made by dumping either upon lowlands or into water. The former usually creates more or less nuisance, but is tolerated because of its economy. In San Francisco the mixed refuse is cremated. In Troy a portion only (the most offensive part) is cremated, while in a few Southern cities parts of the garbage and dry refuse are burned in the open air at the dump.

Night Soil.—By this term is meant the liquid or semi-liquid contents of privy vaults and cesspools, derived from the household wastes of the water-closets, bath-tubs, and other fixtures, including that of the kitchen sink. The latter often proves troublesome in consequence of its greasy character and its liability to clog the traps and pipes through which it flows.

On account of the liability to cause nuisance in the performance of this work it is customary to require that the scavengers, or other persons who conduct it, shall be licensed and placed under careful supervision.

It is customary to require the use of tight receptacles for the removal of night soil, either barrels or tubs provided with handles and tight-fitting covers. (Fig. 3951.) Boxes or movable tanks are also employed. The so-called pneumatic odorless pumps are well adapted for this class of work. (Fig. 3952.) Tubs and barrels are conveniently handled, ten or twelve of such receptacles usually constituting a load.

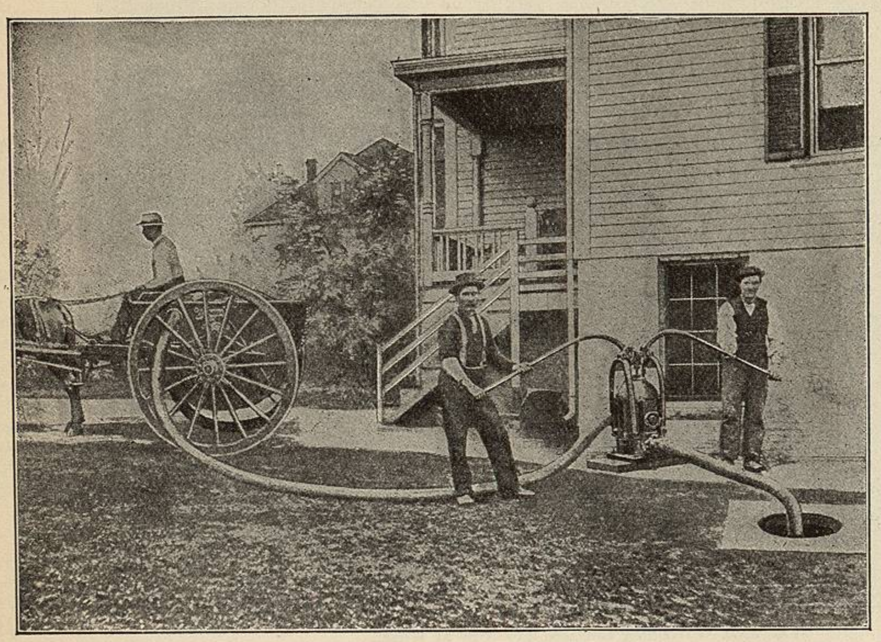

FIG. 3952.—Odorless Excavating Apparatus in Operation.

It was formerly customary to remove such material only at night, but this practice is gradually being substituted by removal in the daytime, when with improved apparatus it is practicable to do the work in a more cleanly manner.

Disposal.—In some cities the night soil is taken to suburban districts and there used upon farms as a fertilizer, a practice liable to cause nuisance to the neighborhood, if thus disposed of in the vicinity of dwellings. In some places it is dumped into water, either a large river, lake, or the ocean. In a few English cities where the pail system of excrement removal is still in use, the contents of the pails are taken to the crematory and burned together with other sorts of refuse.

The Refuse-disposal of Public Institutions and Other Establishments.—Special destructors are now made of smaller size than those in use by cities, for the destruction of the waste products of public institutions, hotels, department stores, factories, medical colleges, and other isolated establishments. Such forms of apparatus (Fig. 3953) are specially adapted to the destruction of infected bedding, clothing, rags, and other infected material which is not worth the trouble and expense of saving by any process of disinfection.

Samuel W. Abbott.

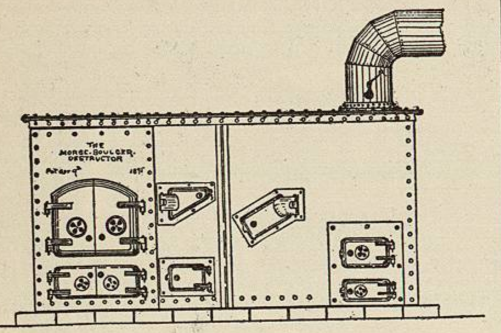

FIG. 3953.—Garbage Destructor for Use in Public Institutions, Factories, etc.

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[2] W. F. Goodrich, A.I.M.E.: The Economic Disposal of Town's Refuse, London, 1901.
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[4] Boston Post, January 21st, 1900.

REGENERATION.—Regeneration is the process which leads to the replacement of lost tissue. The term is applied equally to the reproduction of cells to take the place of those cast off under normal physiological conditions on the one hand, and to the repair of tissue destroyed by lesion on the other. In both cases the process is essentially the same. The new tissue is formed by the proliferation of cells in the immediate neighborhood of the area from which the cells have been cast off or destroyed. Moreover, the new tissue is derived from remaining tissue of like kind, connective tissue from connective tissue, epithelium from epithelium. Where reproduction of epithelial tissue does not take place, or where the reproduction is incomplete, the defect, it is true, is filled with connective tissue forming a scar; but this scar tissue is derived from the connective tissue around the defect, and not from the epithelium. Physiological regeneration goes on continuously to supply tissue waste; regeneration after lesion occurs in the healing of wounds, in the restoration of the mucosa after catarrh, and in similar processes.

There are two methods of multiplication of cells, direct division or amitosis, and indirect cell division or karyokinesis, mitosis or karyomitosis. In the former of these two methods the mother cell simply increases in size and

finally divides into two daughter cells after a division of the nucleus. In karyokinesis the process is more complicated, consisting as it does of a cycle of definite changes in the chromatin of the nucleus preceding the division. Direct division or amitosis is said by some authorities to take place only in cells which are no longer capable of forming new tissue, whereas mitosis occurs in active vigorous cells which form new tissue. Other authorities, on the contrary, regard the two processes as of equal significance.

The power of reproduction differs in different tissues. Surface epithelium and epithelium lining glands are capable of regeneration to a very large extent, as are also nerve fibres and many of the other tissues of the connective-tissue group generally. Of the latter, the periosteum is capable of the greatest degree of regeneration, whereas cartilage has only a limited power of regeneration. Ganglion cells once destroyed are probably never replaced by ganglion cells in the adult, and glandular epithelium is completely restored only where the defect is slight and where some of the original cells remain uninjured in the area of the lesion.

Regeneration of Epithelium.—In regeneration of epithelium the protoplasm of the cell divides in the later stages of karyokinesis, or after this is complete. In some cases the cell sends out processes of protoplasm, and the new nuclei wander out from the parent cell into these before the protoplasm is constricted off to form the new cell.

In a lesion of an epithelial surface, where the area destroyed is not too great in extent, the epithelial cells proliferating from all sides completely repair the defect; if it is more extensive, the defect is incompletely filled by connective tissue, thus forming a permanent scar. In the latter case, the connective tissue forming the scar results, as in all cases, from the proliferation of pre-existing connective tissue, not from proliferation of epithelium. Epithelium reproduces epithelium, never connective tissue, but it is also true that the character of epithelium reproduced is not always the same as that which is lost; for squamous epithelium may replace cylindrical epithelium, as is seen in atrophy of the mucosa of the bronchi and stomach in chronic inflammation of these surfaces.

Minor defects of epithelial surfaces, where underlying tissues are not destroyed, are quickly and completely restored by the proliferation of the surrounding epithelium. In recovery from acute Bright's disease, or in the healing of an ulcer of the stomach or intestines, the epithelium is often completely restored by the multiplication of the remaining mucous glands as well as by growth downward of the epithelium on the surface. Lesions of the liver are also completely restored by multiplication of the cells of the parenchyma and by the formation of new bile ducts.

Larger defects of epithelial tissue, particularly where the underlying structures are involved, result in a scar, with more or less incomplete reproduction of glands which are for the most part usually atypical.

Regeneration of Structures Consisting of Connective Tissue.—Defects of structures made up of connective tissue in any of its various forms may under favorable circumstances, as where the lesion is not too extensive, or in lesions of certain of the tissues of this group, even though they be extensive, be completely restored by the new formation of tissue identical in character with that originally present. Where the lesion is more extensive, particularly in one that occurs in certain of the connective-tissue structures, the defect is more or less filled up by connective tissue of a different type from that originally present. Connective-tissue structures such as the periosteum, bone marrow, blood and lymphadenoid tissue, are all readily restored completely after lesion. On the other hand, cartilage does not readily reform, and defects in this structure are replaced by ordinary scar tissue or by bone. New bone is formed not from pre-existing bone but from the remaining periosteum and from the bone marrow. Sometimes new bone is formed in other connective tissue, as in the intermuscular connective tissue and from the perichondrium. But muscle tissue is formed only from pre-existing muscle, never from connective tissue of any other kind. Ganglion cells are probably never restored after injury, defects in this tissue being replaced by scar tissue or by glia cells; but nerve trunks are readily restored, provided that the ganglion cells from which they spring are uninjured, for the regeneration is brought about by the growth of the axis-cylinder processes. The peripheral portion of a severed nerve always undergoes degeneration, it is never restored; all parts of it finally break down into granules and are dissolved away. The central portion of a severed nerve, on the contrary, undergoes degeneration for only a short distance from the end, back to the first or second Ranvier constriction from the end. In a few days after the occurrence of the lesion the axis cylinder in the central portion of the severed nerve begins active proliferation. At first the new axis cylinders are naked save for a layer of protoplasm rich in nuclei, but sooner or later the proper sheaths are reformed. If the degeneration of the peripheral segment of the severed nerve has not yet affected the Schwann's sheath, the new axis-cylinder processes may enter these and fill them out again. The regeneration of nerves takes weeks or months for completion.

In regeneration of connective tissue, as in reconstruction of other tissues, the proliferating cells are always much larger than the cells at rest. The proliferating cells, or formative cells as they are also called, not only possess a relatively large amount of protoplasm, but the nuclei are large and vesicular and contain nuclear bodies, many of them showing, by proper methods of hardening and staining, the various stages of karyokinesis. Frequently the formative cells contain more than one nucleus, sometimes even a large number of nuclei, forming giant cells (Fig. 3954, *i*). The tissue made up of these

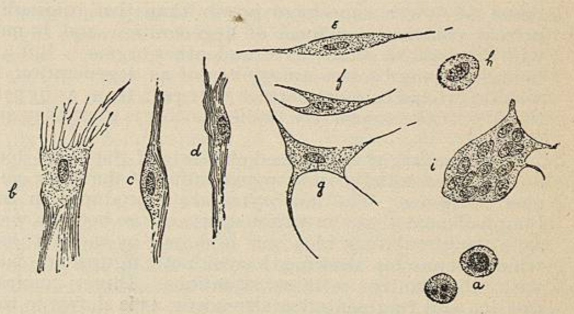

FIG. 3954.—Cells met with in New Formation of Connective Tissue. (After Ziegler.)

formative cells is appropriately called embryonic tissue, and the character of the permanent tissue arising from the development of the embryonic tissue is determined by the nature of the embryonic cells. If the formative cells are destined to form scar tissue they are called fibroblasts; if they are to form cartilage they are called chondroblasts; if bone, osteoblasts.

The formative cells are of many different shapes; some are large, round, or oval masses of protoplasm (Fig. 3954, *h*), others spindle-shaped (Fig. 3954, *e*), others roughly star-shaped (Fig. 3954, *g*), others irregular with long processes (Fig. 3954, *c*, *d*, *f*), or bundles of fibres at the ends as if the protoplasm were frayed out (Fig. 3954, *b*).

If fibrillated connective tissue develops from the embryonic tissue, fibres make their appearance between the formative cells, and by continuous increase they encroach more and more upon the latter till the formative cells finally lie compressed in fissures between the densely packed fibres.

Cartilage develops by the formation of a homogeneous intercellular substance instead of fibres, but the formative cells are encroached upon and compressed just as in the case of ordinary fibrillar connective tissue. The formative cells become much reduced in size, and finally lie in the characteristic openings in the homogeneous intercellular substance.

The process of new formation of bone is similar to the new formation of cartilage; the lime salts are finally deposited in the intercellular substance.

Mucous, lymphadenoid, and fatty tissues may all arise from embryonic connective tissue.

Fibrillated connective tissue may arise from any form of connective tissue that has suffered lesion, and has been converted into embryonic tissue.

From what has been said, it is evident that the stage which is preliminary to the new formation of permanent tissue after lesion is characterized by the appearance of embryonic tissue. The further development of this embryonic tissue is not the same in all cases; in some instances there is perfect reproduction of tissue in all respects like the original, or *restitutio ad integrum*. In many cases the restitution is only partial; in others again there is replacement of the original structure by connective tissue of a different type from the original. The replacement of the original structure by connective tissue of a different type from the original may occur in any kind of connective tissue, and is in truth a very common occurrence, constituting the formation of a scar, where the original structure is replaced by dense fibrous connective tissue, whose only function is to fill a gap. Where the original structure is endowed with any special function, the scar tissue is incapable of taking on this function. If, for example, the scar tissue is formed to fill up a defect caused by a lesion in a muscle, the scar tissue serves only to unite the divided ends of the muscle fibres, but is not itself capable of contraction. Scar tissue formed in the brain, in the liver, in the kidney, in the spleen, or in the lung does not perform the peculiar function of the tissues of these organs. The substitution of connective tissue of lower functional power than that originally present constitutes a form of degeneration, and is met with in cirrhosis of the liver and other organs. But although these processes are spoken of as degeneration, it would perhaps be more correct to regard them as hypertrophies of the connective tissue, for this is what they are in fact.

Regeneration of the formed elements of the blood does not differ essentially from regeneration of the other connective tissues. The leucocytes are reproduced in the lymphadenoid tissue in various parts of the body as well as in the circulating blood, as is shown by the fact that white corpuscles showing karyokinetic figures are met with abundantly in these situations. Direct nuclear division and fragmentation also occur, as is shown in the lobed and disrupted nuclei of the polymorphic nuclear leucocytes.

New Formation of Blood-Vessels.—A very important factor in regeneration is the new formation of blood-vessels.

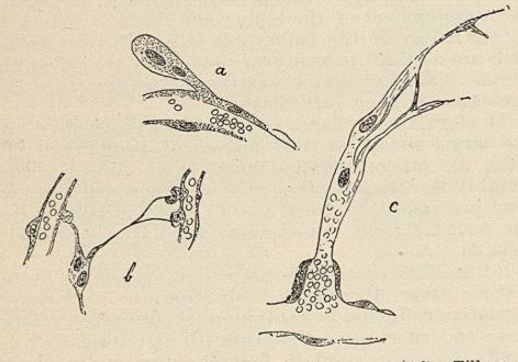

FIG. 3955.—New Formation of Blood-Vessels. (After Tillman.)

These are formed by sprouts arising from the vessels beneath the injured area. Each sprout is at first a solid arch, in reality a long cone or horn, of protoplasm projecting from the wall of the vessel (Fig. 3955, *a*) terminating in a long-pointed process. The solid arch may even send out several long processes (Fig. 3955, *b*, *c*). Sooner or later the solid arch becomes hollowed out by liquefaction of the interior, forming a cavity which finally opens into the parent vessel, or the arch may be hollow from the start and consist merely of a bulging of the wall of the parent vessel (Fig. 3955, *c*). In either case the arch next becomes a tube by the pressure of blood flowing in from the parent vessel, and this also causes the long processes to split and form hollow, conical tubes. Meanwhile nuclei formed by karyokinesis of the cells of the endothelium of the parent vessel wander into the walls of the hollow tube and convert it in this way into a capillary. The capillaries produced in this way have walls consisting of flat endothelial cells, but these cells are frequently quite thick; and when this is the case, the new-formed capillary resembles a tubular gland on cross section. The long, slender processes from the arches unite freely with the similar processes from other vessels (Fig. 3955, *b*) and even directly with the walls of other vessels, or they may unite with the parent vessel at a different point from the origin (Fig. 3955, *c*). In this way abundant anastomosis between the vessels is formed.

This is not the only origin of the new capillaries, for certain cells which at first have no connection with blood-vessels also take part in the process. These cells are spindle- or club-shaped masses of protoplasm which lie free in the tissue at first, but later become united with the long processes from the arches (Fig. 3955, *b*). After they become united with the processes from the arches they become perforated by a central canal and finally assume the character of capillaries.

Many of these new-formed capillaries subsequently change to arteries and veins by thickening of their wall through proliferation of the cells of the walls. The different coats finally become differentiated.

The account here given of the new formation of blood-vessels is that given by Ziegler, and is the one that has found universal acceptance. But some authorities claim that the blood-vessels are also formed in other ways. They maintain that the cells that wander into the area of the lesion unite to form capillary vessels, and that they also form new red blood corpuscles in a manner similar to the formation of vessels and blood in embryonic tissue. This method of new formation is not recognized by Ziegler in pathological regeneration.

Another process of new formation of blood-vessels claimed by some authorities is that certain spindle-shaped cells form in parallel rows, leaving a canal between them. Vessels apparently formed in this way may be seen in organizing blood clot, but Ziegler regards these as really deceptive offshoots from pre-existing blood-vessels.

B. Meade Bolton.

REGISTRATION OF DISEASES. See *Vital Statistics.*

RELAPSING FEVER.—(Synonyms: Febris recidiva; typhus recurrens; famine fever; bilious typhoid; spirillum fever; epidemic remittent fever; remitting icteric fever; fièvre à rechute; fièvre récurrente; typhus à rechute; Hungerpest; Rückfallsfieber; Wiederkehrendes Fieber; Armentyphus; tifo recidivo, etc.)

DEFINITION.—A specific, contagious fever, which may prevail as an epidemic among the destitute, and especially among those who live in overcrowded tenements, during seasons of unusual scarcity of food; hence the name famine fever. It is characterized by the presence of a mobile spiral filament in the blood—a spirillum or spirochaete (*S. Obermeieri*)—which is found during the relapses as well as during the initial paroxysm, but is absent during the apyretic intervals. The first febrile paroxysm lasts for from five to seven, or even nine days, and is terminated abruptly in profuse perspiration; after an apyretic interval of a week or more a relapse commonly occurs, which is similar to the initial paroxysm, but of shorter duration; in some instances a second, a third, or even a fourth relapse occurs.

HISTORY.—The attempt has been made (Spittal, 1844) to show that some of the fevers described by Hippocrates correspond with relapsing fever. This view is considered by Hirsch to be quite erroneous. He says: "It is clear that Hippocrates speaks there of bilious remittent malarial fever." The first notice of the occurrence of relapsing fever in Europe is found in the writings of the Scotch and Irish physicians of the early part of the eighteenth century. Hirsch says: "I have searched in vain, in the descriptions which the physicians of the sixteenth and seventeenth centuries have given of the fever epidemics observed by them, for any indications of relapsing fever that would be in some measure precise." The fact that the disease was not recognized, and differentiated from other specific febrile affections, cannot, however, be taken as evidence that it did not exist prior to the date of the first clearly recorded epidemic in Ireland (1739). An account of this epidemic has been given by Rutty, who wrote in 1770. The earliest accounts of the disease in Scotland date from 1741 (Hirsch). But the literature relating to relapsing fever belongs for the most part to the past century. It prevailed in Ireland and in Scotland during the years 1799–1800, 1817–19, 1826–27, 1842–48, and in the latter year (1848) it invaded several of the larger towns of England. In 1868–70 it again prevailed in England and Scotland, and cases are reported to have occurred in London as recently as the year 1873. On the Continent the first accounts we have come from Russia—Odessa, in 1833; Moscow, 1840–41. In the autumn of 1863 the disease reappeared in Odessa; the following year it became epidemic over extensive areas in Russia, and extended to Livonia and Finland (1865), to Siberia (1866), and to Poland (1868). According to Hirsch, the disease continued to prevail in Russia over extensive areas during subsequent years, and was observed among the Russian troops as late as 1878–79. In Germany an extensive epidemic broke out in 1868, as a result of importation from Russia (Hirsch). A second, more restricted, epidemic occurred in 1871–72, and a third in 1878–79. In the west and southwest of Europe—Switzerland, France, Italy, Spain—the disease is as yet unknown. In India relapsing fever has, no doubt, prevailed for many years, but the differential diagnosis between it and remittent fever, or the specific continued fevers which prevail there so largely, was not clearly made out by the earlier observers. During the last forty years, however, numerous outbreaks of this disease in various parts of India have been recorded, and Carter has demonstrated that the disease, as it occurs in that country, is identical, as regards its clinical history, with relapsing fever as described by recent European authorities, and also that it is characterized by the constant presence of the spirillum discovered by Obermeier in blood drawn during a febrile paroxysm. Relapsing fever has several times been imported to the United States, but its prevalence has been limited to restricted areas in our largest seaport cities. In 1844 fifteen cases were received into the Philadelphia Hospital from an emigrant ship sailing from Liverpool; in 1848 a few cases arrived in New York, and in 1850–51 Dr. Austin Flint saw a number of cases, among recently arrived Irish emigrants, received into the Buffalo City Hospital; but no epidemic resulted from these importations, and it was not until some years later (1869–70) that the disease became epidemic in certain sections of the cities of New York and Philadelphia. Parry, who made a careful investigation with reference to the origin of the first cases in Philadelphia, was unable to trace it to importation; but this can scarcely be questioned in view of what is known of the history and etiology of the disease, and in consideration of the fact that Philadelphia is a seaport city which has constant communication with ports on the other side of the Atlantic which at that time were known to be infected. Parry and Pepper have given us admirable accounts of this epidemic in Philadelphia. We quote from a recent article by the last-named author the following statement, relating to its progress and extent: "In Philadelphia, of 1,176 cases in which the date of occurrence is known, there occurred in September (1869), 4 cases; December, 6 cases; January (1870), 5 cases; February, 13 cases; March, 124 cases; April, 209 cases; May, 325 cases; June, 293 cases; July, 115 cases; August, 19 cases; September, 28 cases; October, 15 cases; November, 1 case; December, 2 cases; January (1871), 2 cases; February, 1 case; March, 2 cases; May, 7 cases; June, 2 cases; September, ? cases; October, 2 cases.

The coincidence of relapsing fever and typhus has been noted in many of the epidemics which have occurred in Europe, but the history of this coincidence does not justify the supposition that there is any etiological relation between these diseases other than that furnished by common predisposing causes, viz., the depressing effects of overcrowding, insufficient food, and filthy surroundings. This view is supported by the fact that either disease may occur alone, and by the circumstance that sometimes one and sometimes the other has the precedence in time in those epidemics in which coincidence has been observed.

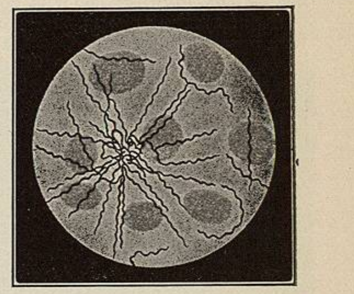

FIG. 3956.—Spirilla of Relapsing Fever. (After Soudakewitsch, *Annales de l'Institut Pasteur*, vol. v., 1890.)

ETIOLOGY.—The discovery by Obermeier, in 1873, of a minute vegetable parasite—*Spirochaëte Obermeieri*—in the blood of patients suffering from relapsing fever, and the subsequent demonstration, by numerous observers in various parts of the world, that this micro-organism is constantly present in the blood of relapsing-fever patients during the febrile paroxysms, has thrown a flood of light upon the etiology of this disease, and is one of the most significant facts with reference to the etiology of the infectious diseases in general which have been brought to light by modern microscopical researches. Conservatism suggests the possibility that the parasite may be simply an accompaniment of the disease, and not directly concerned in its etiology as the essential and specific cause. This hypothesis seems to us to be hardly tenable in view of what is now known of the pathogenic action of certain other micro-parasites of the same class, and of the following facts: (*a*) The parasite is constantly present in the blood during the febrile paroxysms, and in smaller numbers during the latter part of the period of incubation, and is absent during the apyretic intervals. (*b*) This parasitic organism is peculiar to the disease under consideration, *i.e.*, repeated researches by competent microscopists have failed to demonstrate the presence of a similar organism in any other disease. (*c*) The parasite is present in the blood in such numbers that its pathogenic power can scarcely be questioned. Carter says: "During specific fever several organisms (*e.g.*, five to ten) are visible in the field at one time; not seldom they are too numerous to count, and occasionally they are present in swarms, being apparently nearly half as common as the red discs themselves." (*d*) The disease may be communicated to man (Motschutkoffsky) and to the monkey (experiments of Koch and of Carter) by inoculations with blood containing the spirillum, and the parasite is found in great numbers in the blood of the inoculated individuals during the febrile paroxysm which results—after an incubation period of three or four days (Carter)—from such inoculations. The morphology of the relapsing fever "germ" is shown in Fig. 3956. The spiral filaments are exceedingly slender, their diameter being not more than 1 μ (0.001 mm., Lebert), or, according to Carter, $\frac{1}{50000}$ to $\frac{1}{40000}$ of an inch. The length varies from two to six times the diameter of a red blood disc (Carter). The motion of these spiral filaments, in blood recently drawn, is very lively, "rotary, twisting, and rapidly progressive, but soon ceases under the ordinary condi-