action of bacilli, and these might pass through it without exerting any influence, the tuberculous tissue being formed only where this immunizing influence is not felt. A somewhat different action of the bacilli is seen when numbers of them are distributed over a surface the cells of which can take part in the formation of the tubercle, as in the lung. When the bacilli are injected into the trachea the cells lining the alveoli proliferate and the alveoli become filled with large desquamated cells and giant cells.

A more important influence exerted by the bacilli is the production of exudations. The specific tissue changes are in themselves relatively unimportant; except in cases of acute miliary tuberculosis or in cases of tubercle formation in organs essential for life, the specific tissue lesions are rarely sufficient to destroy life. The bacilli can lead to the production of a serous, fibrinous, or purulent exudation. The formation of such exudations is almost inseparable from the action of the bacilli, and may predominate over the tissue formation. I have seen a case of acute miliary tuberculosis in a child, in which vast numbers of bacilli were found in the lesions and in the vessels without changes in the tissue around them, where the miliary foci in all the tissues really represented miliary foci of exudation. The tubercles, particularly in the liver and spleen, appeared as foci of fibrin. These exudations are seen particularly in organs whose structure permits a great multiplication of the bacilli, as in the lungs, where the bacilli can grow in the walls of a cavity, as on the surface of a culture tube. The ease with which exudation can take place in an organ like the lungs or in a serous cavity facilitates this action of the bacilli. They may be considered among the true pyogenic organisms. Experiments have shown that the subcutaneous injection of avian bacilli into an animal usually leads to the formation of an abscess. The distinctly pyogenic action seems to take place when great numbers of bacilli are present, and when they have little power for the production of those chemical substances which lead to caseation. I have seen small definite abscesses in the lung due to purulent exudation and liquefaction of tissue in which the polynuclear leucocytes were filled with tubercle bacilli. Neither the exudation nor the surrounding tissue showed any caseation. This suppuration must be distinguished from the formation of puriform material due to the liquefaction of caseous tissue and the mingling of the broken-down granular fatty mass in a serous fluid. This is seen in the contents of a psoas abscess, and in this the tubercle bacilli may not be demonstrable. In the meninges the bacilli lead to the formation of an exudation which is chiefly fibrinous, which contains various numbers of polynuclear and epithelioid cells, and which is difficult to distinguish from that of other forms of meningitis. The exudation undergoes caseation just as the tuberculous tissue does. It is difficult to avoid the conclusion that such an exudation maybe due to the local action of the chemical products of the bacilli.

Tuberculous ulcers may be produced either in the skin or in the mucous membrane. They may be the result of an infection extending from the surface or taking place in the tissue beneath. The process of their formation is a simple one, and due to the formation of tubercles or tuberculous tissue, with subsequent caseation and liquefaction of the caseous tissue. The ulcer increases in size by the continued formation of foci and their subsequent softening. This gives to the base of the ulcer, which is usually undermined at the edges, an irregular, eroded aspect. As the lymphatics afford the easiest route for the diffusion of the bacilli in the tissue, the ulcer usually enlarges in the direction of the lymphatics. The tuberculous ulcers of the intestine tend to enlarge transversely to the axis, and girdle ulcers, extending completely around the intestine, may be produced.

From a single focus the tubercle bacilli may be carried to other parts of the same organ or throughout the body. The distribution can take place by means of the blood, by the lymphatics, or by natural channels which connect a tuberculous focus with other parts of the organ, as the bronchi in the lung, or with other parts of the body, as the intestine. These methods of distribution give rise to special forms of tuberculosis.

Distribution by Blood.—A few bacilli probably enter the blood in all cases. The lesions which are produced in the different organs are due to the character of the circulation, which in some organs peculiarly facilitates the deposit of insoluble substances suspended in the blood, and to the character of the tissue, which may or may not facilitate multiplication and action of the bacilli. A careful microscopic examination of the liver will show the presence of a few miliary tubercles in almost every case of tuberculosis. The circulation in the liver, giving an enormous capillary stream bed with slow movement of blood in the capillaries, facilitates deposit of suspended particles just as a large body of water facilitates sedimentation. Miliary tubercles are never found in voluntary muscles because the tissue offers unfavorable conditions for their development. There is no other way of explaining the presence of lesions in bones than by blood distribution. It is also possible that tubercle bacilli may enter into the blood without the production of a primary focus. How these few bacilli enter into the blood stream is a matter only of conjecture. In most cases they probably come from a tuberculous lymph node, and in these the lesions may be so slight as to elude observation. When the tubercle bacilli enter into the blood in larger numbers they may be deposited in single organs, determined by conditions of the circulation, or they may be generally distributed, producing general miliary tuberculosis. In this, careful examination of the body will almost invariably show the source of the bacilli either in a tuberculous focus in a blood-vessel or in a tuberculosis of the thoracic duct. The exceptions to this are so few that we are warranted in believing that such a source existed but escaped detection. The tuberculosis of the vessel can result either from the infection of the intima or from the extension into the vessel of a tuberculous focus adjoining it. The former is the more common. In both forms the tuberculosis appears as a thrombus formation. A slow thrombus formation takes place at the point of infection, and in the thrombus the tubercle bacilli grow as in a culture medium. The vessel may be completely occluded, but generally the lumen is open and the thrombus may be partially covered by the endothelium. Such thrombi are most frequently found in the pulmonary veins, and may extend a considerable distance, two or more centimetres, along the wall. They may be distinguished from ordinary thrombi by their opaque white appearance. Microscopic examination of the thrombus shows a finely granular or homogeneous mass, denser in places, not unlike ordinary caseation. In tuberculosis of the thoracic duct the infection can also take place from the intima or by the extension of an adjoining focus into the duct. The duct may be converted into a solid caseous cord, or the lesion may be mural. To the theory of general blood infection by such foci various objections have been raised. Sections of the thrombus may contain very few bacilli, and it may appear so firm that apparently the bacilli contained in it have no opportunity to enter the blood. It is true that sections of the thrombi rarely show large numbers of bacilli in immediate contact with the blood-stream. The bacilli were always present in the cases examined by me, and in some cases there were great numbers of them. To show the absence of connection between the bacilli in the thrombus and the blood stream, it would be necessary to examine all parts of it by serial sections. Moreover, it is only necessary for the focus to have been a source of infection; it need not be one at the time the autopsy is performed. We often find all the tubercles at about the same stage of development, showing that the mass of bacilli producing the lesions have entered the blood some weeks previous to the autopsy. The source of the bacilli may be a very unusual condition. In one case of extremely acute miliary tuberculosis in an adult the only old lesion apparent on first examination was a small caseous focus

in the apex of the lung, which seemed to be entirely healed. A closer examination of the veins of the lungs and of other parts showed no source of the bacilli in them. On the examination of the aorta a few miliary tubercles were found at a point forming a line leading up to a small opening in the aorta which was at first considered to be the mouth of an intercostal artery. On slitting this up it was found to be a minute aneurism of the vessel, which was filled with a caseous mass more or less broken down and containing great numbers of bacilli. The aneurism was the result of an infection of the adventitia of the artery, the caseous degeneration extending from this up through the media, which had given way before it. The intima extended a short distance into the aneurism. Although it may not be possible to find the source of the bacilli in a vessel or in the thoracic duct, I have never seen a case in which such lesions were not associated with miliary tuberculosis. The source is not only shown in this way, but it is also evident from a careful examination of the tubercles themselves, which will very often be found in relation to the vessels. The miliary tubercles are never formed in certain places, and are but rarely found in others. They are never found in the voluntary muscles, nor in the skin. It is very rare also that any are found in the mucous membranes. Even in the mucous membranes of the alimentary canal, which is so liable to infection from the lumen, we but rarely find any eruption of the nodules. Whether there is any multiplication of the bacilli in the blood in these cases is uncertain, although one occasionally finds cases in which from the number of bacilli this conclusion seems almost inevitable. I have seen such a case in a child in which great numbers of bacilli were found in the capillaries of the liver, both free and enclosed in epithelioid cells, either within or attached to the wall of the vessel. In another case almost an injection of the capillaries with bacilli was found in the liver of a cat. Cases of chronic miliary tuberculosis are seen in which the tubercles are larger and more advanced in caseation. It is usually considered in these cases that small numbers of the bacilli are constantly entering into the blood stream. It is possible that an advanced tuberculosis of a lymph node may give the necessary condition without our being abie to follow the bacilli through the sinuses and into the blood stream. When the disease has once been established, the number of bacilli in the blood may be added to by their multiplication in the numerous miliary tubercles in the walls of the vessels.

It is a rule without exception that, wherever the seat of the tuberculous lesions may be, the lymph nodes which receive the lymphatics from the affected regions are tuberculous. In the tuberculous tissue the bacilli are in close relation to the cell spaces and the lymphatics of the tissue. They are carried to the lymph nodes by the lymphatics, either free or enclosed in cells. The lymphatic vessels may be affected and converted into white cords filled with caseous material containing bacilli, or tubercles may be formed within them. Such a lymphangitis is often seen in the lymphatics extending from a tuberculous ulcer of the intestine to the mesenteric nodes. The nodes form a barrier to blood infection; they retain the bacilli in their sinuses. This barrier ceases to be when the node itself is infected, and tubercles develop in the tissue of the sinuses, from which bacilli pass into the blood. Miliary tubercles are formed in the nodes, either in the sinuses or in the follicles. The entire node may be almost filled up with typical miliary tubercles or by the formation of diffuse tuberculous tissue. In other cases we find the nodes enlarged, partly or wholly caseous, and without any evidence of specific tissue formation. Such a process seems very similar to the caseous pneumonia in the lungs. Few or no bacilli may be found in the caseous tissue, and it seems most probable that the caseation is due not to the action of the few bacilli *in loco*, but to the action of toxins which are formed by the disease in the lymphatic territory, and which are carried to the node and continuously act upon it. The lymphatics also play an important part in the distribution of the bacilli around the focus of infection. The infection of tissue leading to the formation of a conglomerate tubercle takes place by means of the lymphatics. Careful examination of the lungs in tuberculosis will often show tubercles or caseous tuberculous tissue in both the periarterial and the interlobular lymphatics. The infection of the large serous cavities of the body, particularly of the pleura, which is so often the apparent primary focus of the disease, takes place by means of lymphatics. The interlobular lymphatics of the lung pass to the pleura, entering into the subpleural lymphatics, which communicate with the pleural cavity. Tubercle bacilli can enter these lymphatics either without producing any lesion, in the same way that the dust particles can enter the lymphatics, or the lymphatic and pleural infection can be secondary to an inconspicuous primary lesion in the lung. The tuberculous infection can extend just as carcinoma often does in a direction opposite to that of the usual lymph current. There are innumerable anastomoses, and the direction of current may be changed by occlusion of certain vessels.

No other way of extension has the same importance as that along natural canals. These offer the readiest means not only for an extension of the disease in the organ affected, but also for carrying it to other organs. The bacilli find good opportunities for growth in these canals. In tuberculosis of the kidney we may find tubercles containing great numbers of bacilli which are growing in the peculiar spindle-shaped masses which are found at the edges of pure cultures.

All these modes of extension and the interrelation of all tuberculous processes can be studied to better advantage in the lung than in any other organ. I shall give a short account of tuberculosis of the lungs, as the disease here is most important both clinically and pathologically. In other organs the study of the lesions is much simpler. The lungs may be primarily or secondarily infected. Infection takes place by means of the bronchi or blood-vessels. The lymphatics play only a minor part in the extension of the disease in the lungs. In no other organ of the body does exudation become so prominent a part of the lesions. It is difficult to separate clearly the lesions produced by blood infection from the infection coming from the bronchi. The blood infection leads to the formation of miliary tubercles, starting in the interalveolar septa and in the other interstitial tissue of the lung. The epithelium of the alveoli takes part in the formation of the miliary tubercle, and bacilli are contained not only in the tissue but in the interior of the alveoli as well. With the entrance of the bacilli into the alveoli the conditions making bronchial infection possible are given. Exudation also takes part in the process, and fibrinous, serous, or hemorrhagic exudation may be found in the surrounding alveoli. This condition is more often found, and to a greater extent, in children than in adults, and it may be impossible to distinguish miliary tubercles coming from blood infection from miliary foci of tuberculous pneumonia coming from bronchial infection. The infection by the bronchi is of infinitely greater importance than infection by the blood-vessels. The primary infection of the lungs takes place in most instances by inhalation, the bacilli in the dust or in the spray reaching the tissue by the bronchi. This is shown by the predominating situation in the apices of the lungs. A great many reasons have been given in explanation of this, without any of them being satisfactory. So far as can be determined by the situation of other lesions due to solid substances conveyed by the blood-vessels into the lung, this situation of the primary lesions in the apices does not speak in favor of blood infection. The tubercles in miliary tuberculosis are more numerous in the lower lobes of the lung, and abscesses and infarctions are also more apt to be found in other places than in the apices. There can be no doubt that the primary infection may be hæmatogenous, and the primary lesions are frequently found in other parts of the lung than in the apices. It is rarely possible to see the early stages of a primary tuberculous lung. We fre-

quently find either an old caseous focus with cicatricial tissue around it, or a focus of slaty induration with radiate cicatrices extending into the tissue; or, rarely, in cases of death from intercurrent disease we may find an earlier condition. I once found in the apex of the right lung of a young man a single focus of tuberculous pneumonia 0.5 cm. in diameter, which differed in no respect from the foci which are so common in advanced tuberculosis, and which must be referred to bronchial infection. Microscopic examination in most cases enables us to determine the character of the old lesions, and one certainly gets the impression that they are pneumonic in character.

It is rare that we find lesions that can be regarded as definitely healed. Around the old caseous foci single tubercles will be found outside of the cicatricial tissue showing a slow infection, probably by means of the lymphatics. In the cicatricial tissue we shall generally find fibrous tubercles which still show some remains of specific structure, or their situation may be shown by calcareous or hyaline foci. Probably in most cases the primary infection is followed by a period of quiescence, in which the process is slow or arrested. The bacilli increase in number, and infection of the surrounding tissue through the lymphatics and bronchi takes place, leading to new foci, which become united into a single caseous mass by the advance of the caseation in the primary focus. There is usually no marked advance of the process until the softening of the caseous tissue gives opportunity for an extensive general infection by the bronchi. There is still much uncertainty as to the immediate cause of the softening. It may be the result of the formation, in the caseous tissue, of a ferment which liquefies it in the same way that the liquefaction of the exudation in lobar pneumonia is brought about. Or it may be due to the action of other bacteria. The softening takes place either in the middle of the focus, or in one or several places, or at the edge, a line of softening separating the caseous mass. I have seen such softening take place both in the lymph nodes and in the lung. In one case there was complete sequestration of a caseous mass as large as a lemon, which lay in a cavity filled with thin purulent material. All around the edge of both the sequestrum and the lung there were great numbers of both tubercle bacilli and streptococci. With the expulsion of the softened mass there remains a cavity in the tissue. The cavity is usually surrounded by a zone of granulation and cicatricial tissue, in which numerous tubercles are found. The extent of cicatricial-tissue formation indicates the rapidity or slowness of the local extension. The interior of the wall may be covered by a layer of caseous tissue of varying extent, or it may have the soft velvety character of the wall of a chronic abscess. Tubercle bacilli are often found growing in masses in the wall with the same form of growth as in a pure culture in a test tube.

The further infection of the lung by means of tubercle bacilli or their products, carried into other parts of the lung by way of the bronchi, gives rise to several forms of the disease.

Tuberculous Bronchopneumonia.—This is due to the action of the bacilli. The foci of the disease are very similar in their situation and extent to the foci of bronchopneumonia met with in the infectious diseases of children, and due chiefly to streptococci. As in the case of the latter disease tuberculous bronchopneumonia has a relation to Miller's lobule of the lung. The infection begins at the bronchial termination in the atrium, and from this extends into the air sacs and air cells. Macroscopically, these foci have the appearance of miliary tubercles. Microscopically, the bronchial passage, the atrium, and parts of the air sacs contain leucocytes, often a small amount of fibrin, and large epithelioid cells. Some caseation of the contents is always found, but it need not be extensive. We may find the infection taking place not at the entrance of the bronchus, but at the bottom. At this point, however, the tubercles can scarcely be distinguished from those arising from blood infection.

Tuberculous Bronchitis.—Either in connection with bronchopneumonia or independently of it there may be an infection of the wall of a bronchus, due either to infection from within or to the extension into the bronchus of a tuberculous focus in the adjoining air cells, just as in the case of the tuberculosis of the vessels. The bronchus at this point loses its epithelium wholly or entirely; it becomes filled with an exudation which later becomes caseous. On section it appears as a round area of caseation more or less separated from the wall, or extending somewhat into the wall.

Tuberculous Pneumonia.—This is by far the most important of all the processes, and has always been recognized as such. The tuberculous infiltration of Laënnec was really a tuberculous pneumonia. The tuberculous pneumonia has more or less resemblance to foci of ordinary bronchiogenic pneumonia. It may affect at the same time a large area of the lung, or the large areas may be produced by the confluence of smaller areas. The lung varies in macroscopic appearance. In some cases it has a gelatinous, almost transparent, appearance, with scattered small and opaque areas within it; or it may have the granular appearance of lobar pneumonia, but it is usually more opaque; or it may have on section a homogeneous, opaque character. Microscopically a great variety of conditions may be found. In the parts which present a gelatinous appearance we may find the alveoli filled with a homogeneous or slightly granular material in which are large numbers of epithelioid cells. In the process of hardening this material contracts and so leaves an interval between itself and the walls. This seems to be the result of a serous exudation, which has changed the character of the affected tissues, rendering them more dense than is commonly observed when they are infiltrated with ordinary serum. They present somewhat the appearance of an œdematous lung, but the material does not flow from the lung so easily on section, though it can be squeezed out to some extent by pressure. The walls of the alveoli may be but little changed. There is a varying degree of hyperæmia. The vessels may be distended with blood, but most of them may be empty. In most cases the alveolar tissue appears to be thinner than usual, or it may be infiltrated with cells. Shreds of fibrin may be found mixed with this material in the alveoli, or the fibrin may be so great in amount that the exudation can scarcely be distinguished from that of lobar pneumonia. In both the serous and the fibrinous exudations there is a varying number of polynuclear leucocytes. In some cases they may be so numerous that the exudation has a distinctly purulent character. These foci have often a close connection with the foci of bronchopneumonia and bronchitis. The number of tubercle bacilli in them varies. In the most recent foci, in which there is no softening of the caseous tissue, they may be absent entirely. In cases in which there is a close connection with foci of bronchopneumonia the tubercle bacilli may be found in the bronchopneumonic foci, and entirely absent in the diffuse exudation in the surrounding lung.

The etiology of this tuberculous pneumonia is not completely cleared up. It has been attributed to the immediate action of the tubercle bacilli, or to the influence of other bacteria, representing a mixed infection, or to the influence of products of bacilli. I am inclined to consider it due chiefly not to the bacilli, but to chemical products of bacilli. The tubercle bacilli growing in the walls of cavities must produce there a quantity of tuberculin. We do not know how much products from the advancing necrosis of tissue have to do with increasing the action of the tuberculin. In the advance of the process there is a considerable difference as compared with the extension of bronchopneumonias. Foci of bronchopneumonia do not extend by continuity of tissue, but by the continuous involvement of new bronchial territories. They are often sharply limited by the septa of the lung. In tuberculous pneumonia the process is never so sharply limited; the extension may be by continuity without any reference to bronchial territories. Cultures may show

the presence of pyogenic organisms, and they possibly assist the process, but there is no reason whatever for assuming that such infections are primarily due to such organisms. Microscopic examination of the tissue fails to show them, or reveals their presence in very small numbers. We know that the tubercle bacilli are capable of producing these exudations. Exudations similar in character may be found in the meninges of the brain and elsewhere, without any suspicion being excited that their peculiar character is dependent upon the action of secondary invaders. The lesions differ from the ordinary pneumonia lesions chiefly in the changes which they undergo. Even if the action of other bacteria be assumed, the caseation which the tissue undergoes shows that the action of the tubercle bacilli predominates over any other action. Similar lesions cannot be produced by the injection of tuberculin into the lungs of a healthy animal; in animals, it is true, the conditions are different. The tuberculin when injected is rapidly absorbed, and produces generally toxic instead of local lesions. The material which acts here is in a state not capable of ready absorption, and must act locally. It represents the soluble products mixed with mucus, possibly with particles of tissue. In all cases it is in a condition which would not admit of rapid absorption by the lymphatics. I have repeatedly found in the accompanying bronchopneumonia great numbers of bacilli in the bronchi and none at all in the surrounding exudation. Caseation takes place first in the centres of these foci and advances. That the caseation is not due to an absence of vascularity is shown by the fact that it can take place in tissue which, so far as can be judged by the presence of blood in the vessels, still has a circulation, but how active it is, it is impossible to say. Softening takes place with more or less rapidity, and results in the formation of cavities of a different character from the primary cavities in the apices. These cavities are extremely irregular and represent large channels of softening. Their walls are often composed of the caseous pneumonia tissue without any demarcation whatever. Around the periphery of these foci there will usually be found some connective-tissue formation. This shows first as an organization of the process. Beautiful examples of organizing pneumonia may be found. There is a growth of connective tissue into the alveolar spaces, taking the place of the exudation. The connective-tissue formation may be widespread so that a section of a focus may show extensive organization taking place from the periphery and advancing toward the centre, which is represented by exceedingly irregular areas of caseation. When softening takes place, then the cavity will not represent a simple hollowing out in the pneumonic lung; but, however irregular the cavity may be, it will be bounded by cicatricial tissue. The distribution of the areas of pneumonia may be chiefly lobar. Single lobes of the lung are often found chiefly affected, and all parts of the lung are never found homogeneously affected. The infection is due probably not to masses of bacilli which come from the affected portions of the lung and flow on into other parts of the lung, but to spray particles of such material formed in coughing, these particles being formed chiefly, if not entirely, in the larger bronchi. The quick inspiration in violent efforts of coughing is particularly favorable for the injection of the material into the lung. There is always, in connection with these changes, emphysema in the surrounding lung—sometimes of a pronounced character.

Recently an attempt had been made to utilize the agglutination method in the diagnosis of tuberculosis, but up to the present time the success of this does not show it to be a method of practical importance. The agglutination only takes place in such low dilutions as to show its great uncertainty. It is possible that greater success may be attended by the use of certain strains of the bacilli which will agglutinate more rapidly than others.

W. T. Councilman.

TUBERCULOSIS, PULMONARY. See *Lungs, Diseases of: Tuberculosis.*

TUBERCULOSIS: SYMPTOMATOLOGY AND TREATMENT.—Symptomatology.—The general symptomatology of tuberculosis is practically that of all wasting diseases.

In the early history of the malady, often before it is possible to determine the point of its localization, there is a great loss of bodily strength; the patient loses in weight, perhaps even to the point of emaciation; the skin loses its natural color, growing pale; there is marked dyspnœa, due to the anæmia that exists, even though the respiratory organs be not involved; the pulse is habitually accelerated and feeble; the patient suffers from indigestion, either with anorexia, or having a good appetite which is gratified at the expense of subsequent suffering; and finally, sometimes not until later in the disease, there is fever, either constantly or during some part of the day. In addition to these general symptoms, the involvement of individual organs or parts, such as the lungs, the meninges, the peritoneum, the bones, or what not, gives rise to symptoms peculiar to the part involved.

These individual symptoms will be described in detail in the articles which are devoted to the consideration of the diseases of such organs or parts, and therefore need not here be specified. It may be profitable, however, briefly to review the different methods of invasion of the several organs, especially with reference to the presence or absence of the general symptoms enumerated above.

Pulmonary tuberculosis is the most common and the most familiar form of tuberculous disease. It occurs under one of three forms:

First—Acute pneumonic tuberculosis, the onset of which is sudden, with a chill, quickly rising temperature, the physical signs of lobar pneumonia, sputum which may resemble that of lobar pneumonia, but is likely also to reveal the presence of the tubercle bacillus. This form may end fatally, even as early as the second or third week, and is generally mistaken for a severe case of simple lobar pneumonia. The importance of a correct diagnosis may be of more value in protecting others from infection than in saving the life of the patient.

Second—Acute tuberculous bronchopneumonia. This form is more frequent in children, often following other infectious diseases, as measles or whooping cough. The onset may also be sudden, with repeated chills, very high temperature, and death within a few days. Or it may run on for weeks, or even months, terminating in chronic phthisis.

Third—Chronic pulmonary tuberculosis. Here the onset is more gradual and insidious, accompanied with the general symptoms of debility, emaciation, rapid pulse, dyspnœa, pallor, indigestion, and moderate fever during a part of the day, before the pulmonary symptoms are severe, and long before night-sweats or a pulmonary hemorrhage alarm even the unwary.

It goes without saying, that the reverse of this picture may also be seen, when an alarming hemorrhage is the first sign of trouble, or a persistent cough long precedes the more general symptoms.

Tuberculous meningitis occurs more frequently among children than among adults. The prodromal symptoms are loss of appetite, loss of weight, great peevishness and irritability, without fever or local symptoms, until gradually or very suddenly and violently the true meningeal manifestations appear.

Tuberculous peritonitis presents a very varied picture, as a rule showing none of the general symptoms of tuberculosis. It may be entirely unsuspected and has repeatedly been found to exist when the abdomen was opened for the relief of other conditions. It may appear suddenly, with fever, severe pain, and the ordinary symptoms of acute peritonitis. Or the onset may be gradual, with abdominal tenderness, tympanites, and a low grade of fever not unlike the beginning of typhoid fever.

Tuberculous Pleurisy.—The ordinary form of this disease is subacute or chronic in character. The onset is insidious, with no grave general symptoms, and even the local symptoms are so little marked that the diagnosis of