the receptacle. On relaxing the fingers and raising the receptacle the food flows gently into the stomach. Care must be taken to remove the tube gently and quickly; the infant must then be replaced in its warm cradle or couveuse. To the weakest infants 8 gm. of food are given every hour. In spite of this feeding the infant frequently loses weight for the first few days, but soon begins to gain steadily. Dr. Tarnier claims to have achieved marvellous results by this method.

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INFARCTION.—(*Infarcio*, to fill in, to stuff into.) The act of plugging up or filling up; also the organ or tissue which is filled with blood, fluid, or other substances. The development of the various meanings of infarction forms a very interesting chapter in the history of medical terminology. Originally used as a designation for the Galenic teaching of the black bile as the cause of melancholia and hypochondria, *infarctus* gradually came to mean a stoppage of the intestinal canal either through hardened fæcal matter or through precipitations from the blood into the intestinal canal. Later, the idea included also obstruction in the blood and lymph vessels and the ducts of glands. The theory of intestinal infarctus as the cause of hypochondria, melancholia, hemorrhoids, and other conditions reached the height of its development in the latter half of the eighteenth century under the influence of the two Kämpfs, father and son. By them the doctrine was reduced to what was considered to be a scientific basis, the infarcts were obtained for examination by means of injections, and were classified according to symptoms, location, and composition. Their treatment by clysters attained an extraordinary degree of popularity. The abuse of this treatment and the advancement of knowledge of the anatomy and physiology of the gastro-intestinal tract brought about the natural death of the doctrine during the early part of the last century. As outgrowths from the early idea of infarctus were such usages as the following: *infarction of humors*, a term formerly applied to ulcers whose base and borders were infiltrated; *infarctus mammæ lacteus*, the collection of coagulated milk in the ducts of the mammary gland; *infarctus lactei extremitatum*, applied to phlegmasia dolens because it was supposed to be caused by metastasis of the milk to the legs; *infarctus uteri*, applied to a variety of diseased conditions of this organ; etc. The term infarction was also formerly used clinically to indicate a sense of fulness or oppression in the chest. The enlarged spleen in chronic malaria was called *infarctus lienis*. The old meaning of intestinal obstruction still survives in the expression *infarcted hernia*.

At present infarction is unfortunately used as a designation for two classes of pathological conditions essentially different in kind: 1. The infiltration of the tubules or tissue spaces of an organ with blood, blood pigment, uric acid or urates, calcium salts, etc. 2. The anæmic necrosis of an organ or tissue due to an obstruction of its nutrient artery by embolism or thrombosis. The different processes are usually distinguished by some modifying term, as uric-acid infarction, anæmic or hemorrhagic infarction, etc.; but when used without such the term is usually narrowed to the second meaning.

1. Uric-acid Infarction.—In the kidneys of the newborn there is almost always present in the medullary pyramids a number of glistening, golden, or orange-red lines converging to the papillæ. These lines are easily scraped away with the knife and form a glistening orange powder on the moist surface of the organ. On microscopical examination the straight tubules are found to be filled with dark semitranslucent masses which dissolve on treatment with acetic acid, leaving behind an albuminous stroma. From the solution uric-acid crystals are formed. The epithelium of the tubules shows little or no change. The condition is known as the uric-acid infarct of the new-born, and was at one time supposed to be of medico-legal value as occurring only in infants who have breathed; but similar infarcts have been found in the kidneys of still-born children and in the fœtus. In one case of ectopic gestation seen by the writer the kidneys presented an extensive degree of this condition. According to Spiegelberg, uric-acid infarct is present in only one-half of the children examined, being most marked after the second or third day after birth. The condition may persist until the seventy-sixth day, but usually disappears early. When persistent, it may give rise to nephritis or cause uræmia by the simple obstruction of the tubules. Neither the origin nor the significance of the condition is known. The change of metabolism following the establishment of pulmonary oxygenation, the slight degree of oxidation processes in the new-born, lack of ability on the part of the urine to dissolve the uric acid, are among the causes adduced in explanation, but no one of these is adequately supported. Uric-acid and sodium-urate infarcts occur also in the adult kidney in cases of gout or the so-called uric-acid diathesis. The appearances are similar to those in the new-born, but the condition is rarely so extensive. More frequently, scattered concretions are formed in dilated tubules.

Calcium Infarcts, Chalk Infarcts.—In senile kidneys, in osteomalacia and other destructive diseases of bone, in cases of poisoning with mercuric chloride, phosphorus, aloin, bismuth, etc., extensive deposits of lime salts, phosphates or carbonates, may occur in the tubules, the deposit taking place either in or upon the necrotic epithelium of the tubules. When visible to the naked eye, such deposits appear as opaque whitish or yellow streaks corresponding to the tubules, or as minute dots representing the glomeruli. Microsopically the deposits give the specific staining reaction with hæmatoxylin. Calcium-oxalate infarction occurs rarely as the result of ingestion of food containing large amounts of oxalic acid or in cases of disturbed metabolism. White streaks occur in the pyramids similar to those of phosphate infarction, but are distinguished from the latter by their insolubility in dilute acids.

Bilirubin Infarct.—In cases of chronic obstructive jaundice the collecting tubules of the kidney usually contain large numbers of bile-pigment casts. In tissue fixed with alcohol these appear as yellow or brown streaks in the cut surface of the medullary pyramids; in mercuric chloride fixation they have a bright green color. Uric-acid infarction is not infrequently combined with this condition, particularly in cases of icterus neonatorum.

Hæmatoidin Infarct.—In cases of hæmoglobinuria, hæmoglobinæmia, and methæmoglobinæmia, due to poisoning, infections, extensive burns, etc., large quantities of blood pigment may be found in the kidney tubules in the form of brownish casts or irregular masses. The pyramids show radiating yellow or reddish-brown striæ. Usually the pigment does not give the iron reaction, and is therefore to be classed with the hæmatoidin group. Very rarely hæmosiderin infarction occurs.

Melanin Infarct.—Very rarely in cases of extensive metastasis of melanotic sarcomata large quantities of melanin are said to be excreted through the kidneys. In such cases melanin casts are found in the tubules. It is doubtful, however, if the brownish casts described as occurring in these cases were composed of melanin; it is more likely that they were formed of blood pigment.

2. Anæmic and Hemorrhagic Infarction.—The occlusion of a terminal artery by embolism or thrombosis leads to an anæmic necrosis of the part supplied by the

obstructed vessel. In the case of end-arteries having very slight collateral anastomosis the dead area remains anæmic (anæmic or white infarct); in situations where the end-arteries possess sufficient anastomosis to admit of the flow of blood into the affected area from the neighboring terminals the dead area becomes hemorrhagic (hemorrhagic or red infarct). The anæmic variety is found in the spleen, kidneys, heart, brain, retina, and rarely in the liver; the hemorrhagic infarct occurs only in the lungs and the area supplied by the superior mesenteric artery, very rarely in the uterus. Hemorrhagic infarction of the suprarenal has recently been reported.

Infarcts are usually found at the periphery of organs where they form cone- or wedge-shaped areas, the base toward the surface of the organ, the apex pointing inward. They may be single or multiple. As the result of the occlusion of a number of neighboring arterioles they are occasionally very irregular in outline. When recent, the anæmic infarct is opaque, yellowish-white, slightly elevated, and somewhat firmer than normal tissue. A peripheral red zone is almost always present. Older infarcts are usually depressed and softer; in the case of those of large size there is usually a central softening. If organization of the dead area has begun the consistence may be firm. On microscopical examination the cells of the infarcted area show a simple or coagulation necrosis. In older infarcts liquefaction necrosis may be present. The red peripheral zone is found to be due to a narrow line of hemorrhage and congestion surrounding the dead area and may be explained as being of the nature of a hemorrhagic infarct in the zone of collateral anastomosis with the neighboring end-arteries. In later stages this zone usually becomes the seat of a leucocyte infiltration and fibroblastic proliferation. The hemorrhagic infarcts are deep red in color, occasionally pale in very anæmic individuals. They are usually much firmer than the anæmic variety. As they become older they grow lighter in color, and softer if liquefaction necrosis occurs. If organization takes place without softening the infarcted area becomes lighter and firmer. The contraction of the newly formed connective tissue leads to depressions on the surface of organs in the case of both anæmic and hemorrhagic infarcts. On microscopical examination the hemorrhagic infarct is found to consist of an area of necrotic tissue, the spaces of which are filled with extravasated blood. It is in fact only an anæmic infarct which has become hemorrhagic. The term hemorrhagic infarction is applied also to the occurrence of simple hemorrhage in tissues; in the case of the kidney to the hemorrhage produced by obstruction of the renal veins. It would be better to drop entirely this confusing use of the term and restrict its application to the true anæmic and hemorrhagic-anæmic necroses produced by arterial obstruction.

Various theories have been offered in explanation of the fact that infarcts in one case are anæmic and in another hemorrhagic. It is held by some that the anæmic infarct is but a later stage of the hemorrhagic, produced by the removal of the blood pigment from the latter. Old hemorrhagic infarcts are not infrequently lighter in color for this reason, but the fact remains that anæmic infarcts are non-hemorrhagic from the beginning except for the small peripheral zone of hemorrhagic infarction surrounding them. The true explanation is probably to be found in the character of the collateral anastomosis of the affected vessels, as well as in local and general conditions of the circulation. Where there is rich anastomosis, as in the lung and mesentery, the obstruction of an end-artery leads to a temporary anæmia which lowers the vitality of the tissue supplied by the obstructed vessel; later, an inflow from the collaterals takes place, but the damaged condition of the vessel walls in the anæmic area leads to extravasation, chiefly by diapedesis, but also by rhexis. The infiltration of the tissue spaces thus produced leads to further damage of the part. Very rarely the local or general conditions of blood pressure may be such in organs in which hemorrhagic infarcts are usually produced that but little blood flows into the anæmic area and the resulting infarct is pale in color. The anæmic infarct occurs in organs whose end-arteries have but little anastomosis, so that no inflow takes place into the anæmic area except just at the periphery, where a narrow zone of hemorrhagic infarct is formed. In cases of extreme local or general congestion this peripheral zone of hemorrhage may be greatly broadened, or in the case of small infarcts the entire area may become hemorrhagic. The inflow of blood is usually under such slight pressure that the integrity of the part cannot be maintained.

Under ordinary conditions the infarcts of the brain are always of the anæmic variety, with more or less peripheral hemorrhage. Liquefaction of the affected area takes place, and if death does not result brain cysts may be formed. Occlusion of the coronary arteries by thrombosis, embolism, or sclerotic changes in the vessel walls produces anæmic infarction of the myocardium, attended by more or less hemorrhage. The infarcts of the kidney and spleen are almost always of the anæmic variety with a narrow zone of hemorrhage. True anæmic infarcts of the liver are very rare. In the normal lung the obstruction of a terminal artery will not produce infarction owing to the rich anastomosis. In conditions in which the pulmonary circulation is already embarrassed, as in the passive congestion of mitral disease, embolism or thrombosis of the pulmonary arteries is quickly followed by infarction. This is practically always of the hemorrhagic variety except in very rare cases, due to extreme weakness of the circulation. The conditions in the superior mesenteric artery are similar to those in the lungs, but inasmuch as the anastomosis is not sufficient to re-establish the circulation after occlusion, obstruction of the terminals of this artery leads more quickly to infarction than in the case of the pulmonary arteries. According to Chiari, bilateral embolism of the hypogastric arteries or bilateral thrombosis of the uterine veins is followed by hemorrhagic infarction of the uterus.

The sequelæ of infarction are: absorption of the necrotic tissues, organization, scar formation, calcification, cyst formation, encapsulation, abscess, or gangrene. In cases of infective embolism suppuration of the infarcted area usually occurs (septic infarct), or the infarct may be secondarily infected by pyogenetic organisms. Infarction of the lungs is frequently followed by moist gangrene, infarction of the intestine almost always. (See *Heart, Kidneys*, etc.)

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INFECTION.—Probably one of the most universally known facts respecting bacteria is that they are related to the development of certain pathological processes; but it should be clearly held in mind that many play a very beneficent part in nature, chiefly by decomposing dead organic material and thus forming other chemical compounds which may be utilized for food by other plants and animals. It has been seen, in the article on *Bacteria*, that micro-organisms are widely distributed in air, soil, and water; and it is among the habitations of man, where the conditions for their growth and multiplication are specially suitable, that they are most numerous. So far as we know, with few exceptions micro-organisms whose natural habitat is in the soil, air, or water are not under usual conditions harmful to man, for they are invariably present in greater or less numbers in the mouth, nose, upper respiratory passage, gastro-intestinal tract, and on the surfaces of the skin, where they are more or less constantly brought by the respired air, by food and drink, and in many other ways. Notwithstanding, however, their occurrence in these situations in great numbers, they do not often gain entrance into the body tissues, so that under normal conditions the blood and viscera are germ-free.*

Although the cutaneous and mucous surfaces of the body are covered with many micro-organisms, these are for the most part harmless, sometimes serving useful functions, as do those in the intestines. Whenever they

*For a consideration of the bacterial flora of the body surfaces consult Welch: "Surgical Bacteriology." "System of Surgery by American Authors," edited by Dennis.

find their way into the body, conditions are so unsuitable for their existence that they are soon destroyed. Against such incursions the body is guarded in various ways. Among the protective agencies may be mentioned the epithelial investments of the skin and mucous membranes, which so long as they are intact protect the interior from bacterial invasion. Moreover, the germicidal qualities of some of the body fluids and cells also afford important protection. But notwithstanding these protective factors bacteria do sometimes enter, and this may especially occur whenever the skin or mucous membranes are injured, even if ever so slightly so.* Within recent years the entrance of bacteria through the bites of insects has been especially studied.†

Among those gaining entrance into the body, there are some which may under suitable conditions induce phenomena by which disease is characterized. These are the micro-organisms which induce infectious diseases and which we habitually refer to as being pathogenic bacteria. The term pathogenic is, however, a relative one—a fact which should be clearly held in mind for the following reasons: First, an organism quite harmless to one animal may be capable of inducing disease in another; second, an organism which under ordinary conditions is harmless may under special conditions give rise to serious lesions; third, many organisms generally regarded as harmless under special conditions may prove injurious. Thus it is evident from what has been said that infectious disease cannot exist without the presence in the body of living micro-organisms. But, on the other hand, the mere entrance of bacteria into the tissues is not sufficient to constitute infection, for it has been seen that many bacteria are harmless and that the body possesses important safeguards whereby bacteria are destroyed and disposed of. Whether the micro-organism possess pathogenic activity or not will depend largely upon the host and also upon its own variable nature and qualities.

This relationship of host and bacterial excitant has been so admirably stated by Prudden that one can do no better than quote the following:

"In the study of the infectious diseases it is especially important to bear in mind that the abnormal processes through which the disturbances incited by micro-organisms are manifested are processes of the body cells and not processes of the micro-organisms. The micro-organisms do indeed incite the train of phenomena by which the disease is manifested, and the nature or 'species' of the micro-organism may largely influence the character of the phenomena; but the stored-up energy which is released in this manifestation is body-cell energy and not that of microbic metabolism. The microbes are excitants of disease, but the disease is a performance of the body cells. If these obvious considerations be held in view, it will be convenient in considering certain of the infectious diseases to use the familiar and much-abused term 'specific' as indicative of those phases of abnormal body cell performance which are apt to occur in characteristic ways in response to special forms of microbic stimulus. Thus the poisonous substances which the tubercle bacillus build up out of the organic material upon which it feeds are in part such as exert a peculiar influence upon connective-tissue cells, leading to their proliferation and the temporary formation of new tissue—the tubercle. This, together with associated action of the same or other metabolic products of the living bacillus, forms a group of lesions and disturbances which is characteristic of the action of the tubercle bacillus in the body. In this sense tuberculosis is a 'specific' disease. On the other hand, the poisons eliminated by the tubercle bacillus may incite responses on the part of the body cells which are practically identical with those which many other toxic substances, both of bacterial and of other origin, induce—fever, degeneration, etc. These manifestations of the action of the tubercle bacillus upon the living body cells are not 'specific.'"

The Bacterial Excitant.—The reaction of the body cells in infection bears a more or less constant relation to the virulence of the infecting micro-organism and to the number of bacteria gaining entrance into the tissues. The virulence varies considerably under different conditions, and according as these variations are small or great different phenomena will develop within the body. The character of the processes induced will necessarily vary according to the virulence of the infecting bacterium; and this largely depends upon its environment, which may not only modify its morphological characters, but also may change its physiological activities. For example, the Bacillus coli communis as normally found in the intestines is of very low virulence and is not capable of inciting pathological processes, but so soon as its physiological activities are modified by changes in its environment, its virulence is apt to be very much increased. This influence of environment is also well shown by the modifications which may occur in the vital activity of an organism when it finds its habitat in new and unnatural hosts. This point is especially well demonstrated with the mammalian tubercle bacillus, which when transmitted for a long time through another animal host, as birds, will ultimately lose its virulence for the host from which it was originally derived.

As a rule, bacteria lose their virulence with greater or less readiness when cultivated in artificial media. On the other hand, successive passages of a pathogenic organism through susceptible animals exalt its virulence. Thus a streptococcus of attenuated virulence may be exalted a hundredfold by successive inoculations into rabbits.

Differences in the virulence of bacteria often suffice to explain differences in the clinical and morphological types of disease. It is well known that under some conditions an organism of low virulence will incite rather mild reactions of the body cells, whereas under other conditions, when the virulence of the same organism is exalted, the effects are both more marked in severity and in extent of the lesions. Thus I have been able to follow these variations in virulence in the different forms of tuberculosis. For example, in scrofulous lymphadenitis, I have found that the tubercle bacilli which induce this lesion were usually of low virulence, whereas the tubercle bacilli inducing acute miliary tuberculosis are on the other hand of extraordinary pathogenic activity.

The number of micro-organisms which gain entrance into the body is also a factor which modifies the character and extent of the cellular reactions. Within certain limitations the healthy body may dispose without apparent injury of a certain number of given virulence, but when the same organism is introduced in large quantities infection follows. In susceptible animals probably so small a number as one or two anthrax bacilli may incite disease. Usually, however, much larger numbers are necessary for the development of an infection. Further, according to the portal of entry, the number may be increased or diminished. Thus a given dose of bacteria, which when injected into the subcutaneous tissues of an animal may prove quite harmless, will incite, when the same dose is introduced intravenously or into the peritoneal cavity, well-marked disease to follow.

The Host.—This is a factor of the very greatest importance in the consideration of infection in its numerous aspects. It is, of course, well known that certain bacteria will induce infections only in certain species of animals, proving absolutely innocuous for other species. Thus, while the anthrax bacillus usually induces lesions with the greatest readiness in many animals, others, like the white rat, are ordinarily insusceptible to inoculation with these bacilli, unless very large amounts are given or special factors be brought into play. Some diseases are especially peculiar to man, such as typhoid fever, syphilis, leprosy, scarlet fever, measles, etc., these diseases never occurring naturally in other animals. The race and individual peculiarities are also factors which modify the liability to infection. Thus negroes are generally insusceptible to yellow fever, whereas other human beings are quite susceptible to the pathogenic agent of this disease. The influence of age is so well known that investigators commonly make use of this knowledge in their experimental work. It may be stated as a rule that young animals are much more readily infected than older and larger ones. Age as a predisposing factor to infection is well illustrated by the curve of frequency of infectious diseases in man, the maximum point occurring among children. Sex is also sometimes a factor predisposing to infectious disease, although this is not so apparent among animals as it is among human beings. Numerous other conditions affecting the physiological integrity of the body are also predisposing influences for infection. The influence of fatigue, starvation, cold and heat, and loss of blood have been particularly studied from the experimental point of view, the results very conclusively proving that a marked susceptibility to infection is developed.

Animals which are normally insusceptible to anthrax, such as white rats, may be made vulnerable by fatigue. Thus Charrin and Roger have shown that if white rats be made to work a treadmill until thoroughly fatigued, and be then inoculated with anthrax bacilli, they succumb to the lesions of anthrax infection.

Canalis and Morpurgo have shown that starvation also interferes with the natural resistance of animals to bacterial infection. These observers studied this phase of the subject with pigeons, which are also naturally resistant against anthrax. By starving these animals, either before or just following inoculation, they are made extremely susceptible to the influence of the inoculated organism. These experiments have been repeated time and again with other animals, especially by Pernice and Alessi.

The influence of exposure to heat, cold, and moisture has been similarly investigated by Pasteur, Petruschky, and others. Long ago Pasteur showed that by immersing a hen in cold water it loses its natural immunity to anthrax. Frogs, if kept at a temperature (25°–35° C.) higher than is normal for them, will easily succumb to anthrax infection; and guinea-pigs and white mice, which are resistant to fowl tuberculosis, can be made susceptible by keeping them after inoculation in a warm chamber at 43°–45° C.

Any injury to the body tissues also favors the lodgment and activity of whatever bacteria may reach the affected area. It is a well-known clinical fact that tuberculosis of bones and joints of children often follows different forms of trauma. Rosenbach demonstrated this predisposition of damaged tissues to bacterial invasion by traumatizing one of the heart valves and then injecting cultures of the staphylococcus. Malignant endocarditis almost invariably followed, whereas in the control animals in which the valves had not been injured no lesions of endocarditis were found.

Other factors have also been well investigated, such as the action of chemical substances and unsuitable diet, the latter especially by Hankin, who fed refractory rats on sour milk and bread. Such treatment made the animals extemely susceptible to anthrax infection. Leo administered phloridzin in small doses, and reached the conclusion that it markedly predisposed the animals to bacterial infection. There are still other special influences which may more or less modify the conditions in the development of infection. The removal of certain organs, such as the removal of the pancreas from pigeons for example, will make the body tissues more susceptible than they would otherwise be to the infection. Animals kept in dark and damp places also exhibit marked susceptibility as compared with those living in light and airy localities. Trudeau has conclusively established that rabbits kept in places in which no light penetrates contract tuberculosis more readily than others kept in a more healthful environment.

Portals of Entry of Bacteria.—Bacteria may gain entrance into the body tissues through several different ways, and according to the portal of entry differences not only in the susceptibility of the animals but also in the lesions and symptoms of the disease will be noted. For the most part micro-organisms reach the interior of the body from the skin and mucous surfaces. It is very improbable that bacteria present upon the skin can penetrate this tissue when uninjured. It is true, however, that very often they seem to reach the interior of the body from what seems to be an uninjured skin surface, but in these cases it is probable that the skin injury has been overlooked on account of its smallness. Attention has already been called to the fact that bacterial agents may be communicated through the bites of different insects. Thus it is now a well-established fact that the malarial parasite is transmitted through the bite of the mosquito; and the same is probably true for yellow fever. The mucous membranes of the respiratory and alimentary tracts are frequently portals of entry for micro-organisms which have been carried by the air and water and foods. The mucous membrane of the urogenital tract is less frequently a portal of entry. Other mucous surfaces, such as the conjunctiva, may also under special conditions be points of entry for different pathogenic bacteria. It is only very rarely that bacteria directly gain entrance into the general blood current; they ordinarily reach it through the sources which have been mentioned.

Animal experiments clearly show that the portal of entry and channel of infection bear a more or less direct relation to the severity of the infection which follows. Subcutaneous inoculations in animals are usually much less severe in their results than those made directly into the peritoneal cavity or general vascular system.

Action of Bacteria and Their Products in the Body.—Micro-organisms induce their bad effects in several ways, but chiefly by their presence in the tissues and development of their poisonous products, which either affect the physiological activity of the cell or kill the cell outright. These poisonous products act generally or locally in varying degree, according to the nature and quantity of the product formed. These toxic substances become diffused through the system, and their effects are manifested clinically by the occurrence of fever or disturbances in the functions of the respiratory and nervous systems. In some cases changes are found in the local tissues directly involved. The general effects of bacterial poisons may be so slight as to be regarded of little importance, as in the case of a local inflammation, for example; or they may be very intense, as in tetanus and diphtheria. These results are largely directly attributable to the poisons of the bacteria, rather than to the presence of the organisms themselves. In infectious diseases both factors are prominent features, the toxic side being usually more conspicuous.

In diseases like tetanus and diphtheria it is usually only in the local lesion that the bacilli are found. The profound systemic intoxication is accomplished by absorption of the highly toxic products from the local lesion.

Whenever there is a widespread distribution of pathogenic bacteria in the blood the condition is designated septicæmia; and if associated with multiple foci of pus formation the term pyæmia is applied. The body which is already the seat of infectious disease is much more susceptible to the invasion by other bacteria; thus mixed or concurrent infections are often established, as in tuberculosis of the lungs with cavity formation. In addition to the tubercle bacillus there may usually be found in such lungs pyogenic (pus excitants) bacteria. Individuals the victims of long-standing chronic diseases, such as those of the heart, lungs, kidneys, and liver, often succumb to infectious diseases of one kind or another. The phrase "terminal infection" has been applied by Osler to these infectious diseases.

Communicability of Infectious Disease.—Considered in its practical relations, the subject of the communicability of infectious disease is one of the very highest interest and importance. It has been already pointed out that some infectious diseases, like syphilis, cannot be commu-

*The view has been recently advanced that with no apparent lesion of the intestinal mucosa, bacteria in large numbers may gain access through this to the liver under what seems to be perfectly normal conditions (Adami: Jour. of the American Medical Association, December 16th and 23d, 1899).

†For a consideration of the rôle of insects in the transmission of disease consult Nuttall: Johns Hopkins Hospital Report, 1900, vol. viii.

nicated to animals. On the other hand, some of the infectious diseases of animals are not communicable to man. But certain other infectious diseases, like tuberculosis, are readily transmitted to either man or animals.

The infectious diseases of man may be divided into two great classes: first, those which under usual conditions are communicable; and second, those which under usual conditions are very communicable. Among those of the second class there exist the greatest differences in the liability to transmission. It is well known how readily the excitants of smallpox and scarlatina are given off from the patients under conditions which make their transmission from one person to another an easy matter. In diseases like syphilis and hydrophobia, on the other hand, the excitant is transmitted only by direct inoculation. Between these extremes we have many gradations. It should be clearly borne in mind that a classification based on the communicability of a disease is not fundamental, but is closely dependent upon the sanitary conditions under which these diseases are observed.

Immunity.—Immunity is characterized by resistance to infection or its effects. The absence or loss of this capacity is known as susceptibility. Immunity from an infectious disease may be *hereditary*, or it may be *acquired*, either by an attack of the disease from which the individual has recovered—*natural immunization*—or by the introduction into the body of something which diminishes susceptibility—*artificial immunization*. Acquired immunity may be transmitted from parent to offspring.

Many of the infectious diseases confer greater or lesser immunity to subsequent attacks of the same disease, although there are some exceptions to this. A previous attack of erysipelas makes one more susceptible to subsequent infection with the streptococcus.

From the study of infection it is known that two distinct influences are evidently at play in enabling the body to resist infection. It has been clearly shown that the destruction of bacteria may in part be brought about by the leucocytes and other mesodermal cells, which when thus engaged are called phagocytes. In the body fluids there are also certain albuminous ingredients—alexins or "defensive proteids"—which have well-marked bactericidal properties.

Artificial immunization is generally accomplished by making the body tissues tolerant to the presence of bacteria or their poisons. This may be done by the introduction of attenuated micro-organisms or by the introduction of their poisons. The same result may be obtained by introducing the body fluids from an already immune individual.

This immunizing power is attributable to the development of "antitoxic substances" whose nature is still little understood. They seem to be closely related to the globulins.

It is not possible to determine to what extent the immunization effected is specific. In some cases it seems to be so; whereas in others this is less apparent.

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INFILTRATIONS.—The collection of fluid or cells in the tissue spaces, and the deposit in or between the cells of substances abnormal in kind or quantity, are included in the comprehensive class of *pathological infiltrations*. The processes involved are of widely differing nature; each variety of infiltration being usually indicated by some modifying designation, *hemorrhagic, inflammatory, tuberculous, leukæmic, tissue infiltration*, etc. Without such specific designation infiltration is commonly taken to refer to the presence of some pathological substance in or about the cells, and is used as a synonym for pathological deposit. The infiltrated substance itself is also usually spoken of as an infiltration.

Fluid Infiltrations.—Œdema, non-inflammatory in origin, is sometimes spoken of as a hydropic or dropsical infiltration. As a result of injury to the bladder or urethra extravasation of urine into the surrounding tissues, particularly into the perineum, may occur (urinary infiltration). Extravasations of blood, lymph, milk, fæcal material, etc., are also styled infiltrations.

Inflammatory Infiltration.—This may be hemorrhagic, serous, fibrinous, or cellular in character. The collection of serous exudates in tissue spaces is usually spoken of as inflammatory œdema. Fibrinous infiltrations occur in diphtheritic inflammations and other forms of coagulation necrosis. The cellular infiltration of inflammation is for the chief part composed of leucocytes that have migrated from the blood-vessels. Wandering cells arising from proliferating tissue cells may also be found in it. Usually the leucocytes are of the polynuclear variety, but occasionally the infiltration is made up entirely of mononuclear forms. In some cases the cells of the infiltration are chiefly plasma cells; in other cases eosinophile or mast cells may be very numerous. The significance of these phenomena is as yet unknown. A leucocyte infiltration of moderate degree is usually spoken of as a small-celled infiltration; when the leucocytes are in large numbers as a purulent infiltration. (See *Inflammation* and *Exudations.*)

Leukæmic Infiltration.—An increase in the number of the leucocytes in the blood is attended by an increase in the number of the wandering cells in the tissue spaces. This increase is greatest in such organs as the liver and kidneys, and in the submucosa and mucosa of the gastro-intestinal tract. In leukæmia the accumulation of leucocytes in the interstitial tissue throughout the entire body may reach an enormous degree. The liver and kidneys become enlarged, firmer, and lighter in color as the result of such an infiltration. This may be diffuse, or circumscribed grayish-yellow areas of infiltration may occur throughout the organ (leukæmic nodes, lymphoma). The mucosa of the larynx may be infiltrated to such an extent as to give rise to symptoms of œdema of the glottis. In the intestine the swelling of the mucosa may be followed by ulceration. The variety of leucocyte present in the infiltration depends upon the kind of leukæmia, though in some cases of mononuclear leukæmia the proportion of polynuclear forms in the tissues far exceeds that in the blood. Proliferation of the migrated cells occurs, as shown by the occurrence of cell-division figures.

Tumor Infiltration.—The malignant neoplasms, carcinoma and sarcoma, invade adjacent tissues by extension through the tissue spaces in the direction of least resistance, usually through the lymph spaces. This process is styled growth by infiltration. Extension in this manner is always more marked in the case of carcinoma than in that of sarcoma. Around the infiltrating growth there is almost always present a zone of leucocyte infiltration as evidence of the inflammatory reaction produced by the development of the tumor. In many cases it is very difficult or impossible to distinguish between the leucocytes and the young tumor cells.

Tissue Infiltrations.—The term infiltration is very frequently used as a synonym for both pathological degenerations and deposits (albuminoid, hydropic, fatty, glycogenic, cholesterin, mucous, hyaline, amyloid, calcareous, uratic, and pigmentary infiltrations). Inasmuch as in the majority of these conditions the process is of the nature of a deposit in the affected tissue, the designation of pathological deposit seems preferable to that of infiltration. (See *Degeneration* and *Deposits.*)

Tuberculous Infiltration.—The confluence of small miliary tubercles may give rise to a diffuse tuberculous process, or the epithelioid tissue of tubercles may infiltrate into the neighboring tissues after the manner of a new growth. Both of these conditions are included under the head of tuberculous infiltration. The term gelatinous infiltration was used by Laënnec to designate the grayish-red fibrinous exudate filling up the lung alveoli in the neighborhood of tubercles.

Syphilitic Infiltration.—Circumscribed or diffuse gummatous processes affecting bone were formerly designated as gummatous infiltrations.

Parasitic Infiltration.—The presence of large numbers of bacteria in the tissue spaces, the occurrence of the filaria in the lymph spaces and vessels, the invasion of muscle by trichina, etc., are frequently designated as infiltrations.

Aldred Scott Warthin.

END OF VOLUME IV.