cells; the spleen large and firm, with hypertrophy of the connective tissue, and the pulp rich in lymph cells and with the follicles hyperplastic.

Gianturco and Pianese report the pathological findings in a case of Fedde's as a spleen showing no increase in interstitial tissue and with the follicles little developed.

Mya and Trambusti reviewed the lesions found in the spleen and liver, and came to the conclusion that they were more or less diverse. In one of their cases the lesions in the spleen were similar to those found by Banti in cases of adult splenic anæmia, there being marked increase in the fibrous tissue, together with atrophied follicles and thickened trabeculæ.

Von Jaksch, in his description of anæmia infantum pseudoleukæmica, gives very few and very unsatisfactory reports as to the pathological appearances of the disease; he considers them to be a chronic hyperplasia of the spleen affecting in different degrees parts of the organ, with the liver showing slight increase in the connective tissue.

Hayem and Luzet report in their examination of the blood in anæmia infantum pseudoleukæmica large numbers of nucleated red corpuscles, in some of which there was evidence of karyokinesis of some of the nuclei, and they consider this to be of great diagnostic value. Fowler also lays great stress upon the presence of large numbers of nucleated red cells out of proportion to the diminution of the erythrocytes.

It is very evident that there does not seem to be satisfactory post-mortem evidence to warrant a definite pathological condition representing the splenic anæmia of infants. There seems to be present in most cases a chronic hyperplasia of the spleen, while in other doubtful cases the spleen is not altered.

Etiology.—In regard to the etiology of anæmia infantum pseudoleukæmica nothing definite has been proved. Although pathological micro-organisms, finding conditions favorable for their growth in the spleen, may, according to Somma, find their way into the circulation and thus produce changes which are represented by certain clinical symptoms, yet there is no doubt that the true nature of the disease, if such exists, has not yet been discovered. Nothing has ever been found bacteriologically to show a direct relation between the micro-organisms present and the production of the disease.

Symptoms.—It is difficult to describe the symptomatology of a disease which is so closely identified with cases of secondary anæmia with enlarged spleen, and in which the group of symptoms that are supposed to represent anæmia infantum pseudoleukæmica, are somewhat diverse. Both sexes are equally liable to present these symptoms, and the patients are usually between the ages of ten and eighteen months; one has been reported of seven and one-half months, and one at three and one-half years. The onset of the disease is gradual, the nutrition is poor; there are considerable emaciation, a waxy tint of the skin, at times hemorrhages from the mucous membranes and the skin, a spleen much enlarged, and the liver moderately enlarged. There is no tenderness over the bones, and there are often a venous bruit in the neck and functional cardiac murmurs. The blood shows the characteristics of a secondary anæmia of varying intensity, that is, a diminished number of erythrocytes, low hæmoglobin, variations in the size and shape of the erythrocytes and in the number of erythroblasts present. The leucocytes are not characteristic, being markedly increased in some cases and in normal proportion in others. The lymphocytosis, reported by many writers, may occur under any condition, giving rise to an increased number of white cells.

Diagnosis.—There seems to be no doubt that anæmia infantum pseudoleukæmica and anæmia splenica infettiva are the same condition, and, as there does not seem to be reason for believing that the spleen is primarily affected in either of them, they need not be differentiated one from the other. The differential diagnosis should first be made from leukæmia and pseudoleukæmia. The proportionately low leucocytosis and the absence of either a general lymphatic enlargement or an excess of myelocytes in the blood would differentiate it from the former, while the absence of enlarged lymph nodes would exclude pseudoleukæmia. As there are so few cases in which a thorough and reliable examination of the organs has been made in which rhachitis was not present, the diagnosis between rhachitis with enlarged spleen and secondary anæmia, and this supposed especial group of symptoms, would be very difficult and seemingly impossible.

We know that in infants the same blood changes which have been mentioned above often occur in connection with an enlarged spleen in the course of or following any disease of nutrition. We are therefore in the position of attempting to make a diagnosis between two conditions which may in the future be proved to be practically the same. In making a diagnosis we should limit the term anæmia infantum pseudoleukæmica to those cases in which no cause for secondary anæmia can be found, and in which the clinical symptoms and changes in the blood already described are present.

Prognosis.—The prognosis varies according to the extent and serious nature of the cause which produces the condition. The symptoms run an essentially subacute or chronic course; the large size of the spleen does not necessarily imply a fatal ending. When, however, secondary changes in the blood have occurred to such an extent as to warrant the diagnosis of a severe form of anæmia, and when a spleen of considerable size is detected in connection with these blood changes, the prognosis is very bad, as the infants usually die.

Treatment.—What has just been said of the prognosis in this class of cases may also be said of the treatment, which is, in fact, that of a case of secondary anæmia, and depends upon what is most reasonably supposed to be causing the condition that is present. This may be malaria, rhachitis, gastro-enteric disease, or some unknown cause which, simply representing the conditions of anæmia, calls for the usual treatment of arsenic, iron, proper food, and hygiene.

An extensive bibliography of the subject will be found in an article by Dr. A. H. Wentworth in the "Medical Communications of the Massachusetts Medical Society," vol. xviii., No. 3, 1901. *Thomas Morgan Rotch.*

PSEUDOMYXOMA PERITONEI.—The occurrence of free mucoid or colloid-like material within the peritoneal cavity, as the result of the rupture of an ovarian cyst or cystadenoma, or of the direct secretion of a neoplasm into the cavity, gives rise to a reactive proliferation of the peritoneal surfaces covered by such substance and the more or less complete organization of the latter. As a result of such partial organization the peritoneal surface is found to be covered with a jelly-like layer containing blood-vessels and strands of connective tissue, closely resembling myxomatous tissue. To this condition the term pseudomyxoma peritonei is applied.

The gross appearances vary with the amount of colloid or mucoid material lying on the peritoneal surfaces, and with the degree of organization. In the very early stages the surfaces of the peritoneum are covered with a jelly-like substance of varying thickness, which is easily scraped off, or may even be scooped out of the peritoneal cavity owing to the fact that it lies free therein. If organization has begun, the portion of the colloid material lying next to the peritoneum is not so easily scraped away, appears more opaque, and contains minute vessels, as shown by the fine red lines running through it. The peritoneal surface has therefore a reddish, roughened appearance, and is covered with fine whitish or reddish strands enclosing jelly-like colloid substance. As organization progresses there is developed above the peritoneal surface a zone of fibroblastic tissue which in time becomes changed into fibrous connective tissue, so that the peritoneum becomes greatly thickened, the condition resembling that of a chronic fibroid peritonitis. If the amount of colloid material scattered over the peritoneum is small, it may be completely organized and the peritoneum become more or

less thickened. On the other hand, a thick layer of colloid (several inches) is but slowly absorbed and organized, and may remain in the abdomen for a long time with but little change except where it comes into contact with the peritoneal surfaces. The reaction on the part of the peritoneum appears to vary greatly; in some cases it occurs immediately, in others it may be delayed for a long time.

Microscopically, sections cut through the peritoneum and the overlying mass of colloid show a fibroblastic proliferation of the subendothelial layer of the peritoneum, a wandering of fibroblasts into the colloid, and the formation of new blood-vessels which run out into the colloid substance. As organization progresses interlacing strands of connective tissue supporting blood-vessels are formed throughout the colloid substance, enclosing between them masses of the jelly material which have not yet been absorbed. These give the tissue an appearance resembling myxomatous tissue, even under the microscope. It is easily seen, however, that the structure is not that of a true myxomatous tissue, but represents an organization of a jelly-like foreign substance. All stages of organization may sometimes be seen in the same case. The writer has seen a number of cases representing different stages; in one of two years' duration the organization of the colloid was almost complete, the peritoneum being converted into a thick hyaline layer of connective tissue, enclosing here and there bits of unabsorbed colloid. Contraction of the mesentery and matting of the intestinal coils occur in this stage, and the appearance resembles very much the condition of the peritoneum in diffuse scirrhous carcinoma. The surfaces of the liver and spleen are similarly involved, and in the late stages present a picture of marked perihepatitis and perisplenitis.

Localized pseudomyxoma occurs when, from the rupture of a small ovarian cyst or cystoma, a small mass of mucoid substance is distributed in small portions over the peritoneal surface. These show the same stages of organization, hyaline change, etc., and finally come to represent localized thickenings of the peritoneum. This condition occurs most frequently in the pelvis.

If portions of living epithelium or of papillæ are set free into the peritoneal cavity with the mucoid material, after the rupture of an ovarian cystoma, these may proliferate and set up implantation metastases. These may become malignant; if the primary tumor has already undergone carcinomatous change, these implantations are likewise carcinomatous. In the case of the ordinary cystoma the implanted epithelium forms small cysts which become stationary after a while and do not form large growths. When the primary is a papilliferous cystadenoma the implantation metastases are much more likely to develop into larger tumors. It is conceivable that the implantation metastases of epithelium arising from a benign growth may later become malignant.

Small cysts lined with hypertrophic endothelium may also be found in the pseudomyxomatous tissue of the peritoneal covering. These are probably derived from the remains of the surface endothelium. It is also within the range of possibility that these may form centres for the formation of a new growth of malignant character.

Pseudomyxoma of the peritoneum is not in all cases formed by the rupture and discharge into the peritoneal cavity of an ovarian tumor containing mucoid or colloid material. Surface papillomata of the ovary may secrete such material directly into the peritoneal cavity; further, cystic carcinomata of the stomach, intestines, or testicles may give rise to the presence of mucoid or colloid substance in the peritoneal cavity, either from rupture of the primary or from secondaries located in the peritoneum.

In the great majority of cases, however, the mucoid or colloid substance comes from the rupture of a large ovarian multilocular cystoma, in which one chamber has been developed at the expense of the others; or from a primary unilocular cystoma. If the cyst contents are of a thin serous character they may be absorbed by the peritoneum without the production of peritoneal proliferation. The more jelly-like or colloid the contents the more likely the occurrence of pseudomyxoma. In order to excite peritoneal proliferation the substance must be of a fairly firm consistency and not easily absorbed. Pseudomucin may or may not be present in the cyst contents, but in the majority of cases it is a pseudomucin cyst that ruptures. The jelly-like material of the pseudomyxomatous tissue may give both mucin and pseudomucin reactions. Pseudomucin is, however, not necessary to the production of pseudomyxoma. This term should be taken as signifying the formation of a tissue resembling myxomatous tissue. Though spoken of as colloid, the cyst contents in all cases are mucoid, but when firm and jelly-like they may be appropriately designated as colloid or colloid-like.

The cyst contents when poured over the peritoneum act as a foreign body and set up a reactive proliferation which is of the nature of an inflammatory process. The presence of fibrin throughout the pseudomyxomatous tissue may often be shown by Weigert's fibrin stain. Localized collections of leucocytes may also occur throughout the organizing zone. In case of an infected cyst, or following infection as a result of operation, the picture of pseudomyxoma and that of a fibrinous peritonitis may be combined. The writer has seen one case of pseudomyxoma in which the cyst contents were scattered over a peritoneum showing a marked subacute fibrinous peritonitis. The colloid material was deposited on top of a thick fibrinous exudate which was undergoing organization. Organization of the colloid from the new fibroblastic tissue had begun in some areas.

To recapitulate, the writer holds that pseudomyxoma peritonei is a condition of the peritoneum due to a partial organization of a mucoid or colloid material, which has been deposited over the peritoneum as the result of the rupture of an ovarian cystoma or of other cystic tumor containing such material, or from the secretion of certain tumors directly into the peritoneal cavity. This view, however, is not held by all authors. Netzel, Wendeler, and others regard the condition as due, at least in part, to a chronic productive inflammation of the peritoneum associated with myxomatous degeneration. This view may be explained by the presence of pseudomucin in the lymph spaces of the peritoneum, following an absorption from the peritoneal cavity. Westermark and Annell regard the jelly masses on the peritoneum as the product of a specific form of peritoneal disease. Alshauser, Strassmann, Pfannenstiel, and others regard the process as due essentially to an implantation metastasis of tumor cells over the peritoneum. On the other hand, Werth (to whom we owe the designation *pseudomyxoma*), Veit, Kretschmar, and others hold practically the same view as the writer, namely, that the colloid masses are not metastases but are to be explained as the non-absorbable mucoid contents of a ruptured cyst, which, scattered over the peritoneum, act upon it as a foreign body, become enclosed in inflammatory adhesions, and undergo organization after the manner of a thrombus, finally being replaced by hyaline connective tissue.

The prognosis in pseudomyxoma is not necessarily bad. Large masses of colloid material may be kept within the peritoneal cavity for a long time without especial symptoms. Small amounts may be completely absorbed and organized, and the resulting condition of the peritoneum may give rise to the same sequelæ as those which follow chronic adhesive peritonitis. In operations for the relief of pseudomyxoma after the rupture of ovarian cysts, it should be borne in mind that the peritoneum, after the removal of the overlying colloid material, represents a more or less denuded, hyperæmic surface, through which infection may easily take place, giving rise to a fibrinopurulent peritonitis. The general resistance of the peritoneum appears to be lowered as the result of the presence of the foreign substance in the cavity. The danger that a malignant growth will arise from the implantation metastases is not very great in the case of a simple multilocular cystoma, but in the case of a papilliferous cystoma the danger of such an occurrence is much greater.

Such metastases may occur in the operation wound. On the whole, the safest procedure is to operate as soon as possible after the rupture of the cyst, before organization has begun. A more or less marked ascites, which complicates the diagnosis, is often associated with pseudomyxoma; this is particularly true in the case of associated infection. It may occur, however, as the result of the irritation produced by the presence of the foreign body. Recurrence takes place when the primary tumor or the metastases which produce the mucoid or colloid are not removed; or when the implantation metastases become active, and either burst or secrete into the cavity. Recurrence is much more likely to take place in the case of papilliferous growths. *Aldred Scott Warthin.*

PSEUDOPEPSIN.—When the gastric mucosa is allowed to digest for some time in a slightly alkaline medium and in the presence of an antiseptic like toluol, some of the proteids enter into solution and the tryptophan reaction (see *Tryptophan*) can be demonstrated with the latter. According to Glaessner this self-digestion is due to a specific proteolytic enzyme, to which he has applied the name *pseudopepsin*, and which in some respects resembles the trypsin of the pancreas and the autolytic enzyme of the liver. Pseudopepsin is characterized: (1) By acting in alkaline solutions, in which pepsin is destroyed; (2) by forming tryptophan as a product of its activity; (3) by acting in the presence of free acid even to the extent of 0.3 per cent. HCl, and in the presence of pepsin which destroys some enzymes. This behavior toward acids distinguishes it from trypsin. Pseudopepsin occurs in both the fundus and the pyloric portions of the gastric membrane, and in about equally small amounts. According to Glaessner the proteolytic action of the pylorus mucosa is probably entirely due to pseudopepsin. It is also apparently the characteristic proteolytic enzyme of the glands of Brunner—an observation which is of interest in view of the assumed histological resemblance between these glands and those of the pyloric portion of the stomach. It is not unlikely that pseudopepsin, or a similar enzyme, occurs in the pyloric appendages of many fishes. Pepsin can be obtained free from pseudopepsin by appropriate chemical methods. In ordinary commercial preparations the writer has found evidences of tryptophan-forming enzymes in very few instances. The existence of pseudopepsin as a specific enzyme of the stomach has been denied by Klug. *Lafayette B. Mendel.*

Glaessner: Hofmeister's Beiträge zur chemischen Physiologie, 1902, i., pp. 26, 28, 31, 111.
Klug: Pflüger's Archiv f. die gesammte Physiol., 1902, xcii., p. 280.

PSEUDOTUBERCULOSIS.—It seems that the word pseudotuberculosis was first used by Eberth in 1885 as a name for a disease in rabbits, which, although it resembled ordinary tuberculosis of these animals somewhat, was not caused by the bacillus tuberculosis Kochii. Later, the term has been used in a broader sense for all conditions which resemble genuine tuberculosis, but which are produced by organisms other than the tubercle bacillus. Baumgarten and others have objected to the use of the term, and it certainly is not a very good one. If we use the word tuberculosis in an etiological sense, meaning a disease produced by the tubercle bacillus, pseudotuberculosis might be interpreted as meaning a disease caused by pseudotubercle bacilli, that is, by those bacilli which resemble the tubercle bacillus more or less closely; which, at least according to the common acceptation of the term, it does not, altho pseudo-tubercle bacilli sometimes may produce pseudotuberculosis. If, on the other hand, we prefer to use tuberculosis in its anatomical sense, meaning a disease in which there is a production of tubercles, that is, nodules in the tissues, even then the term "pseudotuberculosis" is not very fortunate. The nodules in this disease also are certainly present and not in any way spurious, coinciding, in some instances at least, with true tubercles in all respects, even down to the least histological detail. Nevertheless, despite all these objections, the word will have to be retained for the want of a better one until, perhaps, medical nomenclature is revised and put on a scientific basis, a revision of which it certainly is very much in need.

Taking the word in its broadest sense as meaning a disease with the production of tubercles, that is, nodules of some sort, but not caused by the tubercle bacillus, we find that such a condition may be produced by many different etiological factors.

There is first the pseudotuberculosis of the rodents (the tuberculose zoogléique of the French authors). This form of pseudotuberculosis occurs chiefly among rodents (guinea-pigs, rabbits, hares, mice), but also among birds, particularly chickens, in the form of epizootics. Occasionally it has been produced by the inoculation of the most varying materials—*e.g.*, tissue from a hypertrophied tonsil (Bettencourt), cotton through which the air of the rooms of phthisical patients had been filtered (Chantemesse), material from a case of suspected tuberculosis of the elbow, and also from a nodule from a cow with pearl disease (Courmont), material from a caseous nodule from a child (Malassez and Vignal), pus from a cow suspected of suffering from tuberculosis (Nocard and Masselin), and milk (Parietti). The disease runs a more rapid course than ordinary tuberculosis. At the post-mortem examination one finds small caseous nodules in the spleen, liver, often in the kidneys, more rarely in the lungs, heart, brain, peritoneum. Quite frequently the Peyer's patches in the intestines are diseased and the mesenteric lymph nodes show large irregular areas of caseation and suppuration. On microscopical examination the nodules show more the appearance of chronic abscesses than that of typical tubercles, but at times nodules with large giant cells and typical caseation have been found. Woronoff and Sineff report that giant cells are very numerous in the lesions in chickens, whereas in rodents they found them occasionally only. Apostopoulos found nodules with all the characteristics of genuine tubercles in the liver of rabbits which he had inoculated by way of the anterior chamber of the eye. The disease is produced by a short, rather coarse, non-motile or very slightly motile bacillus, which does not stain with Gram's method, does not form any spores, and does not liquefy the gelatin. It grows luxuriantly except on potato. It does not ferment sugar, does not coagulate milk, does not produce any indol. Some authors describe irregular polar staining. At times the bacilli are arranged in short chains. It is difficult to stain them in the tissues. Tartakowsky has announced recently that in beef tea they form growths resembling stalactites, like those of the bacillus of bubonic plague. The cultures have an unpleasant odor, which is variously described by different authors. The organism seems to belong to the group of bacteria called by Kruse in Flügge's text-book "bacteria of hemorrhagic septicæmia," although Kruse himself classifies it with the bacillus mallei, to which it certainly does not show much similarity. Lehmann and Neumann put it with the bacilli of hemorrhagic septicæmia.

There are two cases on record purporting to be cases of infection with the bacillus pseudotuberculosis rodentium in the human being. One of these was published in 1891 by Hayem and Lesage. The patient suffered from Addison's disease. At the necropsy the left adrenal was found destroyed by caseation. Tubercle bacilli could not be demonstrated, nor were there any typical tubercles or giant cells in the sections. From the blood and the caseous areas the bacillus pseudotuberculosis rodentium was obtained. The other case is that of a child suffering from bronchopneumonia and empyema. In the pus from the empyema, Massa and Mensi (1895) claim to have found the bacilli. We might also cite a case of Courmont, who inoculated a guinea-pig with material from a case of what was suspected to be tuberculosis of the elbow. The guinea-pig developed a typical pseudotuberculosis. In view of the small number of cases recorded, and the possibility of error in the bacteriological diagnosis of the organism, and also in view of the fact that the bacillus pseudotuberculosis rodentium seems to be quite common, and therefore frequently

found as a contamination in all sorts of material, I believe we are justified in being a little sceptical about the nature and importance of the bacteria found in these cases, until more abundant and absolutely conclusive evidence shall have been furnished.

Then, besides, we find reported in literature isolated cases in which a similar disease was caused by other bacteria. Some of them are more or less closely related to the bacillus pseudotuberculosis rodentium. In Du Cazal's two cases, for instance (both in man, one with caseous nodules on the surface of the peritoneum and similar nodules of the size of a nut in pancreas and liver; the other with large caseous nodules in brain, in pleura, along spinal column and in both kidneys), he found a bacterium which differed from the bacillus pseudotuberculosis rodentium largely only by the fact that it liquefied gelatin. Legrain found a similar organism in the pseudotuberculous lesions of a rabbit that had been inoculated with sputum from a case of pulmonary phthisis.*

In other cases the bacteria encountered were quite different. Preisz, for instance, and also Kutscher found organisms which resembled diphtheria bacilli. Still other and even more uncommon bacterial forms of pseudotuberculosis in animals have been reported by Cherry and Bull, Galli-Valerio, Vallée, and others but we cannot very well enter here into a fuller consideration of these forms.

Again other forms of pseudotuberculosis are caused by certain filamentous bacteria. In Eppinger's case of this kind there were a cerebral abscess, a very chronic tuberculosis with calcification of lungs and peribronchial lymph nodes, and tuberculosis of the pleura. The disease was caused by a form of cladothrix (asteroides), which when inoculated into rabbits and guinea-pigs produced pseudotuberculosis. Flexner reports a case which clinically had all the symptoms of pulmonary phthisis. At the necropsy he found pulmonary cavities and tubercle-like nodules in the lungs, omentum, peritoneum, liver, and spleen. Although histologically the nodules were identical with tubercles, no tubercle bacilli were found, but instead branching threads which stained well with Gram's method. Cultures could not be obtained. An inoculated guinea-pig died, but not of tuberculosis.

Infection with certain mould fungi is also one of the many causes of pseudotuberculosis. By intravenous injection of the spores of certain moulds in rabbits, for instance, one can produce a most beautiful disseminated pseudotuberculosis, as Grawitz has demonstrated long ago. An interesting form of pseudotuberculosis, produced by mould infection, is described by Chantemesse and others as occurring in pigeons. The disease starts with a caseous ulcer in the mouth, which is later followed by the formation of nodules in lungs, liver, more rarely œsophagus, intestines, kidneys. Histologically the lesions resemble tubercles very closely. The cause of the disease is the aspergillus fumigatus. The disease seems to be communicated at times to breeders of pigeons, who stuff young pigeons by introducing food into the mouth of the animals directly from their own mouths, in imitation of the parent birds.

Nodules which resemble tubercles very closely are produced in the skin in certain forms of blastomycetic dermatitis, and even more regularly are they found in another rarer form of fungus disease, which has been first described by Wernicke in Buenos Ayres, and has since then been observed several times in California. The disease generally begins as a chronic cutaneous trouble resembling hypertrophic lupus; later, a disseminated pseudotuberculosis of nearly all the internal organs except the heart and gastro-intestinal canal develops. I have seen cases, however, which did not show any cutaneous lesions, but in which the primary infection seems to have taken place by inspiration into the lungs. The fungus which causes the disease in the tissues multiplies by endogenous sporulation, only without formation of mycelia or budding, and was on that account first described as a protozoan by Wernicke and Rixford and Gilchrist, who studied the earliest cases in California. I succeeded, however, in cultivating the organism in artificial culture media, and in these it grows out into long spore-bearing hyphæ. The classification of the fungus is as yet doubtful, and until our knowledge of it is more complete I have proposed the name fungus coccidioides. I can only confirm the reports of earlier investigators that the similarity in the histological structure of the lesions produced by this fungus to typical tubercles at times is truly remarkable. A histological differential diagnosis between them, apart from the difference in the causative factor, is in these instances absolutely impossible. With such typical tubercles one finds simultaneously in the lesions numerous submiliary chronic abscesses, very much like those which commonly occur in glanders.

With this long list of vegetable parasites our list of producers of pseudotuberculosis is by no means exhausted. Among the animal parasites we find quite a few of the smaller parasites or their eggs, which when accidentally disseminated in the tissues can cause the formation of tubercle-like nodules around them. De Jong, for instance, describes cases of pseudotuberculosis in sheep and goats produced by intestinal worms (strongylus rufescens), and claims that to the naked eye the differential diagnosis from ordinary tuberculosis is difficult. Marsden also reports cases of large, more or less tubercle-like nodules in lungs, liver, and kidneys of sheep, hogs, and goats, due to the eggs of filaria strongylus. In 1884 Laulanié entertained the Société Biologique in Paris with the account of pseudotuberculosis of dogs produced by demodex folliculorum, and in 1899 Helbing read a paper at a meeting of the Freie Vereinigung der Chirurgen Berlins, in which he describes a case of pseudotuberculosis of the peritoneum in man caused by the dissemination of the eggs of a tapeworm. The nodules had the typical histological structure of tubercles; the eggs or fragments of them were enclosed in giant cells, but there was no caseation.

Even small dead foreign bodies, particularly when they are of a somewhat irritating nature, will cause the formation of nodules in the tissues. As early as 1869 Waldenburg proved this by experiments, which he relates in his monograph on "Tuberculosis, Pulmonary Phthisis, and Scrofulosis" (Berlin, 1869), a piece of work which is not so well known as it deserves to be; and even before him Cruveilhier ("Traité d'Anatomie pathologique générale," iv., 1862) had attempted to produce, and to his own satisfaction succeeded in producing, tubercles in the lungs by injection of small droplets of metallic mercury into the trachea of dogs. Only recently Meyer described a case, observed in Hanau's laboratory, of pseudotuberculosis produced by foreign bodies. In this instance a gastric ulcer had perforated and small particles of food had been scattered through the peritoneal cavity. Around these small particles of food a development of tubercles had taken place. A similar observation was made in another case in which an ovarian cyst had ruptured and a large number of cholesterin masses were disseminated through the peritoneum.

When we consider the great variety of causes which have just been enumerated, the question seems natural, Are we really justified in calling all these conditions, which are so manifold *etiologically*, by the one name pseudotuberculosis? We shall be all the more inclined to ask this question when we learn that in many instances the lesions histologically do not resemble one another entirely. In the pseudotuberculosis of the rodents, for instance, the nodules on microscopical examination usually present the appearance of chronic miliary abscesses, such as they are observed, for instance, in glanders, and not that of typical tubercles. The same is true of the lesions caused by infection with certain pathogenic moulds. In these lesions giant cells and caseation which are considered to be the more important

* In the last issue of Ziegler's Beiträge (1902, xxxii., 526) Wrede reports an interesting case of pseudotuberculosis in an infant. Pharynx, œsophagus, intestines, liver, and adrenals were full of gray submiliary nodules. The condition was caused by a bacillus which closely resembled the bacillus pseudotuberculosis rodentium, but which differed from it in staining with Gram's method.

characteristics of true tubercles, are often absent. Yet under other conditions the same parasites may produce nodules which in histological structure resemble ordinary tubercles very closely. In chickens, for instance, we learn from Woronoff and Sineff that the nodules produced by the bacillus pseudotuberculosis rodentium contain many giant cells. One must also not forget that in ordinary tuberculosis the nodules show remarkable differences in histological structure according to the age of the nodule and the number and virulence of the tubercle bacilli present. Tubercle bacilli also at times may produce chronic miliary abscesses. Since my attention has been called to this occurrence by some observations made while studying the ependyma of the ventricles of the brain in tuberculous meningitis ("Ueber Ependymveränderungen bei tuberculöser Meningitis," *Virch. Arch.*, cl., 1897, 305), I have seen chronic miliary abscesses produced by tubercle bacilli—and by tubercle bacilli alone without associated infections—quite frequently in other parts of the body in man and animals. In infections with the fungus coccidioides the simultaneous occurrence of "typical" tubercles and chronic miliary abscesses in the same organ, produced by the same parasite, is very bewildering; indeed one is finally forced to recognize that the difference between these two conditions is not a fundamental one—as a matter of fact, I have seen transitional forms of otherwise typical tubercles with central abscess cavity filled with pus cells,—and that whether the tissues respond in one way or the other depends only on the amount of irritation to which they are subjected. If the irritation is less marked, a "typical" tubercle develops; if it is more intense, a chronic miliary abscess is produced. It seems, therefore, that in spite of the variety of causes, and in spite of the varying appearance of the nodules under the microscope, it is advisable to group all these conditions which are closely akin to one another under one name, "pseudotuberculosis," provided we keep in mind that in so doing we use the word "tubercle" in its broadest sense for a nodule, without assuming anything too definite about its exact histological structure. *William Ophüls.*

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PSEUDOTUMOR.—The term *pseudotumor* is applied to certain enlargements or swellings of non-neoplastic character which clinically present features by which they may be mistaken for true neoplasms. In the majority of cases such false tumors are found in the abdominal region. They may be produced by a great variety of causes. When the swelling cannot be constantly felt, but comes and goes, it may be designated as a *phantom tumor*. Such enlargements have no organic pathological foundation, and are dependent upon temporary conditions. On the other hand, enlargements or swellings of the abdominal organs due to organic disease may also simulate neoplasms; these conditions should be included under the designation of *pseudotumor* or *apparent tumor* proper. When caused by inflammatory masses of granulation tissue which later contract and disappear, the condition may be spoken of as *vanishing* or *disappearing* tumor.

Phantom tumors may be caused by a contraction of the abdominal muscles or by meteorism. Those caused by muscle contractions are found usually in the upper part of the abdomen. The right rectus near its costal margin is most frequently the part contracted, but the contraction may affect any segment or portion of the abdominal muscles. The contractions are often spasmodic. The entire rectus may be rigid. Usually, however, but a single segment is affected, the contraction being almost always unilateral. The patients are usually hysterical females who present marked stigmata of hysteria; there are usually coexisting constipation and enteroptosis. The contraction may sometimes be made to disappear in a hot bath; but in cases of marked hysteria anæsthesia or hypnotic suggestion may be necessary for the differential diagnosis. The superficial character of the tumor, its flat, horizontal shape, slightly rounded and indistinct edges, etc., in connection with the stigmata of hysteria, make the diagnosis easy.

Phantom tumors, due to meteorism or localized distention of the intestines with gas, are of frequent occurrence in the same class of patients as described above, and are often found in connection with the muscle contraction. The character of the swelling, the percussion signs, etc., make the diagnosis easy. The swellings occur usually in the lower portion of the abdomen, particularly in the appendix region, and above the pubis. They are found frequently in women who either pretend or believe that they are pregnant (pseudocyesis). The associated stigmata of hysteria, and the characteristic physical signs render the diagnosis of slight difficulty.

Apparent tumors of the epigastrium are, according to Einhorn, of infrequent occurrence. He reports forty-two cases, eight occurring in men and thirty-four in women. The tumors presented in the epigastrium or in the left or right hypochondrium, and formed in the majority of cases smooth masses of the size of a hen's egg or a man's fist. They were frequently pulsating, could not always be distinctly felt, and on light percussion yielded a dull sound. They ran a long course, and there was usually a history of a long-continued malnutrition. The tumors remained unchanged or diminished in size. They were caused by prolapse of the left lobe of the liver, exposure and thickening of the abdominal aorta, hypertrophic conditions of the muscles of the abdominal walls, and probably adhesions around the lesser curvature of the stomach. If the tumor is caused by the prolapse of the left lobe of the liver, it is found usually in the median line below the ensiform; it is of large size and gives a dull tone on percussion. Between the dulness of the tumor and the ensiform there may be an area of tympanitic tone. If the tumor is the aorta, it is deep, usually about two inches long, and one to two thumbs in breadth, and pulsates. Hypertrophic conditions of the abdominal muscles are superficial, usually horizontal, and not globular, and are located at one side of the median line.

In very thin individuals the head of the pancreas may be felt and mistaken for a tumor. Likewise floating kidney, liver or spleen, rolled-up omentum, excessively fat mesentery, fæcal impaction, distended urinary or gall bladder, tuberculous thickenings of omentum or mesentery, hydro- or pyosalpinx, cystic dilatation of the appendix, etc., may sometimes be regarded as presenting the clinical appearances of malignant tumors of these regions.

Inflammatory thickenings, tuberculous and syphilitic nodules, encapsulated hæmatomata, encysted parasites, infective granulomata of unknown origin, localized hypertrophy of muscle, local œdema, etc., are also often mistaken clinically for malignant tumors.

The term pseudotumor is also applied to the nodules of a chronic inflammatory nature caused by the experimental injection of blastomycetes and other fungi. Similar nodules may be produced by the introduction of foreign bodies or the injection of certain chemical substances into the tissues.

Disappearing tumors of the abdomen are usually the result of acute inflammatory tumors of the omentum, following appendicitis or salpingitis. The absorption of exudates and the contraction of the granulation tissue lead to the diminution in size or total disappearance of the tumor. Similar disappearing tumors occur in the skin, subcutaneous tissues, periosteum, and intermuscular connective tissue as the result of the formation of granulation tissue following trauma or hemorrhagic extravasation. These may sometimes be mistaken for sarcomata, both clinically and microscopically. The presence of numerous plasma cells, the character of the blood-vessels, and the general appearance of the granulation tissue are points upon which the differential diagnosis should be based. (See also *Omentum* and *Abdominal Tumors.*) *Aldred Scott Warthin.*

PSITTACOSIS.—An infectious disease occurring in birds, particularly in parrots, and transmissible to man. The disease in parrots is of the nature of a chronic enteritis, characterized by diarrhœa, wasting, loss of appetite, and falling of feathers. In man the symptoms are those of a grave typhoid, with diarrhœa and a malignant atypical pneumonia. The disease may be transmitted directly from parrots to man or through intermediate objects, and, according to some observers, from man to man. The period of incubation is from seven to twelve days; the symptoms begin with malaise, epistaxis, and digestive disturbances, followed by bronchitis and pneumonia. The urine contains a small amount of albumin. There is high fever lasting for from three to four days, and falling by crisis. These symptoms then recur in this order several times, defervescence finally taking place by lysis. During the attack the spleen is enlarged. Sometimes there may be seen a roseolar or petechial eruption. The disease lasts about thirty days. The mortality is about thirty-seven per cent. The prognosis is good if complications do not occur. In the majority of fatal cases, death is due to pneumonia.

Eberth in 1880, and Wolff in 1883, observed the occurrence of a fatal mycosis in parrots which had been imported in great number from the west coast of Africa during 1880. Transmission to man was not observed. In 1879 Ritter saw a house epidemic of severe pneumonia which he thought was referable to a contagion from parrots, or rather from the cages in which the birds had been transported. The clinical and anatomical picture of the disease was that of an atypical pneumonia. Similar cases were observed in 1882 by Ost, and by Wagner in 1882 and 1886. The disease was introduced into Paris in 1891 by some parrots from South America. In 1892 there was an epidemic of the disease in this city, in which fifty persons were affected. Cases of the disease were also observed in Paris during the next four years, and advantage was taken of the opportunity to study the disease closely. The relation of the disease in man to the affection of the parrot was clearly proved. Cases have been observed also in Italy and Germany.

According to Nocard, the cause of the disease is a specific bacillus resembling that of typhoid fever. The organism is short, rather thick, with rounded poles, is motile, and is a facultative aërobe. It does not stain with Gram's method, does not ferment sugar, does not coagulate milk, and does not form indol. The bacillus is very virulent; subcutaneous injections in rabbits, mice, and pigeons kill within from fourteen to forty-eight hours. In the Paris epidemic this bacillus was not found in the human body; but, three years later, Gilbert and Fournier found it in one case, in the heart blood of a woman dying from the disease. Palamidessi observed an infectious disease transmitted from parrots which he regarded as resembling chicken cholera. The organism obtained by him was regarded as identical with that observed by Nocard. Other observers have failed to find the Nocard bacillus; and Leichtenstern and others believe that the disease of the parrots known as psittacosis may be caused by various bacteria (staphylococcus, streptococcus, pneumococcus, colon bacillus, and proteus), and that house epidemics of atypical pneumonia in man may occur without such diseases of the parrot playing any etiological rôle therein. These writers, however, admit the probability of such a relation in certain cases, as in the Paris epidemic of 1892.

On the other hand, Nicolle reports an epidemic attacking eight persons (four dying), in which the Nocard bacillus could not be found; but the serum from these cases produced a typical agglutination of a culture of the bacillus furnished by Nocard, in dilutions of 1 to 50 and 1 to 60. The blood of one of the cases also agglutinated typhoid bacilli, although the patient had never had the disease.

Widal and Sicard claim that typhoid and psittacosis can be differentiated by the Widal reaction. In dilutions of 1 to 10 the reaction occurs with both; but the masses of psittacosis bacilli are smaller and more crowded. In dilutions of 1 to 40 there arrives a moment when the bacilli of psittacosis no longer react.

The bacteriology of psittacosis and the true relations of the parrot disease to the atypical pneumonia in man remain yet to be determined definitely. Further, it should be observed that in the popular mind psittacosis is regarded as a form of avian tuberculosis, and that cases have been reported of a supposed transmission of tuberculosis from the parrot to man, whence the origin of the error. *Aldred Scott Warthin.*

PSOAS ABSCESS is a *cold abscess* located in the psoas muscle. The purulent material gains entrance into the muscle after destroying the vitality of a portion of the sheath by pressure and infiltration.

The iliac fascia which ensheaths the whole muscle con-