the inoculation of the cornea with tubercle-matter, and the production of general tuberculosis, when no other kind of matter effects such results. This is, indeed, regarded by Cohnheim as the *experimentum crucis*—as proving beyond doubt the specific infective property of true tubercle.

Such was the position of this subject when Koch's remarkable discovery was announced. By a special method of preparation and staining, he was able to demonstrate a specific bacillus—the *Bacillus tuberculosis*. This parasite has great vitality, and therefore easily resists ordinary destructive agencies. It is contained in the sputa, in the matter of cavities, in the giant-cells of tubercle, etc. The diagnosis of obscure cases may depend on the recognition of this organism. The number of bacilli is in direct ratio to the intensity of the destructive process, according to Balmer and Fraentzel (*Berliner klinische Wochenschrift*, No. 45, 1882). Hence, when the disease is slow or is stationary, the number of these bodies is small, and those present contain no spores. It appears, also, that if the bacilli are found in great numbers in the sputa, which is a favorite soil for their growth, they are less numerous in the walls of the cavities. If the fever is slight, the bacilli are imperfectly formed. Not only are they contained abundantly in the sputa, but also in the expired air of phthisical subjects. The presence of air is not necessary to their growth and development, for they exist in closed cavities of strumous and tubercular abscesses. The relation of these bacilli to tubercle is further shown in the fact that they have been found in various forms of tubercular ulceration, and in all parts, the seat of this process.

Although the bacillus tuberculosis has been widely accepted, opposition is developing in various quarters. Especially has this opposition to Koch's views taken form in Vienna, and hence something must be allowed to the jealousies which have existed between the two cities. Dr. Spina, who has long held the position of chief assistant to Stricker, may therefore be supposed to be fully acquainted with the technique of the processes. He opposes Koch at all points, and maintains that the bacillus has not a constant form, but varies with the tissue and local condition; that it is not essential to the tubercular process, and that it is frequently absent in undoubted tubercular disease. From the practical side, Koch's theory has received a severe blow in two cases which have recently occurred at Nothnagel's clinic. In both tuberculosis was diagnosticated, because the bacilli were detected in the sputa. Yet, on *post-mortem* examination, both were ascertained to be examples of bronchiectasis, and no tubercles existed at any point. It follows, from these facts, that the parasitic nature of tubercular phthisis must be regarded as *sub judice*. The attitude of the reflecting physician should be that of receptivity, but he should not rush to the conclusion that the parasitic nature of phthisis is proved.

Given a specific bacillus, the communicability of phthisis would thereby seem to be established. Before this discovery the evidence of its transmission, especially from husband to wife, had been accumulating. Indeed, a belief in the contagion has prevailed to a less or greater extent from the earliest times. In Southern Europe and in North Germany, especially during the last century, this belief was almost universal. In a treatise on consumption, by Dr. Morton, published in 1694, and in Dr. Young's work on the same malady, which appeared early in this century, this view was strongly maintained. In England, various writers have alluded to this subject, and by several of eminence the fact of the contagious nature of phthisis has been considered established by the clinical evidence. Thus, Dr. Walshe, who originally opposed the belief in its communicability, subsequently practically admitted it. In 1867 Dr. Budd published a paper in the "Lancet," in which he maintained that the disease is communicable, and that the tuberculous matter in the sputum contains the specific, infective material. Whether the bacillus of Koch continue or not to be regarded as the agent of infection, it must be admitted that strong reasons exist for adhering to the view of the contagious character of tuberculous phthisis. But, as every germ needs a special soil for its growth, so the specific infective material of consumption must fall into a properly prepared organism, to proceed to full development.

If the bacillus tuberculosis is to be found in all suppurative inflammations of tubercular origin, we have in this fact an explanation of the occurrence of pulmonary tuberculosis as secondary to catarrhal pneumonia, to pleuritis, to glandular affections in various situations, etc. The conditions of its spread from a point where it first appears, the manner of its propagation from one individual to another, and the descent by inheritance, are points of which we have no certain knowledge. Especially is it difficult to reconcile the fact of heredity with the present knowledge of the behavior of the parasite.

Pathological Anatomy.—The miliary tubercle is a grayish-white, translucent, and semi-solid granulation, about the size of a millet-seed, composed of a reticulum, with cells, giant-cells, and nuclei, the cells resembling white blood-corpuscles except that they are smaller, and the giant-cells having many nuclei. The reticulum is an extremely delicate network, inclosing the cells in its meshes, the giant-cells being placed nearly at the center of the granulation. The specific element is a minute, flat, long bacillus, rounded at its extremities, straight or curved, and beaded in appearance. Bacilli are usually single, but are sometimes found in pairs, and they multiply from spores, apparently only in the body, since they require a high temperature for their growth. They are found in the cells of tubercle nodules or masses, especially in the giant-cells. Tubercle, then, according to the bacilli theory, consists

in the deposit and pullulation of the bacillus tuberculosis, and it is the matter thus formed which is deposited in the lungs, and constitutes pulmonary tuberculosis. According to Rindfleisch, tubercle takes its origin from the connective-tissue cells of the blood and lymph vessels, and the first deposits occur at the point where the bronchioles unite with the acini. (A group of acini communicating with a bronchus is a lobule.) A whitish nodule—a tubercle granulation—is thus formed around the termination of the bronchiole in the acini, in the angle at their point of junction, the deposit being in the connective tissue. The nutrient vessels are included in the granulation, and their adventitia become swollen and infiltrated. It is this development of tubercle in the connective-tissue cells of the adventitia that weakens the vessel, and which may finally cause a rupture and hæmorrhage. So many vessels at the apex are occluded by the mass of the deposits, that the pressure in the remaining vessels is much increased. When the walls of the vessels are infiltrated, rupture occurs the earlier by reason of the increased pressure from the cause just named. Tubercular deposition also takes place abundantly in the bronchioles, not only those in immediate relation to the lobules, but for some distance beyond. The lymphatics distributed to the mucous membrane are infiltrated, and next those of the peribronchial space, so that all around the alveoli and bronchioles are thickly placed masses of tubercle granulations. The intervening connective tissue is also densely infiltrated. With the deposit of tubercle, there are associated the results of inflammation excited by the presence of these granulations. According to Rindfleisch, a desquamative pneumonia plays an important part in the subsequent changes. The cheesy transformation of the products of catarrhal pneumonia, atelectasis, bronchial dilatation, assist materially in enlarging the area of structural changes. The masses of miliary tubercle, in a variable period after their deposition, and often within a few weeks, undergo a cheesy transformation, by which they are brought into close resemblance to the cheesy products of caseous pnuemonia. It is a process of fatty degeneration, beginning in the central portion of each nodule. In acute tuberculosis, to be studied hereafter, the gray granulation is disseminated throughout both lungs. In the pulmonary tuberculosis, the deposits occur chiefly in the superior lobes, and are often limited to the apex, but are very rarely indeed confined to one lung, and, when this is the case, the left is more often attacked than the right. When the process of cheesy transformation is completed, the resulting mass is opaque, yellowish, and has the friability of cheese. The infiltration of all the parts, ultimately, of which the parenchyma of the lungs is composed, the closure of the vessels and entire arrest of the nutritive supply, and the compression exerted by the contracting connective tissue, necessarily cause a necrosis of the pulmonary elements. When the stage of softening comes on, the products, although having a puriform appearance, are not purulent. Inflammation and suppuration are excited in the tissues, with the necessary result of disintegration. On the surface of the mucous membrane the destruction of the tissue in and about the site of the tubercle granulations is an ulceration; in the mass of disease in the body of the lung the destruction of tissue produces a cavity. The fluid matter resulting from the softening of the yellow tubercle is homogeneous, of the consistence of cream, and having a greenish-yellow or grayish color. Mixed with it are necrosed pulmonary elements, solid particles of a yellowish color, and the whole is contained in a small cavity, surrounded by masses of cheesy tubercle. The softening proceeds from the center to the periphery, and in its progress the pulmonary elements are disintegrated with it. When discharge of a cavern takes place by the ulceration opening a bronchus, or, according to Rindfleisch, by the tubercular ulceration of a bronchus, the elastic fibrous tissue may be recognized in the sputa. Large caverns are formed by the breaking down of the intervening septa and the coalescence of smaller ones. The increase in the area of destructive ulceration is greatly promoted by the attacks of catarrhal (desquamative) pneumonia, which induce softening and dilatation of the bronchi, and collapse of lobules (atelectasis); and catarrhal products fill the alveoli and bronchi, and there caseate. Cavities are produced under these circumstances by the softening and extrusion of the caseous masses as described under the head of caseous phthisis. In this case the tubercle granulation is the exciting cause of the catarrhal pneumonia; in the former the products of catarrhal pneumonia undergo the caseous change in consequence of a peculiar "invulnerability" of the constitution, without which the catarrhal products would pass through the ordinary changes. Dilatation of the bronchi, or bronchiectasis, plays an important part. In catarrhal pneumonia, the walls of the bronchi yield in consequence of an extension of the inflammatory process to them, and, as the existence of dyspnœa renders greater inspiratory efforts necessary, and as the area for the admission of air is much reduced, obviously the interbronchial pressure is raised, so that greater force is exerted against the weakened tubes. According to Rindfleisch, the walls of some cavities are in part formed by dilated bronchi. Cavities, still extending, have no proper boundary, and are surrounded by tubercle and caseous masses undergoing softening, and by detritus of the lung-tissue. Others are lined by a connective-tissue membrane, which continuously pours out a puriform matter of a greenish-yellow, often having a foul odor by reason of decomposition from the presence of air. When the cavity is recently formed, not only are its sides ragged and uneven, but large bands traverse it, remains of pulmonary tissue not destroyed. Other organs besides the lungs are affected. The *pleura* is usually the seat of a chronic inflammation; it may take the form of a dry pleurisy, and close adhesions form universally, so that the cavity

is obliterated ; or the adhesions may be local and partial when they are chiefly at the apex ; or a neo-membrane is formed, and both the pleura and the new membrane may become tuberculous. Extensive effusion may be formed in consequence of the rupture of a cavity and the escape of its contents, when a pyopneumothorax results. A cavity perforated and firm adhesions having formed, the pleura may ulcerate and discharge take place through the thoracic parietes, a fistula remaining. The *bronchial glands* enlarge by hyperplasia of their contents, which undergo caseation. They may be dry and cheesy, or suppurate and discharge, the pus finding an exit by the trachea, or by a bronchus, or by the œsophagus. In infants and children, enlarged bronchial glands may compress the trachea or bronchi, or the pneumogastric, and thus give rise to suffocative attacks. It may be well to mention that the late Dr. Fuller, of London, had secondary pyæmic abscesses of the brain, from suppurating bronchial glands. The *larynx* always suffers from some morbid change in pulmonary tuberculosis. From simple hyperæmia up to extensive tubercular ulcerations, destroying the epiglottis, vocal cords, etc., there are numerous gradations in the severity of the lesions. Tubercular ulcerations also occur in the œsophagus, stomach, and intestines, but the point of greatest development of the ulceration is the lower part of the ilium and the large intestine. The tubercular troubles of the intestinal canal are found in two stages : the initial deposit, and the softening and destruction of tissue or ulceration. The peritoneum is granulated, and chronic lesions of the peritoneum coincide with the formation of ulcers in the intestine. The liver is usually in an advanced stage of fatty degeneration, but in rare instances the change is that of amyloid disease. In the kidney, the amyloid degeneration is more common than the fatty. Tubercular ulcerations are often found all along the urinary tract.

Symptoms.—There is a peculiar type of constitution, as a rule, associated with tuberculous phthisis, which, being present, may serve to excite suspicions, at least, in obscure and doubtful cases. These peculiarities are observed in growing youths and young men, and may be described as follows : They are tall and rather thin ; the neck is long and small ; the thorax flat, narrow, and having but little expansile mobility ; the muscles, especially of the chest and neck, are thin and poorly developed ; the intercostal spaces are wide ; the hair is fine, the eyelashes long ; the eyes are large and bright, the sclerotic glistening ; the skin is transparent and thin, the color quickly changes, and the veins are blue and distinct ; the fingers are long and tapering, but their extremities are incurved or club-shaped. These subjects possess certain moral and mental characteristics also : they are impressionable, the disposition is variable ; they are fond of activity, but fatigue easily ; others are more phlegmatic, speak slowly, and differ in complexion, being dark, with thick, muddy skins. When these peculiarities of constitution coexist, with an hereditary tendency to phthisis, they possess a high degree of significance. In such subjects, cough, losing flesh and strength, with a red line along the margin of the gum, are strongly indicative of the onset of phthisis, even when the physical signs may not be positive. A large proportion of the cases begin by loss of appetite, indigestion, decline in weight, without cough or any symptom referable to the lung. In women these symptoms are accompanied by disorders of menstruation. Again, an attack of hæmoptysis may be the first symptom. Most usually, the onset of the disease is characterized by a short, dry cough, which is rather more troublesome at night, preventing sleep, some shortness of breath, pains in the chest, either wandering or fixed in the position of an intercostal nerve, or a sharp stitch indicative of pleurisy, some nocturnal perspiration, confined at first to the neck and face, decline in flesh and strength, poor appetite, and often, more or less diarrhœa. At this period, too, some alteration of the voice is beginning to be perceptible and bronchial hæmorrhage occurs. The progress of the case is more rapid if the fever now appears. This may be an early symptom ; it may be postponed until the period of softening. The action of the heart is excitable and is accelerated by slight causes from the very beginning, and the pulse is soft and compressible, the tension of the vessels being low. The usual type of fever in the beginning is the quotidian. There is a daily morning remission, an evening exacerbation terminating in a sweat—the co-called *hectic fever* (*septicæmic fever*). The type may be double quotidian—two paroxysms of fever each day—the first in the morning, the second at night. The range of temperature at this period is not great, the minima about 98° Fahr., the maxima 102° Fahr. The range of fever-heat is an important indication of the degree in which the morbid processes are proceeding, especially those involving the lungs. In illustration of this may be mentioned *phthisis florida*, in which the highest temperature of this disease is attained because of the immense extent of the caseous deposits undergoing softening and extrusion. As the case proceeds, all the rational signs become aggravated. The appetite is almost gone ; in severe paroxysms of coughing, in the last straining effort to dislodge the sputa, vomiting is excited, an accident very apt to occur after meals. The diarrhœa also increases, and becomes very difficult to restrain. The cough, also, grows more troublesome and painful, the expectoration more abundant, and the voice harsh and husky. Difficulty of swallowing comes on in consequence of ulceration of the epiglottis, and sometimes the attempts at swallowing are embarrassed by the dropping of particles of food and drink into the glottis, exciting violent suffocative attacks. The expectoration assumes a different character at various periods. At first there is brought up, often with a great deal of effort, some frothy mucus ; after a time the sputa become purulent or muco-purulent, greenish or greenish-

yellow in color, without air, and without viscidity, unless there is a complication of pneumonia, when the sputa will have a grayish, vitreous, adhesive character, and may also present a slightly rusty aspect from the admixture of blood, or may be simply streaked with blood. These adhesive sputa may be seen in large muco-pus expectorations, as isolated particles. The sputa often have a striated appearance, at one time supposed to have much significance, but now known to be produced by the diminution of the cellular elements and the presence of deformed and atrophied cells and of granules—changes of a degenerative kind due simply to retention in the lung. A very significant element in the sputa is the presence of elastic fibers of the pulmonary tissue. These bodies are most easily detected by boiling the sputa in a solution of caustic soda in distilled water (18—100) according to the method of Fenwick. The most distinctive element is the *bacillus tuberculosis*, which is necessary to constitute tubercle, according to the modern conception. Well-developed specimens can usually be obtained from the expectorated matters, even in cases of the so-called "miner's lung." The next change in the sputa is the characteristic impressed on them by formation in small cavities. They then consist of two parts, a frothy muco-pus from the bronchi, and isolated, globular, compact masses without air, of a greenish or grayish color; when allowed to stand, the former rises and the latter sinks to the bottom, and, if put in water, sinks quickly. The quantity of expectoration varies; in the beginning, because then it is derived from a bronchial catarrh; afterward, according to the extent of the cheesy masses undergoing softening, the size of the resulting cavities, and the degree in which bronchiectasis exists. When there is a large cavity, quantities of little more than pus are expectorated. When the patient lies in a position to permit accumulation to take place, the expectoration may be suspended, but, when the position is changed, the pus is discharged in a stream. Sputa streaked with blood and rusty sputa have already been alluded to; but expectoration of blood, or hæmoptysis, is a different affair. According to some, phthisis may be due to pulmonary hæmorrhage. This notion arose from the clinical fact that hæmoptysis is sometimes the first symptom of the disease, and after its occurrence there is an immediate development of the symptoms. The presence of blood-clot is supposed to excite an irritation which has for its ultimate effect the formation of tubercle. The most generally accepted view is, that hæmorrhage is merely a symptom, and a symptom that may occur at any

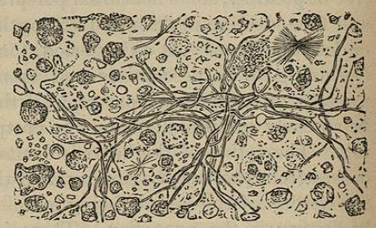

FIG. 35.—Fragment of Lung-Tissue and Sputa. (Beale.)

period. If we accept Rindfleisch's demonstration, that the formation of tubercle begins in the connective-tissue cells of the adventitia of the vessels, there can be no difficulty in comprehending the early appearance of hæmorrhage in the course of phthisis. At any subsequent period, the extension of the area of tubercle formation may be accompanied by hæmorrhage. Again, hæmorrhage, often considerable, may be due to the erosion of an unclosed vessel in the process of destruction, ending in the formation of a cavity. The amount of blood lost varies from a drachm or two to several pounds. The blood is bright colored, more or less aërated, and comes up with coughing; but a sudden large hæmorrhage may pour up in a stream and be ejected by the nose as well as the mouth. A considerable part of the blood may be swallowed, and subsequently vomited, and, as it is then acted on by the gastric juice, presents the appearance of hæmatemesis; but the history of the case, the rational and physical signs of pulmonary disease and the absence of stomachal disease will afford the data for a correct diagnosis. After the hæmorrhage has taken place, and the flow is arrested, for some days clots of small size and blackish in color are expectorated. Occasionally there are indications of the approach of a hæmorrhage, the significance of which the sufferers from them soon learn: these are a feeling of warmth in the chest, oppression of breathing, excited action of the heart, and a rather sweetish and saltish taste in the mouth. Usually, nothing in the nature of a warning of the approaching hæmorrhage is observed. When the blood-taste is experienced, the mouth should be examined, for the gums may be the source of the hæmorrhage. Bleeding from the posterior nares may also be confusing, as there may be a coincident cough. A pulmonary hæmorrhage may be vicarious of the menstrual flow, and it may be determined by the sudden arrest of hæmorrhoidal bleeding.

3. FIBROID PHTHISIS.

Definition.—By this term is intended a form of consumption characterized by hyperplasia of the connective tissue of the lung and atrophy and degeneration of its proper structure. In this respect the disease corresponds to fibroid liver, fibroid kidney, etc., but the changes do not begin in and are not limited to the connective tissue. Bronchial inflammation, bronchiectasis, bronchorrhœa, and exudative pleuritis are among the initial changes, the pulmonary tissue being involved subsequently. Ultimately tubercular deposits occur, and the lesions produced by these are added to those already existing in the connective tissue and the bronchi.

Etiology.—Heredity is concerned to the extent that the type of pulmonary tissue favorable to the development of this disease is transmitted. It is a disease of mature life, after the middle period, and is extremely rare before thirty. Next to heredity, chronic bronchitis and

a form of pleuritis characterized by extensive organized exudation are the most influential factors. The causes of chronic bronchitis are, therefore, indirectly the causes of fibroid phthisis.

Pathological Anatomy.—The mucous membrane of the bronchi is of a dark red in the more recently inflamed parts, of a slate-color in the older, traversed by dilated vessels, its glands much thickened and elevated above the general surface. The sub-mucous connective tissue is thickened, the muscular layer hypertrophied at first, but in the further progress of the case the whole tube is softened and dilated. These dilatations may be fusiform or sacculated. The latter predominate, and are often mistaken for cavities, the resemblance being the more striking if the dilatation contains an accumulation of pus. The atrophic changes in the walls of the bronchi are not the only factors concerned in producing dilatation. The force of the expiration in coughing, the contraction of the adjacent connective tissue, and of pleural adhesions, are also concerned. From the bronchi the inflammation slowly extends to the peribronchial, perivascular, and interlobular connective tissue. A hyperplasia of its constituent elements takes place, with the result to compress the vessels, the acini, and the bronchioles. The contraction of the newly formed connective tissue, by cutting off the blood-supply and encroaching on the neighboring parts of the pulmonary tissue, causes an atrophy. Some of the lobules collapse (atelectasis); all within the affected area contain less blood, and are narrowed by pressure. The collapsed lobules undergo the changes already described. In the progress of these cases catarrhal pneumonia ultimately plays a part; the bacillus tuberculosis appears in the caseous masses, and these undergoing softening and extrusion, cavities are formed. So that the cases of fibroid phthisis, although differing in their rate of progress and in the greater importance of the sclerosis to the other morbid processes, nevertheless are brought into close relation to the other forms of phthisis. A considerable increase of the connective tissue of the lungs occurs in chronic tubercular phthisis; the longer the duration of the disease, in fact, the greater is the development attained by it. The walls of the cavities are composed of a dense layer of connective tissue, closely united to the same tissue of the lung. In caseous pneumonia there is less production of connective tissue, because of the rapid progress. In a fibroid lung the cavities do not attain to great dimensions; they appear as interspaces in the dense trabeculæ. When these intervening portions of the condensed tissue are divided, they are ascertained to be exceedingly firm, of a grayish or slate color, containing here and there patches of brown pigment, and possess but little vascularity. The early compression and closure of the vessels is a source of mischief to the heart. The pulmonary circulation being obstructed over a considerable portion of the lung, the right cavities yield to the increasing pressure and dilate. There is, therefore, a stasis of the venous circulation; the liver enlarges, and

ascites is produced; the kidneys are congested, and albumen is present in the urine. These complications develop toward the close of the malady.

A considerable proportion of the cases of fibroid phthisis originate in "dry pleurisy"—characterized by the formation of extensive solid exudation. Three varieties of this form of pleuritis are recognized: the fibrinous, the croupous, the proliferative;* but it is probable the three processes are mixed in every case. An extension of the pleuritis takes place by contiguity of structure; the connective tissue of the lung is entered by the interlobular fissures; the peribronchial and perivascular connective tissue is invaded and undergoes proliferation, and then the alveoli become affected. Thus the morbid process comes to involve all the pulmonary elements. At first a general congestion of the affected parts ensues; then the exudative process occurs, and at length the deposit and pullulation of the bacillus are manifested by caseation, ulceration, and extrusion of the products of its development. The general course of the structural alterations of the pulmonary parenchyma is as already described.

Symptoms.—Fibroid phthisis is the most chronic form of the disease; its early history is that of bronchial catarrh, or of dry pleurisy of the exudative form; and it is not until after months, even years, that, extension taking place to the lungs, the progress becomes more rapid. For months there is merely a dry cough, not very troublesome, but persistent. The expectoration is slight, and is nothing but mucus. The appetite is but little impaired, and the weight and strength are not materially reduced. During the fall, winter, and spring months the symptoms increase in severity; the cough becomes more troublesome, and the expectoration more abundant and having the appearance of muco-pus. The symptoms ameliorate during the warm months, but to increase again with the changeable weather of winter. After two or three years of this alternation, there is less and less improvement in the warm months, but the symptoms of catarrh continue throughout the year. Fever comes on toward evening, the temperature at first rising to 100° Fahr. The appetite lessens, digestion becomes poor, and the body-weight progressively declines. The cough is harassing and prevents sleep; the expectoration becomes more profuse and entirely purulent; and the food now and then comes up in the attempt to clear the larynx and fauces. Some difficulty of breathing is experienced; the pulse is small and weak; the skin is warm toward evening, while slight chilliness is felt in the morning, and sweating occurs during the night. As the disease advances, the temperature reaches 101° and 102° in the evening, but it does not attain to the altitude reached in caseous or tubercular phthisis. When the bronchi dilate, the expectoration becomes profuse, especially in the morning—

* Sir Andrew Clark's "Lumleian Lectures," "Lancet," vol. i, 1885.