optic thalami, whose action is in part at least to prevent the reflex closure of the sphincter under the stimulation of the sensory nerves of the rectum by the descent of fæces. In the infant defecation is probably entirely involuntary. In certain diseases of the brain or spinal cord, in sleep, or in intoxication, involuntary evacuation sometimes occurs, and certain psychic states, the disturbance of the emotions as by fright or fear, are capable of inducing it. Such accidents are attributable either to the removal of some cerebral control or to the action of the inhibitory centre.

Defecation begins ordinarily with the voluntary inhibition or withdrawal of the action of the external sphincter. This inhibition is of limited duration, however. It may be terminated voluntarily, but it cannot be continued indefinitely by the action of the will; a rather sharp contraction, as a rule, follows the passage of a fæcal mass. Relaxation of the sphincter is accompanied by a contraction of the abdominal muscles and a variable action of the diaphragm, corresponding to the amount of expulsive force required. The sensation produced by the distention of the rectum is peculiar and should not be disagreeable, but in some diseased conditions of the bowel it becomes exaggerated into a painful tenesmus. If the peristaltic action has been strong and the demand for evacuation imperative, the diaphragm is not necessarily brought into requisition, but when additional force is required, as in constipation, the diaphragm is depressed, causing full inspiration, and its ascent is prevented by a closure of the glottis. In this way and by the subsequent contraction of the abdominal muscles, not only is the rectum emptied, but the passage of the contents of the colon and sigmoid flexure into the rectum is assisted. Defecation is assisted also by the levator ani muscles, whose contraction tends to draw the sphincters upward over the fæcal mass. The rectal peristalsis can be increased also by repeated voluntary contractions of this muscle and the external sphincter. During defecation the mucous membrane of the anus is slightly everted, especially when the expulsive force is strong. After the completion of defecation the sphincters relax to some extent, as their contraction is not required to retain the closure of the anus in the intervals between the acts of defecation.

It is generally admitted that defecation should occur once in twenty-four hours, yet it may occur twice or thrice within that limit of time or only once in two or three days with apparently normal regularity. Its frequency is largely a matter of habit with the individual. In cases of habitual constipation evacuations may occur as seldom as once in a week, but in inflammatory diseases of the intestine they may occur at intervals of only a few minutes. The frequency and character of defecation are often valuable factors in diagnosis.

James M. French.

DEGENERATIONS AND DEPOSITS, PATHOLOGICAL.—Among the most important retrograde changes are those classed as the degenerations and the deposits. While the term tissue degeneration is very often loosely applied to all kinds of retrograde changes, it is also used in a specific sense to indicate that particular class of retrograde change which is characterized by the formation of new substances out of the cell protoplasm. These substances may be retained within the cell or discharged from it. Any pathological increase in the production of substances normally manufactured by the cell would be included under this head. To avoid the confusion arising from the loose use of the term degeneration, it might be of advantage to class these changes as the *true degenerations*. The *deposits* or *infiltrations*, on the other hand, are characterized by the deposit in the tissues of pathological substances which have either formed within the body or have been introduced into it from without. The deposit is often a result of degeneration either at the point of deposit or elsewhere in the body, or it alone may represent the chief feature of the pathological change. It is very difficult sometimes to draw a distinct line between degeneration and deposit, since identical or similar substances may be both formed within the cell out of its own protoplasm or be brought to it, and deposited either in it or about it. Some substances such as fat, melanin, glycogen, etc., thus play the rôle of both degeneration and deposit; while others such as mucin, pseudomucin, amyloid, etc., occur in but one form, either that of a degeneration or that of a deposit as the case may be. Many of the substances which are regarded as deposits are usually found in or near the walls of the smaller blood-vessels, suggesting the possibility of their formation through changes in the secreting cells of the vessel wall. The new substances are either taken out of the blood by the endothelial cells or are formed by the protoplasm of the latter, and passed out into the lymph spaces of the surrounding tissue after the manner of a secretion. Both amyloid and hyalin appear to be formed in this way.

True Degenerations.—1. Cloudy swelling. 2. Hydropic degeneration. 3. Fatty degeneration. 4. Colloid degeneration. 5. Mucin. 6. Pseudomucin. 7. Cholesterin. 8. Epithelial hyalin. 9. Cornification. 10. Colloid-like bodies. 11. Pigments formed by cell activity. 12. Glycogen.

Deposits.—1. Fat. 2. Amyloid. 3. Hyalin. 4. Calcification. 5. Uric acid, urates, etc. 6. Cholesterin, cystin, xanthin, etc. 7. Glycogen. 8. Pigment. 9. Extrinsic substances.

True Degenerations.—*Cloudy Swelling* (Parenchymatous, Granular, or Albuminous Degeneration).—This is the most common form of the true degenerations and is characterized by the splitting up of the cell protoplasm into fluid and albuminous granules. These granules are soluble in acetic acid, insoluble in alkalies and ether, and are not affected by osmic acid. Their presence gives to the cell a cloudy granular appearance: it is swollen and its normal form and structure are lost. In slight degrees of this change the nuclei show but little evidences of disorganization, but in severe cases there are disintegration and loss of staining power of the chromatin. Recovery from the moderate degrees of this degeneration is possible, but after diffusion of the nuclear chromatin the cell undergoes complete destruction, breaking up into a finely granular débris. Cloudy swelling occurs in the cells of the parenchymatous organs, particularly in the liver and kidneys, in the course of the acute infectious diseases, severe intoxications, etc. It is the chief lesion in the kidneys in the acute degenerative nephritis occurring in scarlatina, septicæmia, eclampsia, variola, erysipelas, diphtheria, typhoid fever, etc.; and in such intoxications as mercuric chloride, carbolic acid, phosphorus, arsenic, cantharides, etc. To the naked eye the affected organs appear larger, swollen, less shining than normal, and of a grayish color. In severe cases the organ has the appearance of having been boiled, and possesses a color and consistence similar to that of putty. Fatty degeneration is often associated with cloudy swelling.

Hydropic Degeneration.—In hydropic degeneration there is a partial liquefaction of the protoplasm, leading to the formation of clear vacuoles within the cell. The nucleus may be similarly affected, being indicated by the presence of a large clear globule consisting of the nuclear membrane distended by fluid. This degeneration occurs most frequently as a precursor to liquefaction necrosis, and is found in blisters of the skin, tumor cells, severe œdema, inflammatory processes, etc. It is distinguished from œdema in that the staining power of the nucleus is affected, and from liquefaction necrosis in that in the latter the nucleus is entirely lost.

Fatty Degeneration.—This form of degeneration is characterized by the formation of fat out of the albumin of the cell body. Cells which are in a state of fatty degeneration contain small, colorless, highly refracting droplets which are soluble in alcohol and ether, insoluble in acetic acid, and stain black with osmic acid. These droplets are very irregular in size and shape, but are usually small except in severe cases, in which they may become confluent, forming larger drops. The parenchymatous organs, liver, kidneys, etc., are chiefly affected, but the degeneration occurs also very frequently in heart muscle, voluntary muscle, connective-tissue cells, and leucocytes. Fatty degeneration occurs in a great variety of pathological conditions: acute infectious diseases such as diphtheria, pneumonia, septicæmia, etc.; poisoning by phosphorus, arsenic, chloroform, camphor, etc.; acute anæmia resulting from severe hemorrhage; chronic anæmia and leukæmia; chronic congestion; diminished blood supply caused by sclerosis, thrombosis, etc.; chronic pulmonary tuberculosis, nephritis, chronic alcoholism, etc. The degeneration is dependent partly upon changes in the nutrition of the cell and partly upon a lowered vitality of the cell. The chief factor is a diminished supply of oxygen to the cells, which results in an increased breaking down of the cell albumin, partly into fat and partly into nitrogenous products which are excreted by way of the urine. In the infectious diseases the degeneration must be attributed primarily to the effects of the poisons produced in these diseases, or to the effects of fever. Prolonged high temperatures lead to extensive fatty degeneration, especially of the heart muscle. Cells out of their normal environment, tumor cells, pus cells, etc., also undergo fatty degeneration. Cloudy swelling and hydropic degeneration are very frequently associated with fatty change.

Colloid Degeneration.—Colloid is a more or less firm, hyaline, jelly-like substance, clear or slightly colored, and is produced by the cells of the thyroid follicles. Its chemical nature is unknown; it is probably an albuminous body containing iodothyrin. It is not precipitated by alcohol or acetic acid. When formed in excessive amount the follicles become distended and the gland enlarged (colloid degeneration of thyroid, colloid goitre or bronchocele). This pathological accumulation may occur both in the normal gland and in tumors arising from it. Substances similar to thyroid colloid are found in various conditions of the kidneys, prostate, parotid, etc. Since their chemical nature is not identical, they are classed as *colloid-like bodies*.

Mucin, Pseudomucin.—The pathological formation of a stringy, gelatinous fluid from epithelial cells or connective tissues is known as mucous or myxomatous degeneration. This change has a physiological prototype in the formation of mucus in mucous membranes and glands, and in the umbilical cord, tendons, bursæ, and synovial membranes. In mucous membranes the production of mucus takes place in goblet cells. These are swollen epithelial cells whose protoplasm has become altered into a clear substance containing small granules or strings. In mucous degeneration of epithelium the number of goblet cells becomes greatly increased (catarrhal inflammation). Pus cells, the epithelium of cystadenomata, and carcinoma cells very frequently undergo mucous change. In mesoblastic structures the intercellular substance undergoes a mucous change, becoming altered into a gelatinous mass. The cells of the tissue may also be converted into mucus. This change occurs in fibrous connective tissue, cartilage, bone, bone marrow, and fat, and especially in the case of the mature connective-tissue tumors and the sarcomata. In severe cases the entire tissue may become changed into a clear, translucent mass. The chemical nature of mucus varies greatly in different cases, and at present but little is known of its real composition. Its chief constituents are known as mucin and pseudomucin, and several varieties of these are described, so that it is probable that they do not represent single chemical substances. Mucin is precipitated by acetic acid while pseudomucin is not. From both mucin and pseudomucin a carbohydrate may be formed, from which fact one is justified in classifying them as glyco-proteids. Pseudomucin is found especially in the cystadenomata of the ovary and is the product of the epithelial cells lining the cysts. It is found also in mucous carcinomata.

Cholesterin.—This occurs as a pathological degeneration in tissues and exudates which are in process of fatty degeneration. Its source is not clearly understood, but it is probably an intermediate product in the splitting up of proteids. It occurs in the form of thin, rhombic plates having usually a notched corner, in atheromata, fatty degeneration of sclerotic vessels, old extravasations, and purulent exudates.

Epithelial Hyalin.—Under this head may be grouped all of the substances that arise from the degeneration of epithelial cells and that have a structure like that of the colloid of the thyroid. These substances are, however, very different in origin and chemical nature and cannot be regarded as being identical in kind. It is therefore better to restrict the use of the term hyalin to those degeneration products of epithelial cells which resemble the hyalin of connective tissue in that they stain with fuchsin. Those substances which resemble colloid may then be classed as the colloid-like bodies. As epithelial hyalin may be considered the hyaline granules and globules found especially in cancer cells. These have been held to be parasites, but they are formed as the result of degenerative changes in the cancer cell.

Colloid-like Bodies.—Those pathological substances found in various portions of the body which bear a close resemblance to the colloid of the thyroid, and which like it are the products of epithelial cells, may be classed as colloid-like bodies. The chemical composition of these bodies cannot be identical; their grouping together into one class is based upon general resemblances only. They represent transformed products of epithelium, and in common are hyaline, gelatinous substances which do not stain like mucin. Such bodies occur pathologically in the kidney tubules (casts), ovarian and parovarian cysts, retention cysts in mammary glands, parotid, skin glands, pancreas, kidneys, endometrium, etc. Similar substances are found also in the prostate, hypophysis, central nervous system, lungs, etc. They occur partly in the shape of homogeneous hyaline bodies and partly in the form of laminated concretions. Some of the latter give a reaction resembling that of amyloid (corpora amylacea). Since in the majority of cases these bodies are found in gland spaces, they must be regarded as a modified epithelial product which is formed under conditions very different from those attending the deposit of amyloid, and though occasionally giving a similar reaction cannot be considered as being identical. The colloid-like bodies found in the nervous system are derived either from degenerating neuroglia cells or from fragments of axis cylinders.

Cornification.—Pathological cornification occurs in a great variety of conditions. Hyperplasia of the horny layer of the entire skin may occur (ichthyosis, lichen pilaris, etc.), or local thickening may take place (ichthyotic warts, callosities, corns, etc.). These changes may be included under the general term of hyperkeratosis. Pathological cornification also occurs in regions of the body where normally it does not take place at all or but to a very slight degree. The ducts of the skin glands may be so affected; cornification may also occur in the mucous membrane of the mouth, middle ear, vagina, urinary passages, in the mastoid cells, etc. Further, keratohyalin is formed in tumors of the skin, meninges, brain, etc. In all of these conditions the process is a true cell degeneration, the keratin being formed by the cells at the expense of their nuclei. As the keratohyaline granules are formed and escape from the cells there is at the same time a shrinking of the nuclei, leading ultimately to their disappearance.

Cell Activity.—The pigments which are formed by cell activity—melanin, hæmofuscin, and lipochrome—may all be produced in excessive amounts under certain pathological conditions. This increase of pigment manufacture is analogous to the increased production of mucus, colloid, etc.; and for this reason it may be classed with the true degenerations. An excessive formation of melanin occurs in local melanosis of the skin (pigmented moles, sun-burn, etc.); in general melanosis, in Addison's disease, and in severe cachexias. Melanin degeneration occurs also in the cells of melanotic sarcomata. In these, death of the cell usually follows the formation of the pigment. Hæmofuscin is produced in excessive amount

in the atrophy of heart and unstriped muscle. Lipochrome is formed to such an extent in certain forms of sarcoma as to give them a green color (chloroma).

Glycogen.—Glycogen is formed in excess in certain cells and tissues in a number of pathological conditions—in pus cells, in the leucocytes in certain cachexias, and in tumor cells of various kinds. It is not clear whether in these cases the glycogen is derived as such wholly from the blood or is manufactured by the cell from carbohydrates or albumin.

DEPOSITS.—*Fat.*—A pathological deposit of fat may take place in tissues where fat is found normally or in those in which it is not a normal constituent. In excessive accumulation of fat the subcutaneous and subserous adipose tissue, bone marrow, and liver are first affected: later, the intermuscular connective tissue of the heart and voluntary muscles, endocardium, etc., may become the seat of deposit. The fat is deposited in the shape of small droplets which become confluent into larger drops, so that ultimately the entire cell body may be replaced by it and converted into a globular mass of fat. Fatty infiltration occurs in general obesity, congenital and acquired; in chronic anæmia and cachexia; in chronic alcoholism, etc. The causes of pathological accumulations of fat are: excess of nutritive material taken into the body, inability on the part of the body to break down the fat received into it or already stored there, deficient oxygenation of the blood and tissues, chemical substances reducing the metabolic activity of the cells, etc.

Amyloid.—The terms amyloid and lardaceous degeneration have been applied to the deposit of a hyaline, homogeneous, wax-like substance in the walls of the smaller blood-vessels. It may occur in almost every part of the body, but is most commonly found in the intestine, stomach, lymph glands, pancreas, adrenals; and rarely in the muscles, ovaries, uterus, respiratory tract, etc. As a result of an extensive deposit of amyloid the affected organs become enlarged, translucent, waxy and more resistant. The amyloid substance gives a reaction with iodine somewhat resembling that of starch, and for this reason it was once regarded as a body closely related to cellulose or starch; hence its name amyloid. Later, its nitrogenous nature was discovered, and it is supposed to be a coagulated albuminous body intermediate between the proteids on the one hand and fat and cholesterin on the other. It is very resistant to the action of chemicals and the agents of putrefaction. With certain of the aniline dyes amyloid exhibits a metachromatic reaction. Amyloid is never deposited in living cells, but is found in the interstices of the connective tissue in or near the walls of the smaller blood-vessels. In the early stages of the deposit it lies always just outside of the endothelium. It is therefore probable that it is formed by the secreting cells of the vessel walls and deposited in the lymph spaces in and near the vessels. But little is known concerning its significance and etiology. It occurs most frequently in the various cachexias, especially in pulmonary tuberculosis, chronic suppuration, syphilis, chronic dysentery, leukæmia, etc.

Hyalin.—Closely related to amyloid in its general appearances and mode of formation is the homogeneous deposit found in the walls of blood-vessels and in the interstices of connective tissue which is known as hyalin. It differs from amyloid chiefly in that it does not give the characteristic reactions of the latter. That there exists a very close relationship between the two substances is shown by the fact that amyloid organs often contain hyaline areas which do not give the amyloid reactions, and that pieces of amyloid when introduced into the peritoneal cavity of animals lose their characteristic reactions. In further contradistinction hyalin stains a deep red with Van Gieson's method, while amyloid stains a pinkish-yellow or brown. Hyaline deposit is found chiefly in the walls of blood-vessels (sclerosis), endocardium, connective tissue of the thyroid, lymph glands, ovaries, chronic inflammations of the kidneys, and in the connective tissue of many forms of tumors. In the connective tissue of certain organs, especially in that of the conjunctiva, hyalin appears to be more of the nature of a degeneration than of a deposit. The connective-tissue cells may undergo a change into a hyaline substance containing no nuclei, or the hyaline formation may owe its origin to a secretory activity on the part of the connective-tissue cells. Conjunctival hyalin appears to be formed in this way. In other cases hyalin partakes both of the nature of a degeneration and of that of a deposit, the connective-tissue spaces being first filled with a clear, homogeneous substance into which the cells are gradually fused, the whole ultimately becoming converted into hyalin. The small hyaline granules (fuchsinophile bodies) found in connective tissue are also included by some writers under the head of hyalin. They are partly the results of cell degeneration and partly derived from inflammatory exudates. The deposit of hyalin occurs under conditions similar to those in which amyloid develops: it is found in old age, syphilis, various cachexias, chronic inflammations, etc.

Calcification.—The deposit of lime salts in the body tissues is of very frequent occurrence. Such deposits may consist either of granular and amorphous masses or of crystals. They may occur in the tissues which form a normal part of the structure of the body, or an incrustation may be deposited around tissues which have been separated from their normal surroundings; or foreign substances which have entered the body may become the centre of deposit. In the first case the process is spoken of as calcification of tissue; in the latter as the formation of concretions and calculi. Calcification never takes place in perfectly normal tissue. It is usually preceded by some degenerative change: cloudy swelling, fatty degeneration, hyaline change, necrosis, etc. Dying tissue which has undergone more or less change appears to possess a certain attraction for the lime salts held in solution within the body and to cause their precipitation. Connective tissue which has undergone hyaline change is especially likely to become calcified. Calcification occurs also in areas of caseation in any part of the body, in tissues showing fatty degeneration or beginning necrosis, as in the renal epithelium in cases of poisoning by mercuric chloride, aloin, and bismuth. In old age extensive calcification may occur in tissues which show but little change. This takes place to the greatest extent in cases showing marked osteoporosis. The lime salts removed from the bones are deposited in the capillary walls in the lung, kidneys, and mucosa of the stomach, as well as in the intima of the smaller cerebral vessels. Concretions of lime salts are met with in the meninges, caseating areas, psammomata, nodular growths in connective tissues, organizing thrombi, etc. Calculi composed partly or wholly of lime salts may be found under varying conditions in all of the ducts and cavities of the body (salivary, bronchial, urinary, biliary, prostatic, etc., calculi). All of these possess an organic basis around which the deposit has taken place. The calcium salts found in the body are either the phosphates, the carbonate, or the oxalate. Magnesium salts are not infrequently found in combination with these. Other substances such as uric acid, bile pigment, etc., are also found mixed with lime salts in various forms of calculi.

Uric Acid, Urates.—A deposit of uric-acid salts takes place in gout, chiefly in the form of sodium urate, with which small quantities of calcium phosphate and carbonate are combined. These deposits occur in the kidneys, skin, subcutaneous tissues, tendon sheaths, tendons, ligaments, synovial membranes, and articular cartilages. The great toe is a favorite seat of deposit. In marked cases every organ in the body may be affected. Fine acicular crystals of sodium urate are found in the tissues, which show at the seat of deposit a more or less advanced necrosis. The larger deposits about the joints are called tophi. Uric acid and urates are found in the kidney tubules in the condition known as uric-acid infarct, which is of rather frequent occurrence in the new-born. Calculi of the same substances are found frequently in the tubules and pelvis of the kidney, the ureters, and in the bladder.

Pure uric-acid calculi are small, hard, and reddish in color. Calculi of urates are usually partly composed of lime salts.

Cholesterin, Cystin, etc.—Cholesterin is found as a deposit in gall stones. These may consist of pure cholesterin or be combined with bile pigment, lime salts, etc. Cystin calculi are found in rare conditions when cystin is excreted by the kidneys as the result of peculiar metamorphoses of albumin produced in the intestine through the action of bacteria. Cystin calculi are yellowish, soft, and waxy. Calculi composed of xanthin are found rarely in the bladder. They are red in color, and of a soft, friable consistence.

Glycogen.—In diabetes excessive amounts of glycogen are conveyed by the blood to certain organs where it is deposited in the parenchymatous cells. This takes place to the most marked extent in the kidneys where the glycogen is deposited in the epithelium of Henle's loops, especially in the isthmus of these where the cells are almost entirely filled with it. It appears in the cells in the form of hyaline droplets which are usually collected near the nuclei. After the glycogen has been dissolved out by water clear vacuoles are left in the cell.

Pigment.—Melanin may occur as a deposit in various organs, especially in the spleen, lymph glands, and kidney, in the case of necrosing melanotic sarcomata in which the pigment is either set free in the blood or lymph or carried there by leucocytes. It may also appear in the urine, and casts of melanin may be found in the renal tubules. In icterus the bile pigment may be deposited in all of the tissues and organs of the body, giving to them a yellow or greenish color. Hæmatogenous pigments, hæmatoidin and hæmosiderin, are found in the tissues as remains of hemorrhages. The pigment granules may be taken up by leucocytes and carried to the spleen, lymph glands, bone marrow, etc., being deposited in these organs in the shape of yellow or brown granules which may be so numerous as to impart a distinct color to the part. In many of the acute infectious diseases, malaria, pernicious anæmia, leukæmia, various forms of poisoning, etc., large numbers of red blood corpuscles may be broken down and an increased amount of blood pigment be carried to the spleen, liver, kidneys, etc. This may be compensated for by an increased activity on the part of the liver in the formation of bile, but if the amount of hæmoglobin is too great to be disposed of in this way pigment may be deposited in the liver cells and in other organs and tissues. Such organs become yellow or brown if the deposit is at all marked, and to this condition the term hæmochromatosis has been applied. Since hæmosiderin is the pigment most commonly found it is proper to speak of the pigmentation as a hæmosiderosis. In the liver cells hæmatoidin is usually deposited in the central zone of the lobule, while hæmosiderin is found in the periphery of the lobule. In the kidneys the pigment is deposited chiefly in the cells of the convoluted tubules; in the spleen, lymph glands, and bone marrow it is contained chiefly within the endothelial cells lining the blood spaces. Through the absorption of hydrogen sulphide from the intestine, organs containing hæmosiderin in excess may become black in color (pseudo-melanosis). In addition to the hæmotogenous pigments formed in malaria through the destruction of the red blood cells, the pigment formed by the plasmodia themselves may collect in the smaller arterioles and capillaries. It is black, contains no iron, and its exact chemical nature is unknown.

Extrinsic Substances.—Foreign substances may be introduced into the body from without and be deposited in the tissues. The variety of such substances is very great and their mode of entrance into the body is also greatly varied. Insoluble substances may be introduced through open wounds of the skin (tattooing, trauma). The lungs are the most frequent point of entrance: through them coal dust, stone and iron dust, soot, etc., enter and are deposited in the lung tissues and in the bronchial lymph glands. Under certain conditions (tuberculous caseation, etc.) these substances may get into the general circulation and be deposited in the liver, spleen, bone marrow, etc. Soluble metallic salts, such as those of silver and lead, may be taken in through the stomach and intestine, and deposited in certain organs and tissues in an insoluble form.

The causes leading to the pathological degenerations and deposits may be either intrinsic or extrinsic. The former may be inherited or it may arise by primary germ variation. For the greater part both degenerations and deposits are produced by injurious extrinsic influences to which the body is exposed during life. Disturbances of nutrition, deficient oxygenation, circulatory disturbances, intoxications, infections, etc., play the chief part in their production, but any external force which may injure the body may lead likewise to pathological alterations of its organs or tissues which may assume either the form of a degeneration or that of a deposit. These changes may be of limited extent or the entire organism may suffer as the result of general disturbances of nutrition. Deposits may be followed by secondary retrograde changes, such as atrophy, fatty degeneration, etc., as the result of pressure or diminished blood supply. No general statements can be made regarding the course and symptomatology of the degenerations and deposits, as these vary within the widest limits according to their etiology and association with other pathological conditions. (See also *Amyloid, Colloid, Concretions*, etc.)

Aldred Scott Warthin.

DEGLUTITION.—Deglutition is the physiological act or process by which food is conveyed to the stomach. There are two principal theories in regard to its mechanism, one based upon the description of Magendie, the other upon the investigations of Kronecker and Meltzer.

Following Magendie, it is customary to describe the act of swallowing as occurring in three stages, each representing the passage of the food through one of the three anatomical regions involved, namely, the mouth, the pharynx, and the œsophagus. After the food has been masticated and collected into a bolus, the mouth is closed and the jaws are brought together. Deglutition then begins with the elevation of the tongue against the palate, its tip, middle, and base pressing upward in succession, through the action of its intrinsic muscles and the stylo-glossus. The bolus is thus forced backward through the isthmus of the fauces into the pharynx. During its passage the soft palate is elevated by the contraction of the levator palati muscles and is made tense by the tensor palati; the pillars are made straight and tense through the action of the palato-pharyngeus, which also assists in the closure of the posterior nares. There is at the same time a slight protrusion forward of the posterior wall of the pharynx through the contraction of the salpingo-pharyngeus, and the uvula is elevated by the azygos-uvulæ to complete the closure of the passage into the posterior nares.

This, the first stage, is regarded as voluntary in character; but it is not fully under control of the will, for in the absence of a moist bolus or of fluid to be swallowed the act becomes difficult and cannot be repeated indefinitely; the presence of dry food or of a dry powder on the tongue exerts an inhibitory influence.

The second stage consists in the passage of the food through the pharynx, which is accomplished by a number of rapid muscular movements. The contraction of the stylo-pharyngeus and palato-pharyngeus muscles raises the pharynx like a funnel and tends to draw it over the descending bolus, which is now seized and carried rapidly downward by the constrictors of the pharynx. The approximation of the anterior pillars of the fauces through the action of the palato-glossi prevents its regurgitation. The other muscular movements which accompany these have for their object the closure of the glottis. This is accomplished by the elevation of the thyroid cartilages behind the hyoid bone through the action of the laryngeal muscles; the approximation of the arytenoid cartilages and the vocal cords by the lateral crico-arytenoids and the constrictors of the glottis; and the depression of the epiglottis, which is sometimes ac-

complished, perhaps, in a purely mechanical way, but is normally brought about by the concerted action of the thyro-hyoids, digastrics, genio-hyoids, mylo-hyoids, and the fibres in the aryteno-epiglottic folds.

The third stage is a much slower movement, requiring six seconds for its completion. The food enters the œsophagus with great rapidity and continues to move rapidly through its upper third, the part supplied with striated muscle fibres. Through the lower two-thirds the movement is slower, depending solely upon the involuntary musculature.

The Kronecker-Meltzer theory attributes the most important part in deglutition to the contraction of the mylo-hyoid muscle and is based upon carefully conducted experiments. After the bolus of food has been collected on the dorsum of the tongue and its passage forward has been prevented by the elevation of the tip against the palate, these muscles contract with force and put so great pressure upon the bolus that it is shot through the pharynx into the œsophagus, the action being assisted by the simultaneous contraction of the hypoglossi which draw the tongue backward and downward and thus increase the pressure. This action also serves to depress the epiglottis and to protect the larynx. Soft or liquid food passes rapidly through the œsophagus to the cardiac orifice of the stomach, the time required for its descent being not more than one-tenth of a second. The peristaltic wave of the œsophagus is regarded as a reserve movement intended for the removal of such fragments as lodge in descent or for the completion of deglutition when the bolus is not of a proper character for rapid descent. It is needed, however, in many individuals to force the food through the cardiac constriction. Six seconds are required for the completion of the act in this manner. The œsophagus contracts in three successive segments. The first is about 6 cm. long and is the neck portion, the part endowed with striated muscle. It begins to contract in about one or one and two-tenths seconds after the beginning of deglutition and requires two seconds for its completion. The second segment, the upper thoracic portion, is about 10 cm. in length; its contraction begins about one and eight-tenths seconds after the first segment begins to contract and lasts for from six to seven seconds. The remaining third portion begins to contract about three seconds later than the second and continues for from nine to ten seconds. The act of deglutition is thus divided into five instead of three stages, corresponding to the contraction of the mylo-hyoids, the constrictors of the pharynx, and the three œsophageal segments. A single act of swallowing requires six seconds for its completion, as stated; but if the act be repeated within this time, it was observed that the peristaltic wave is arrested and that the bolus is carried to the stomach by a subsequent wave which is completed six seconds after the beginning of the last deglutition.

Nervous Control.—Deglutition is a reflex act. A certain stimulus, as that of food or drink or a few drops of saliva, seems to be required for the perfect accomplishment of even the first stage, the most voluntary part of it. The afferent impulse is conveyed, presumably from the mucous membrane of the mouth or pharynx, to the deglutition centre by the glosso-pharyngeal, trifacial, and pneumogastric through the internal branch of its superior laryngeal division. No deglutition centre has been definitely located, but it is supposed to lie near the anterior surface of the medulla. It probably comprises parts of the nuclei of origin of the nerves employed in the act of swallowing, namely, the hypoglossal, facial, trifacial, glosso-pharyngeal, and pneumogastric, and is not an individual collection of cells.

Mosso found that when an entire segment of the œsophagus is removed the contraction of the part below the injury takes place in its natural order as though the tube were intact. Kronecker and Meltzer observed the same action after ligature or division. This is made possible by the manner in which the tube is innervated by branches of the pneumogastric and sympathetic. These form a plexus between the muscular coats in which are groups of ganglion cells and another in the submucous layer. Although the part taken by each of these cannot be exactly described, the impulses are conveyed through them in such a manner that the wave of contraction travels downward from the upper portions to the lower regardless of the integrity of the tube. *James M. French.*

DELIRIUM.—The term *delirium* does not indicate any special disease, but a psychopathic condition, which may be observed during the course of a great many mental diseases. This condition is characterized by an incoherence in the chain of conceptions, and by the appearance of symptoms of psycho-sensory and psycho-motor irritation. The incoherence of conceptions is evident in the disconnected and confused speech and in the aimless movements of the patient. Hallucinations are produced by the psycho-sensory irritation, and the psycho-motor irritation is responsible for the impulsive movements sometimes manifested in a very high degree.

As is well known, delirium appears most frequently in the course of the acute infectious diseases. One must, however, bear in mind that not every psychical disturbance during these diseases must necessarily be a delirium. Isolated delusions, maniacal excitation, and acute hallucinations appear here just as frequently as delirium, and it is well to distinguish sharply between these conditions as they may each have a different significance. A genuine delirium indicates often a state of inanition and demands energetic stimulation.

Delirium may appear in the course of various mental diseases, and in such instances always indicates general exhaustion. It is of bad prognostic significance when it develops gradually under these circumstances, and is not infrequently the first sign of approaching death. There seems to be a special tendency toward the development of delirium in the course of functional psychoses which are complicated by some organic disease, such as a valvular lesion of the heart, a chronic nephritis, etc. Frequently delirium arises in organic diseases of the brain as a sequel to some acute lesion of the brain substance, such as minute hemorrhages, emboli, or œdema.

When a delirium suddenly develops without warning in a previously healthy person, a correct judgment of the case becomes much more difficult than when it appears in the course of certain diseases. These are the cases which the general practitioner sees in relative frequency. A person, who has heretofore been perfectly well, suddenly becomes confused in speech, runs aimlessly about his room, and appears to recognize no one about him. The nearest physician is sent for and recognizes an acute delirium. The question then is: What is the nosological significance of such an acute delirium? Is it an independent disease, which has suddenly developed, or is it but a complex of symptoms, which may be evoked by different diseases, and, if so, what means have we at our disposal to clear up the pathological condition?

The relative frequency of acute delirium gave rise to the idea that these attacks were due to a special form of disease, which was described under the name of "delirium acutum." This view, however, has been proved to be erroneous. An acute delirium does not constitute a *morbus sui generis*, but represents only a transitory condition of the most varied mental disorders. Let us, therefore, consider the various diseases which may be responsible for an attack of acute delirium and attempt, at the same time, to bring out the different points which may lead us to a correct diagnosis in every individual case.

The previous history of the patient may be of great value in our conclusions concerning an acute delirium. A history of previous epileptic attacks, for example, may put us upon the right track of the etiology. The psychic epileptic state is, as is well known, mainly characterized by a disturbance of consciousness, and even though this disturbance does not, as a rule, produce a delirious condition, still it is not so very rare for the clinical picture of a delirium to appear during the life of an epileptic individual. The relation of a delirium to a true epileptic

seizure may be varied in particulars. The delirium may either follow or precede the attack. In some instances the epileptic convulsions may be wholly absent and the delirium may take their place as any other psychical equivalent of a genuine seizure. Delirium may sometimes be the only symptom in those cases which are known as larvated or psychic epilepsy. The diagnosis must be made in these instances by the periodicity of the seizures, by the sudden disturbance of consciousness, by aura-like appearances, by the stuporous condition following the attack, by hereditary taint, as well as by certain neurotic symptoms, showing themselves in the intervals, and by the well-known epileptic character of the individual.

The history of a recent acute infectious disease may lead to the correct judgment of an acute delirium. It is a well-known fact that the acute febrile diseases play an important factor in the etiology of mental disturbances. The clinical form of the psychoses belonging to this group is very variable, and, in fact, the changeability of the various psychopathic states is characteristic for these diseases. The patient may rapidly pass from a melancholic to a maniacal condition, and soon manifest all sorts of psychopathic symptoms. The mental disturbances in cases of this kind are frequently interrupted by lucid intervals, or by intermissions, lasting for several days. The beginning of the psychic disturbance in this group may not manifest itself for weeks or months after the termination of the infectious disease. Occasionally the psychosis may start with an acute delirium.

In general, the prognosis of this group of psychoses is favorable. Those cases, however, in which delirium constantly predominates usually represent a state of inanition, which occasionally may prove fatal, particularly in weak persons. This form, on the other hand, is not nearly so apt to run into a secondary chronic psychosis as another type, in which a regular set of delusions is developed soon after the outburst of the delirium, as, for instance, in acute hallucinatory paranoia.

Besides the acute infectious diseases, such as typhoid fever, malaria, influenza, pneumonia, pleurisy, multiple neuritis, articular rheumatism, erysipelas, scarlatina, etc., acute poisoning may produce psychoses which belong to the class under discussion. Delirium has been observed after large doses of salicylic acid, cannabis indica, belladonna, conium, ergot, etc. It has also been seen after the use of iodoform in and about wounds, and sometimes after a lengthy administration of ether or chloroform. The inhalation of poisonous gases, such as carbonic oxide, hydrogen sulphide, carbon disulphide, nitrogen monoxide, etc., may have as a sequel psychic disturbances of the kind described.

Psychopathic phenomena beginning with delirium may develop from chronic toxic conditions produced by the abuse of chloral and chloroform, or by the habitual use of opium, morphine, cocaine, absinthe, and, above all, alcohol. The predominance of delirium over all other psychic symptoms is responsible for the origin of the well-known term *delirium tremens*, so frequently seen in chronic alcoholism. Here, too, delirium is seen only in connection with other existing conditions. The delirium itself may sometimes be only of minor importance and, indeed, may be totally absent, being then replaced by such phenomena as hallucinatory confusion or maniacal exaltation. An acute delirium in the course of chronic poisoning may arise without direct cause, it may immediately follow excesses, or it may appear as a sequel of abstinence.

Chronic forms of metallic poisoning are sometimes characterized by similar psychic derangements. The principal features of saturnine encephalopathy are of an acute delirious nature. Cases of so-called "transitory lead mania" have been observed frequently. Mercurial psychoses, too, are mainly characterized by delirious conditions. Further, it is proper to place in this category those cases of lyssa the psychical disturbances of which, as a rule, take the form of an acute delirium. A comparatively large percentage of acute delirium is formed by the puerperal psychoses. Acute delirium, following even minor operations, has been frequently described. Traumatic injuries, especially those affecting the skull, may also evoke delirium.

Those cases which have been described under the name of *acute general paresis* begin, as a rule, with an acute delirium, and end fatally within a few days or weeks. These instances form a large proportion of the cases described as *delirium acutum*. If one examines carefully the previous history of such patients, it will almost always appear that, although the patient was in seeming good health, a series of derangements will be found which are characteristic of *general paresis*. The relatives will state that the patient had shown changes in character for some time past, that he had become indifferent to things which had previously interested him, that he frequently forgot important things, that his dealings appeared to be queer and without motive, etc. The autopsy in such cases shows pathological changes of some duration, whose influence had remained latent, such as a chronic interstitial encephalitis, atrophy and softening in isolated cortical areas, thickening of the dura, etc.

Despite the most careful research, it may prove impossible, in a certain number of cases of acute delirium, to draw any conclusions regarding the pathogenesis from the history of the patient. In this event, we may have to deal with a sudden attack in a previously perfectly healthy individual. Here a thorough examination of the internal organs may give some clew as to the origin of the disease.

I have already alluded to a twofold relation between delirium and the acute infectious diseases. I have mentioned the delirium arising during a febrile disease and that accompanying a psychosis which is the result of such an ailment. I shall now consider a third group, which shows a relation between the two conditions. A person, previously in perfect health, may suddenly be thrown into a state of agitation and confusion. An acute psychosis is diagnosticated, and the patient may be transferred to an institution. After a while it appears that he is suffering from typhoid fever or some other infectious disease. Acute delirium of this nature without any prodromes occurs especially in erysipelas and acute articular rheumatism, in typhoid fever, and in meningitis. But even minor infectious diseases, such, for example, as a follicular tonsillitis, have given rise to similar phenomena.

The diagnostic and therapeutic importance of a careful physical examination in these cases is self-evident. A number of cases, no doubt, starting with elevated temperature, and diagnosticated as so-called *delirium acutum*, may belong in this category. The diagnosis of meningitis usually offers no great difficulty on account of the typical symptoms on the part of the cranial nerves. In typhoid fever, the enlarged spleen, the quality of the dejections, and the presence of a roseola will lead to the correct diagnosis, although all these symptoms may sometimes remain absent for a considerable time. The correct somatic diagnosis of acute rheumatism is, as a rule, not difficult.

The examination of the urine in a case of delirium of acute origin must never be neglected. Although it is a well-known fact that in the course of psychoses, and specially during delirium, albumin and hyaline casts may be found in the urine with no post-mortem changes in the kidneys, yet it is not rare for an acute delirium to start a psychosis which is dependent upon a chronic, possibly latent nephritis. Occasionally the cerebral features of a uræmia may present the clinical picture of acute delirium, although, as a rule, they are characterized by epileptiform seizures or comatose conditions.

The cerebral symptoms of some other forms of disturbed metabolic processes may assume the form of delirium. Now and then an acute delirium may take the place of the familiar diabetic coma, and it may, in fact, be the first symptom of an overlooked diabetes.

So then I may conclude by saying that *delirium* constitutes an independent psychopathic condition, which, however, does not form a *morbus sui generis*, but occurs during the course of a great many mental diseases. A