bronchitis are often present, and these obscure the finer râles. A further means of diagnosis lies in the examination of the choroid which may be affected by the tubercles. The disease may terminate in the development of typhoid symptoms and death, in two, to four, or six weeks; or the case may become chronic, and the patient phthisical. The presence of tubercles in other organs—as in the brain, with the symptoms of meningitis; or the presence of jaundice when the liver is affected; or pains in the joints, &c.—may help to a correct diagnosis.

Physical Signs of Phthisis.—In the early stages the signs may be very doubtful, and repeated examinations, at intervals, may be necessary before a positive diagnosis can be made. Amongst the earliest signs noticeable are diminished mobility and slight flattening at the apex of the affected side. The vesicular murmur may be deficient or absent, but the plugging of the bronchial tubes with mucus may be the cause of this, and the patient should be instructed to cough during the examination. Instead of deficiency, there may be harshness with prolonged expiration, limited to the apex. This should not be mistaken for the *puerile* breathing found on the sound side, and compensatory to a consolidation of the opposite side. A “jerky” or “interrupted” form of breathing, limited to the apex, is also suggestive of early phthisis, the irregular opening up of the small bronchioles and air vesicles, from adhesion of their surfaces, being the cause of this sign. It should be noted that the heart’s impulse may give rise to this jerky breath-sound, and in very

nervous people it may occur without organic disease being present. A few moist râles limited to the apex, or even one little “clinking” râle—brought out by coughing during the examination—is highly suggestive of incipient phthisis, when associated with one or more of the symptoms. The vocal resonance is increased if there be sufficient consolidation, and percussion reveals dulness if the disease be sufficiently advanced. The normal breath-sounds heard under the right clavicle, and at the level of the fourth dorsal vertebra behind, are not to be mistaken for exaggerated breath-sounds due to consolidation.

As the disease advances the signs become more marked. The mobility of the chest becomes still more diminished, and the flattening and hollowing above the clavicles are more obvious. *Palpation* also reveals the diminished mobility, and if, at the same time, the *vocal fremitus* be tested, it will be found to be increased. The cyrtometer, in very advanced cases with shrinking of the lung from contraction of fibrous tissue, reveals diminution of the affected side; but the same thing happens in old cases of pleurisy. *Percussion* reveals dulness over the consolidated parts. *Auscultation* may discover the form of breath-sound to be bronchial, tubular, amphoric, or cavernous; and the vocal resonance to be increased (bronchophony). The “metamorphosing murmur of Seitz” is a murmur, very harsh during one-third of the inspiratory part and then replaced by bronchial breathing in the remaining two-thirds. It is sometimes heard when there is a cavity. In large cavities whispering pectoriloquy is often heard, although it is not absolutely diagnostic of a cavity. It is sometimes *not* heard when a cavity is present. Post-tussic suction or “india-rubber ball sound” (*Mitchell Bruce*) consists of a “high-pitched sucking inspiratory sound immediately following the forced expiration of cough.” The *accompaniments* vary from simple crepitations (fine and coarse, or consonating), to the large mucous *gurgling* râles heard in cavities.

The heart-sounds are sometimes very distinctly heard, owing to the consolidated lung being a better conductor; and a systolic pulmonary murmur is often present, due to compression of the pulmonary vessels by the consolidated tissue around them. The percussion dulness is never so absolutely toneless as in pleuritic effusions. The “cracked-pot” sound (*bruit de pôt fêlé*) is frequently elicited in percussing over a cavity. It should be noted, however, that it is sometimes heard on percussion over effusions and in pneumonia; or on percussion over the chest of a screaming child. Comparison of the state of the two lungs is more difficult when both lungs are affected, the normal standard of comparison being lost. In the very chronic fibroid forms of phthisis there is great dulness and shrinking; and the heart, stomach, or liver may be drawn up by contraction of the fibrous tissue.

The prognosis in phthisis is always grave; but as regards the duration of life it is almost always doubtful, and it should be very cautiously given. A severe acute phthisis may become chronic; and the prognosis in the third stage may be more safely given than in the early stages, as the disease is more likely now to remain