The above formulæ are calculated for use with a 16-per-cent. cream. If 20-per-cent. centrifugal cream is used, the denominator of the cream formula will be 16 instead of 12, and if a 12-per-cent. cream is used it will be 8 instead of 12. It must, of course, be remembered that a milk with a very high fat and a very low proteid percentage cannot be made from cream with only a moderate percentage of fat. From a 16-per-cent. cream it is possible to make the fat percentage only four times the proteid, and with a 12-per-cent. three times the proteid. The great advantage of these equations is their simplicity and the ease with which odd amounts of milk mixtures can be calculated.

Sterilization.—In the infant the digestion of milk is for the most part accomplished in the small intestine. There is no long delay in the stomach with prolonged exposure to an acid and therefore antiseptic medium, as in the adult, but the milk after an imperfect curdling is quickly passed on to the duodenum. Gastric digestion, the safeguard in older children and in adults against the entrance of disturbing micro-organisms into the alimentary tract, affords in infants but a feeble barrier. For this reason one of the more important conditions in the infant's food is that it be sterile. This is the more necessary should the infant's food involve a longer time in its digestion than breast milk.

It is, therefore, extremely desirable that the milk employed in infant feeding be obtained with such careful precautions as to render it practically free from extraneous bacteria. It is not always our good fortune, however, to be able to obtain such with the regularity necessary for the daily preparation of an infant's food, and when our supply cannot be depended upon it appears the lesser of two evils to have the milk pasteurized at the lowest efficient temperature—that is, 60°–68° C. (140°–155° F.). Milk sterilized at over 70° C. is altered to an extent varying according to the elevation of the temperature employed and the duration of the exposure in the following respects: (1st) its proteids are modified and rendered less digestible; our knowledge on this subject, however, is still indefinite and dependent chiefly on clinical experience; (2d) the combination of its saline ingredients with the proteids is to some extent broken up and the salts assume a condition in which they are less readily absorbed; (3d) natural ferments, whose presence in milk may with much probability be inferred and which may materially assist its digestion in the infant's stomach, are destroyed; and (4th) an alteration takes place in the emulsion normal to milk, which may also have distinct effect in lessening its digestibility by the infant. For these reasons, it is important that the milk for infant feeding should not be sterilized at too high a temperature. After pasteurization it should be kept at a continuously low temperature; and it is to be remembered that the employment of sterilized milk which has been kept for many days is not free from danger.

Gärtner's Fettmilch.—In Germany during the last few years a modification of cows' milk has been prepared by Gärtner, as follows: Into the drum of a large centrifuge a mixture of milk and sterile water, at a temperature of from 10°–15° C., is allowed to run, care being taken that the milk used is obtained in the most cleanly manner from healthy cows properly fed. The drum of the centrifuge is made to revolve rapidly, driving the fat, as it is only in suspension, toward the centre of the drum, while the sugar, casein, and salts, which are in solution, remain uniformly distributed. Two tubes which carry off the milk enter the drum, one near the centre and one at the periphery. By arranging how near to the centre the inner tube opens, and the speed at which the drum revolves, milk containing fat in various percentages may be drawn off. As ordinarily set the milk drawn off near the centre has the same amount of fat as mother's milk, and is only deficient in sugar. Its composition, which is fairly constant, is fat 3, sugar 2.5, proteids 1.76. By adding a certain proportion of milk-sugar, milk closely resembling breast milk in composition is obtained.

This plan has also the advantage that any dirt present in milk and most of the bacteria are thrown off toward the periphery, thus leaving the central portion free. It is also claimed that the cream is not so completely separated from the milk as in Rotch's plan, but remains in a more perfect emulsion. Thus prepared the Fettmilch resembles ordinary milk in appearance and has an agreeable taste. Before using it as an infant's food it is recommended that it should be sterilized. Escherich has laid down the following rules for the amount and frequency with which this food may be given:

During each 24 hours children of 2 weeks require 16 ounces in 9 meals; from 2 to 4 weeks, 25 ounces in 8 meals; from 4 to 8 weeks, 33 ounces in 8 meals; from 3 to 4 months, 42 ounces in 8 meals; from 5 to 6 months, 50 ounces in 7 meals.

The employment of this milk is strongly recommended by many eminent German authorities.

Condensed Milk.—When pure fresh milk cannot be obtained, several writers have recommended the use of condensed milk. In the form of fresh evaporated milk (milk condensed without the addition of cane sugar), it may be of much service when pure fresh milk is impossible to obtain. The heat employed during the earlier stages of its preparation has rendered it sterile, and it will therefore keep well for a few days. For ordinary use it should be diluted with from seven to ten parts of water, and fresh cream, if this is obtainable, and sugar of milk added. Keating advised the following as making a useful preparation closely resembling breast milk: Preserved milk, 1 ounce; boiled water, 8 ounces; fresh cream, 1 ounce; sugar of milk, 3 drachms.

The ordinary canned condensed milk has the disadvantage of containing a large amount of cane sugar and a very small amount of fat. When diluted with six parts of water the resulting product on analysis yields fat 1, proteids 1.2, and sugar 8, the latter being chiefly cane sugar. This dilution is generally too strong for the majority of infants, and produces colic and indigestion. When diluted with twelve parts of water, its composition is fat 0.05, proteids 0.06, and sugar 4—a fluid altogether too weak to be efficient as a permanent diet. Holt states that he has not yet seen an infant who was reared exclusively on condensed milk which did not show on careful examination more or less evidence of rickets. During the summer season its use may be permissible for those who are unable to obtain fresh milk. In these cases Kerley advises that a weak meat broth or an albumin water be used as a diluent instead of boiled water, thus increasing the amount of the proteid and rendering it more valuable as a food. The deficiency in the fat may in many cases be supplemented by an emulsion of cod-liver oil.

Patented Food.—When for any reason failure has attended the attempt to feed an infant on cows' milk more or less modified, there is with many parents a strong inclination to resort to one of the numerous patent artificial foods placed before the public by interested agents who loudly vaunt their own special preparations as the exact analogue of mother's milk and the only food calculated to help the infant in its dire distress. We cannot deny that many of these foods have met with much popular favor and that some of them have proved of occasional service. It seems desirable, therefore, that the physician should have a knowledge of their physical properties and their composition further than the meagre and often incorrect statements contained in their advertisements. In almost all these foods dependence is put, for the greater part of the nourishment supplied, on milk which is either added by the user or previously introduced by the manufacturer. They are therefore to be regarded as modifications of cows' milk; a fact which may explain the apparently successful results which sometimes attend their employment.

With the exception of those which contain the milk in a condensed form, all, if administered alone, show a great deficiency in many of the most important ingredients of an infant food, and especially in the element of fat. It is to be noted also that they are secret preparations, over which we as physicians can exercise no supervision or control in regard to the quality of the material or to the amount of care used in their manufacture; that analyses made by many able chemists have given very variable results, often differing widely from the manufacturer's published composition; that they are liable to be old and musty; and that for the poorer classes they are an expensive food. To the analyses of Dr. Leeds we are principally indebted for a knowledge of their more exact composition. He grouped them into three classes. The first class contains the farinaceous foods prepared from the flour of one or more of the cereals, with the starch to some extent modified by baking. All of them, therefore, add a large proportion of starch to the milk dilution, and for this reason are not suitable for infants under six months. They are all liable to deterioration through long keeping. Among the best known of this class are the following: Imperial Granum, Blair's Wheat, Hubbell's Wheat, Hard's Food, Ridge's Food, and Robinson's Patent Barley.

In a second class are placed all those in which the starch is almost entirely converted into a mixture of dextrin, dextrose, and maltose, with a small amount of cane sugar. These foods have in some instances a distinctly laxative action, and may sometimes be of service when the infant is of a constipated habit. The principal foods of this class are Mellin's, Horlick's, and Malted Milk.

A third class comprises those foods to which the milk has already been added during the process of manufacture. All of them contain starch in considerable quantities, to a small extent only converted into dextrose. Their mode of preparation insures more or less complete sterilization of the milk, an important advantage during the summer season. Nestlé's Food, American Swiss, and Gerber's are examples of this class.

An additional class may be made of miscellaneous foods recently introduced, which do not conveniently fall under any one of these divisions, but which may be regarded as to some extent a combination of them. Carnrick's Soluble Food and Allen & Hanbury's Infant Food are examples of this class.

The following table of analyses, copied from Holt, represents the composition of several of these foods when prepared as directed for the infant's use:

INFANT FOODS DILUTED WITH WATER TO COMPARE WITH MILK.

	Breast milk. Per cent.	Condensed milk diluted 6 times. Per cent.	Mellin's food. Per cent.	Malted milk. Per cent.	Nestle's food. Per cent.	Ridge's food. Per cent.	Imperial Granum. Per cent.	Carnrick's soluble food. Per cent.
Fat	4.00	0.99	0.04	0.39	0.76	0.16	0.14	1.12
Proteids	1.50	1.20	1.50	2.28	1.54	1.67	1.98	1.35
Soluble carbohydrates (sugar)	7.00	7.23	11.56	10.18	6.38	.41	.25	4.06
Insoluble carbohydrates (starch)	.....	.....	.....	.....	4.19	10.91	10.65	5.61
Inorganic salts	.20	.17	.45	.50	.24	.07	.03	.56
Water	87.30	90.41	86.45	86.65	86.89	86.78	86.92	87.30

Amount to be Given.—Not only is the character and composition of the food itself of importance, but the amount to be given and the times of giving it demand the physician's careful attention. As Biedert remarks: the infant has no instinct which guards against over-feeding. Even breast-fed children are apt to drink too much, but those artificially fed have a greater tendency to excess, since in general they obtain their food more quickly and with less exertion on their part. The truth should be carefully impressed upon mother and nurse, that a bottle-fed infant thrives best upon the minimum amount of food necessary for good development; excess tends to interfere with digestion, and thus unfavorably affects nutrition.

Dr. Rotch quotes the results of Smitkin's investigations at the Children's Hospital in St. Petersburg, to determine the amount that should be given during the first few weeks of life. He finds that the greater the weight the greater is the gastric capacity, and says that one-one-hundredth of the initial weight of the infant should be taken as the starting figure for each meal, and to this should be added 1 gm. for each day of life.

Illustrations of above rule to serve as guides for especially difficult cases.

Initial weight.	Each feeding. Early days.	At 15 days.	At 30 days.
3,000 gm	30 gm. = (about ʒ i.).	30 + 15 = 45 gm. (about ʒ iss.).	30 + 30 = 60 gm. (about ʒ ij.).
4,500 gm	45 gm. = (about ʒ iss.).	45 + 15 = 60 gm. (about ʒ ij.).	45 + 30 = 75 gm. (about ʒ iss.).
6,000 gm	60 gm. = (about ʒ ij.).	60 + 15 = 75 gm. (about ʒ iss.).	60 + 30 = 90 gm. (about ʒ ij.).

The following table (from Rotch) will afford an indication, for an average infant, of the amount to be given at each feeding and the number of feedings which should take place in the twenty-four hours:

GENERAL RULES FOR FEEDING DURING THE FIRST YEAR. (Rotch.)
(The Day feedings are supposed to begin at 6 A.M. and to end at 10 P.M.)

Age.	Intervals in hours.	Number of feedings in 24 hours.	Number of night feedings.	Amount at each feeding. Ounces.	Total amount in 24 hours. Ounces.
One week	2	10	1	1	10
Two weeks	2	10	1	1½	15
Four weeks	2	9	1	2½	22½
Six weeks	2½	8	1	3	24
Eight weeks	2½	8	1	3¼	26
Three months	2½	7	0	4	28
Four months	2½	7	0	4½	31½
Five months	3	6	0	5½	33
Six months	3	6	0	5¾	34½
Seven months	3	6	0	6¼	37½
Eight months	3	6	0	7	42
Nine months	3	6	0	7	42
Ten months	3	5	0	8½	42½
Eleven months	3	5	0	8¾	43¾
Twelve months	3	5	0	9	45

Very rarely, in the cases of immature and very sickly babies and of infants with cleft palate, the ordinary feeding-bottle is ineffectual, owing to the infants not having power to suck. Feeding by spoon is slow and wearisome to the infant. In these cases Dr. Jacobi recommends the use of Soltmann's *biberon pompe*. In this feeding-bottle simple pressure on the mouthpiece, either by the lips or gums of the infant, or on the expanded portion of the tube outside the bottle by the fingers of the nurse, will cause the food to escape from the bottle. Dr. Jacobi says that in many cases it has done good service. Dr. Rotch recommends the use of a glass cylinder, graduated and capable of holding nine drachms. One end gradually tapers till a small nipple can be fitted on to it; the upper end is covered by a compressible rubber bulb. By gentle compression of this bulb the food may be slowly forced into the infant's throat without any effort on its part.

This method appears to be specially desirable in the case of a weak infant, as it involves no loss of strength.

Gavage.—Some years ago Dr. Tarnier, of Paris, advocated the method of gavage in infants prematurely born or in such as are congenitally weak. The apparatus necessary for the purpose is very simple: a soft-rubber catheter, No. 14 or 15 French, or a piece of gutta-percha tubing with a small glass receptacle, of sufficient size to hold the amount of milk to be administered, attached to the distal end. The child is placed on the knee of the nurse with its head slightly raised. The tube is moistened with milk and introduced at the base of the tongue and then very gently forced down the œsophagus to a distance of 15 cm. The sound is then pinched between the two fingers and the food is introduced into

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.

Alexander D. Blackader.

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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.)

Aldred Scott Warthin.

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.