found to have on them, at regular intervals, alternate swellings and constrictions. The swellings, or nodes, are about 1 mm. long, fusiform in shape, and darker in color than the constricted parts. The latter are about one-third the length and diameter of the nodes. The constricted parts contain neither medulla nor pigment and may consist of the cuticular layer of the hair alone. There is atrophy of the hair bulbs. The nodes are all along the hair from the root to the point. Fracture invariably takes place through the internodular portion, and frayed-out ends may be found. Sabouraud found that the constrictions formed at two days' interval. Gilchrist believes that the disease originates in the hair follicle very near to the papilla. He was able to trace the fusiform swelling to the lower fourth of the hair shaft, where there were constrictions in the walls of the hair follicle. He found no change in the hair papillæ.

Etiology.—In many cases the disease is hereditary. Several observers have reported instances of a number of cases in the same family. At times it shows a tendency to descend in the same sex, a peculiarity shown in other dermatoses. It is probably a trophoneurosis.

Treatment has thus far been without effect. Stimulation might be tried, but the prognosis is bad.

Besides these well-defined forms of atrophy of the hair we have reports of allied diseases from single observers. Thus Crocker describes a case of *end atrophy* of the hair in which the distal ends of the hairs are bulbous and of lighter shade than the rest of the hair.

Phagmesis is another abnormality of the hair in which feathers instead of hair are met with. This is of doubtful character and comes down to us from 1831, at which time a boy showing this anomaly is said to have been on exhibition in Bremen.

Two peculiar changes in the texture of the hair are reported by Ferber (Virchow's *Archiv*, 1866, xxxvi., 598). Both patients were of nervous temperament, and their hair in a few hours would change from being soft and curly to become straight and bristly. After a time the hair would assume its natural condition.

Noduli Laqueati is an anomaly of the hair in which it seems to tie itself into knots. The loops of the knots catch dust. The hair looks as if infested with nits, but under the microscope it is seen that it is the knots that give this appearance. *George Thomas Jackson.*

ATROPHY.—(Wasting, lack of nourishment, wasting of the body due to defective nutrition.) The word is at the present time used in pathology to signify the decrease in size of an organ or tissue which results either from a decrease in the size of its individual elements, or from a diminution in the number of these elements, or from a combination of these two factors. We may speak, therefore, of a quantitative and of a numerical atrophy, but no practical distinction can be made between these two forms, as a diminution in the number of the tissue elements almost always accompanies a decrease in size.

In its narrowest sense the term is used to indicate a decrease in size of the individual histological elements due to a simple loss of substance without including the idea of other retrograde changes. The essential idea of atrophy is, therefore, separated from that of degeneration, and the term should be applied only to those conditions in which the primary pathological change is a loss of bulk.

On the other hand, the meaning of atrophy must not be confused with the ideas expressed by the words agenesia, hypoplasia, and aplasia. These terms, unfortunately, have been used rather loosely as synonyms with atrophy, but the best usage is to assign to each one a distinct technical meaning: agenesia, total failure of development or destruction of a part after it has begun to develop; aplasia, marked defective development of an organ; hypoplasia, under-development; atrophy, decrease in size after development has been reached. From this it will be seen that the causes leading to these changes operate at different periods of life; thus agenesia and aplasia are the results of disturbances in early fetal life; hypoplasia the result of changes occurring later, but at any period before complete development is reached; while atrophy may occur at any time in the history of the organism when any tissue or structure has reached its full anatomical and physiological maturity. The latter process is, therefore, a retrograde change occurring in parts that were originally normal and perfectly formed.

All stuntings and defective development of the body and its parts, either intra- or extra-uterine, are to be considered under the head of aplasia or hypoplasia, and not under that of atrophy. But the cells of an aplastic or hypoplastic organ may also undergo a decrease in size due to the same causes that lead to atrophy of perfectly formed organs. A hypoplastic organ may become atrophic; hence in its broadest sense the meaning of atrophy must be extended to include the retrograde decrease in size of imperfectly developed cells and organs. The fundamental idea of atrophy is, therefore, *a retrograde decrease in size of either perfect or imperfect cells.*

The decrease in size and the disappearance of the tissue elements in atrophy must be referred to a failure of the processes of restoration to keep equal step with the never-ceasing processes of tissue waste and destruction. All cells possess a histogenetic energy which is manifested in the functions of nutrition, assimilation, and reproduction. For all cells, for every organ, for every individual, and for every species there is a certain limit to the ultimate amount of inherent histogenetic energy. This limit is fixed by intrinsic forces acquired through the agency of external forces in the process of evolution of the species, and represents the physiological duration of life of each organ, and of each individual of that species. Could all external injurious influences be avoided the organism would after a certain period of time come to a physiological death through physiological atrophy, or, in other words, as soon as such a limit of histogenetic energy is reached that the necessary vital functions of the body fail to be properly performed in a degree sufficient to preserve life. We may, therefore, speak with propriety of a physiological or histogenetic atrophy.

Further, the histogenetic energy of the cells is to a certain extent maintained by means of certain stimuli, and as a result of the removal of these stimuli an inaction atrophy may result. Atrophy must result also from any deficiency or disturbance in the supply of nutrition, and likewise excessive consumption or waste of tissue must lead to atrophy. Further, atrophic changes may be set up by mechanical hindrances to growth, as in pressure, constriction, etc. On the other hand, the loss of normal pressure or tension may produce atrophic changes in the tissues so affected, and the separation of an organ or tissue from its nerve centres is usually followed by a similar condition. We may consequently classify the various forms of atrophy as histogenetic, inaction, lack of nutrition, neuropathic and pressure atrophy. Only the first of these, the histogenetic form, is essentially an active process: the cells are unable to assimilate the food brought to them. The other forms are passive in character: either insufficient food is brought to the cells, or harmful substances are formed which injure their nutritive function.

Histogenetic or Physiological Atrophy.—This form of atrophy is the result of a decrease in the histogenetic energy of the cells. As stated above, the potential energy of each cell and organ is limited in direct relation to the part which its function plays in the general economy of the organism. Hence the duration of life varies with different organs and tissues, and in the life history of the organism from the very beginning the processes of atrophy go hand in hand with those of development. In the earliest stages of growth up to the time of puberty there is a preponderance of cell growth over cell decay; in adult life there is a period of equilibrium; but with the beginning of old age the loss of histogenetic energy is shown in the fact that cell decay preponderates over cell restoration.

Even in earliest fetal life certain structures fulfil their

function and pass out of existence. In the formation of the fetal placenta portions of the membranes disappear at a very early stage, and in the development of the chorion there is from the beginning a progressive atrophy of the villi. The placenta at term must be regarded as a senile organ. Portions of the fœtus itself, as the Wolffian bodies, the Wolffian ducts, the ducts of Müller, the umbilical vesicle, the omphalo-mesenteric duct, etc., disappear very early, even in the period of most vigorous development of the fœtus. Numerous blood-vessels undergo obliteration even before birth, and very soon after this event the closure of the ductus Botalli and the umbilical vessels takes place. Likewise the casting off of the umbilical cord must be considered under this head; and later the shedding of the milk teeth is another example of the disappearance of structures that have fulfilled their aim and reached their limit of existence. The disappearance of the thymus after the fifteenth year is one of the most striking of the histogenetic atrophies. During the period of most active development it is one of the largest lymph glands of the body, but soon after puberty it disappears, becoming entirely replaced by fat tissue. During middle life single portions of tissues, as certain portions of the petrous and sphenoid bones, vanish. Hyaline cartilage may also be regarded as an essentially temporary structure, as in many individuals it entirely disappears from the body during middle life and is replaced by bone.

Some organs do not atrophy, but very early suffer a cessation of growth, so that they become no larger in the adult than in the new-born (adrenals, male mammæ, etc.). The failure of these organs to increase in size is not due primarily to any failure of nutrition or disturbance of circulation, and can be explained only by the assumption that the original histogenetic potentiality of growth has reached its limit. The atrophy of the ovaries and of the uterus after the forty-fifth year, before all of the ova are discharged, must be similarly explained. In the case of the ovary the primary change is in the blood-vessels of the organ, which normally show sclerotic changes before the blood-vessels of any other part of the body. The menopause is essentially a process dependent upon changes in the ovarian vessels, and to these changes the atrophy of the Graafian follicles must be considered secondary. The atrophy of the ovary at the menopause may, therefore, be explained by the assumption that the histogenetic energy of the ovarian vessels has an earlier limit than that of the systemic vessels.

In the atrophy of old age (senile atrophy) the blood-vessels, lymphadenoid tissues, muscles, and bones suffer most. The changes in facies, posture, and gait of the old individual are dependent upon these conditions. The brain may also undergo a marked atrophy, and of the internal organs the kidneys, liver, and lungs may suffer to a greater or less extent. On the other hand, there are certain organs and tissues which undergo but little senile change: the formation of the red blood cells continues in old age without decreased energy, and defects of epithelium, blood-vessels, and connective tissue are as completely repaired as in earlier life. There are very marked individual differences as to the organ which shows the greatest degree of senile change: environment, disease, etc., may lessen the inherent histogenetic energy of certain organs so that they may become prematurely senile, or atrophy to a greater degree than others. Further, there are individual differences dependent upon the amount of histogenetic energy inherited; the variation in this is a well-known pathological fact. Nevertheless, in spite of these individual differences typical senile atrophy is always confined to certain organs and tissues.

Senile atrophy is not only active but is also passive, as it depends not only upon the gradual decrease of energy on the part of the cells, but is in part the result of the narrowing and obliteration of the blood-vessels supplying nutrition to the cells. The changes in the blood-vessels are, therefore, to be regarded as the most important of the senile processes, and it is probable that the chief part of the glandular atrophies is secondary to these.

Atrophy of Disuse.—The histogenetic energy of many organs and tissues is dependent upon certain regular stimuli. If these stimuli are removed for any length of time an atrophy results which we may call an inaction or disuse atrophy. The atrophy of the optic nerve after destruction of the eyeball; the atrophy of peripheral nerves and portions of the spinal cord following amputations of the limbs, are familiar examples of this form. Likewise, if through any influence glands or muscles remain inactive for a certain period of time atrophic changes occur. In the case of muscle the loss of substance is usually not very great. The testicles are said to become atrophic in cases in which sexual abstinence is extended over a long period of time, and even the bones undergo a loss of substance when kept in undisturbed rest for several years. Further, the physiological atrophy of the uterus and mammæ after pregnancy may be included in this class.

In the case of nerves, glands, and muscle, inaction atrophy is essentially active, but as the result of the cessation of function there is also a decrease in the nutritive activity of these structures and a lessened blood supply which leads to further disturbances of nutrition. In other tissues the chief cause of the atrophy is a decrease in the nutrition of the unused part, but with this there is also a lessened power of assimilation. The involution of the puerperal uterus, by means of which the organ is restored to very nearly its original size, is a very complicated process, partly atrophy and partly degeneration, involving all of its structures, endometrium, muscle, and blood-vessels. It is essentially active in its nature, but the obliteration of its enlarged blood-vessels through sclerotic changes and the organization of thrombi plays a very important part in the retrogression.

If the conditions leading to the inactivity of certain parts are in operation during the period of development and the tissues in consequence of disturbed nutrition fail to reach their normal size, the process is to be regarded in the light of a hypoplasia rather than of atrophy. It is, however, impossible in all cases to separate these processes, since in hypoplastic organs there may be an atrophy or disappearance of tissues which had undergone a certain degree of development.

Atrophy Dependent upon Impaired Nutrition.—If there is a deficiency in the amount of nutritive material brought to the cells these will undergo atrophic changes. The degree and rapidity of the atrophy are in direct relation to the degree of metabolic change of which the affected organ or tissue is capable. Hence adipose tissue quickly disappears if fat or fat-forming substances are not adequately supplied to it. The bones become softened and fragile if the supply of lime salts is withheld for a period of time, and it is also probable that a decrease in the amount of hæmoglobin contained in the red blood cells is the result of deficient absorption of iron. The nature of rachitis, osteomalacia, and the various forms of anæmia is as yet but little understood, and these diseases may be the result of more complicated disturbances than the mere withdrawal of certain food elements.

Local atrophies may result from disturbances in the blood supply of certain regions following arterio-sclerosis, thrombosis, or inflammatory processes involving the vessels. Degenerative changes almost invariably accompany this form of atrophy. If the blood supply of any part is completely shut off, necrosis results. When there is an insufficient supply of food to the body as a whole, or if the fluids of the body are not adequately restored, a general atrophy of the body takes place. The fat, muscles, blood, and abdominal organs suffer to the greatest degree. The fat disappears first and may be reduced to seven per cent. of its original amount. The muscles may lose as much as fifty per cent. in weight. Of the abdominal organs the liver, spleen, and intestines suffer most. The brain, spinal cord, bones, and heart muscle undergo but little loss of substance even in cases of death from starvation. Lipomata remain unchanged

in spite of the almost complete loss of the normal fat tissue of the body. This remarkable phenomenon has not as yet been explained.

The histogenetic energy of the cells is most intimately connected with their physical and chemical integrity, and disturbances of this lead to a decrease of this energy. The presence of certain foreign elements in the circulation leads occasionally to atrophy associated with degenerative processes. The protracted use of iodine may cause atrophy of the thyroid and mammary glands, and in chronic lead poisoning the extensor muscles of the forearm may become atrophic.

Pressure Atrophy.—Closely allied to the atrophy resulting from insufficient nutrition is that produced by continued slight pressure. As the result of artificial mechanical pressure may be mentioned the examples of "corset liver" and "corset spleen" the constriction produced by rings, belts, and garters, the Chinese foot, the flat head and flat nose of certain Indian tribes, etc. Skin, muscles, and bone may disappear as the result of pressure from aneurisms and tumors. Varicose veins may likewise lead to the atrophy of the neighboring structures. In scoliosis, genu valgum, and pes valgus, atrophy of certain portions of the joints may be caused by the abnormal pressure produced by an oblique position of the bones. After loss of the teeth the alveolar processes of both jaws may disappear as the result of the pressure brought to bear upon them in mastication. The skull cap may present erosions which have been produced by the pressure of atheromata of the scalp or by hypertrophic Pacchionian bodies. In hydrocephalus and hydronephrosis the brain and kidneys respectively may undergo extreme atrophy. Further, atrophic changes may be caused in any part of the body as the result of pressure from inflammatory exudates.

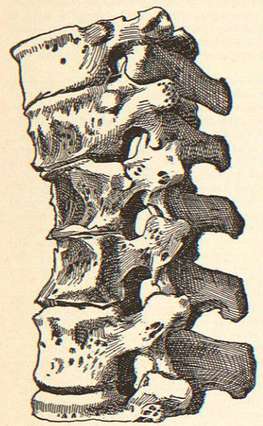

FIG. 380.—Pressure Atrophy of the Spinal Column, Caused by the Encroachment of an Aneurism of the Aorta. (After Ziegler.)

The various forms of pressure atrophy are caused, as a rule, by slight pressure continued through long periods of time. It is the result of direct injury to the tissue and of circulatory disturbances. It is therefore passive in its nature. If the pressure is so great that the blood-vessels become closed, degeneration and necrosis must follow. In many cases the causes of the atrophy resulting from pressure are complicated, mechanical force and disturbances of nutrition playing the chief parts in its production.

Neuropathic Atrophy.—The question of the existence of trophic nerves and trophic centres and their relation to the individual tissues is still unsettled, and the existence of a neuropathic or trophoneurotic atrophy must at the present time be viewed more in the light of a possibility than as being an established fact. It is not to be doubted that as the result of disturbances of innervation both atrophic and degenerative changes arise, but it is probable that these changes are not entirely dependent upon the loss of nerve influence, but for the greater part are secondary to the loss of functional activity and to disturbed nutrition caused by vaso-motor changes in the regions supplied by the affected nerves. As a result of these changes inflammations are easily set up, even by slight causes which ordinarily produce no lesions, and the result of the inflammatory process may be either atrophic or degenerative in its nature. The majority of the changes following disturbances of innervation are not of the nature of a true atrophy, but are degenerative in character. The application of the term atrophy to these processes is justifiable only by the fact that the ultimate condition is a loss of tissue.

Disease of the anterior horns of the spinal cord or of the motor roots is followed by atrophy of the corresponding nerves and muscles. Anterior poliomyelitis, progressive muscular atrophy, and bulbar paralysis are well-known examples of this form of atrophy. In syringomyelia and tabes dorsalis atrophy of the bones and joints not infrequently occurs. Injuries of the peripheral nerves may be followed by thinning of the skin, exfoliation, loss of hair, and atrophy of the glands of the skin. Disease or injury of the nerve trunks of one side of the face may lead to atrophy of the tissues of that side. Unilateral affections of the brain in fetal life or during early childhood may cause an atrophy of the opposite side of the body (congenital or infantile hemiatrophia). General atrophy of the body occurs in progressive paralysis and to a less extent in melancholia and dementia. It is denied by many authors that these forms of atrophy are neuropathic, and it is much more probable that vaso-motor disturbances, loss of function, and general as well as local disturbances of nutrition are the chief factors in their production.

Atrophy Due to Excessive Waste.—In all conditions in which the repair of the tissue is exceeded by tissue consumption a loss of substance must occur. In fetal life and in the period of early development only a portion of the substances taken into the body is compietely oxidized, the greater part is used in the building up of tissue. In adult life the larger part is burned up, the smaller remaining portion is used in tissue repair. The two processes of waste and repair stand in different relations to each other at different periods of life, and must be considered as independent processes. This is very clearly shown in those pathological conditions in which tissue waste exceeds tissue repair. Over-use of any organ leads to fatigue, poisonous products of metabolism are retained, and the cells are not given sufficient time for rebuilding. If a condition of chronic fatigue develops as a result of chronic over-use anatomical changes occur. Chief of these is a loss of substance. The brain is the most important organ which may be so affected. Of the glandular organs the testicles most frequently suffer. Atrophy of the heart and voluntary muscles may also occur as the result of over-use. The failure of compensation in chronic valvular disease is also to be placed in this category.

Repeated severe hemorrhages, chronic suppurative processes, long-continued excretion of albumin, diabetes, fever, rapidly growing tumors, and many other similar processes produce general wasting of the body with marked atrophy of certain tissues. In general the nature and course of the various cachectic atrophies are very closely allied to senile atrophy, and the microscopical appearances are identical.

Atrophy Caused by Decreased Tension.—Through a permanent loss or decrease in the normal tension, as in the case of muscles, tendons, fascie, and blood-vessels after amputations, or of tendons after tenotomy, atrophy may take place. The involution of the puerperal uterus may also be considered under this head.

A large number of conditions, such as the decrease in size of the orbital cavity after removal of the eyeball and of the sockets of joints after unreduced dislocations, are not properly included in this class, as the decrease in size of the cavity is not of the nature of an atrophy, but

is usually caused by an increase of tissue in and about the cavity.

MACROSCOPICAL APPEARANCES.—The most striking change in atrophic organs is their decrease in size. This is directly dependent upon the decrease in size and the diminution in number of the structural elements. The

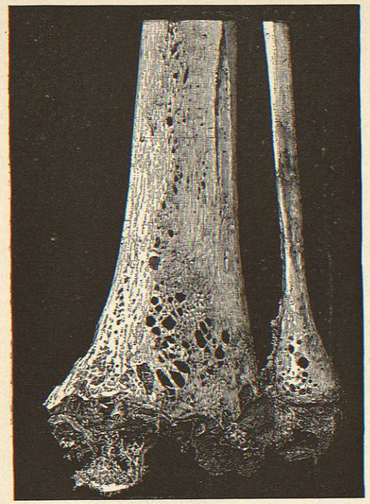

FIG. 381.—Excentric Atrophy of the Lower Ends of the Tibia and Fibula, with Osteoporosis. Natural size. (After Ziegler.)

muscles, kidneys, liver, and spleen show this decrease in size to the most marked degree. In extensive atrophy of the muscles as seen in progressive muscular atrophy the disappearance of the tissues may be so marked that the impression is given that there is nothing between the skin and the bone (so-called living skeleton). In many cases, however, there is a deposit of fat in the atrophic tissues which may be so extensive that the normal size may be preserved or an actual increase may take place (atrophia musculorum lipomatosa). The atrophic liver and heart may likewise be increased in size through fatty infiltration.

The size of atrophic lungs may be increased through the stretching of the atrophic alveolar walls and the consequent dilatation of the air spaces (atrophic emphysema).

In the case of atrophy of fully developed bones no decrease in size takes place, but the Haversian canals and medullary cavity become enlarged (excentric atrophy or osteoporosis). The spleen as a rule suffers a symmetrical decrease in volume, while the liver usually shows more marked loss of substance in the left lobe than in the right.

If the atrophy of an organ proceeds symmetrically in all parts a uniform decrease in size results with preservation of normal form. It, however, progresses most frequently with greater rapidity in one part than in another, and as the result of this unequal atrophy nodules and furrows are formed so that the organ comes to present a nodular or granular surface. This is especially marked in atrophy of the glandular structures, liver, kidney, etc. As a rule atrophic organs contain less blood and are drier than normal ones. The increase of connective tissue, either relative or actual, causes an increase in consistency with loss of elasticity. As a result of the decreased blood content the natural color of the organ stands out more distinctly; hence atrophic muscle, especially heart muscle, is much browner in color than normal. An increase of the normal pigment or an increased deposit of hæmatogenous pigment is also a very frequent accompaniment of atrophy (brown atrophy of heart and liver). In other cases the color of the atrophic organ is lighter or more grayish than normal because of the relative or actual increase of connective tissue. In all cases in which much fat is deposited the color becomes yellowish.

MICROSCOPICAL CHANGES.—The microscopical examination of atrophic organs shows a decrease in size and a diminution in number of the normal elements. This may occur without other changes, or the atrophy may be accompanied by a deposit of fat or pigment, or an increase in the amount of the normal pigment, or it may occur in association with various degenerative processes. We may therefore distinguish: *simple atrophy, fatty atrophy, pigment atrophy*, and *degenerative atrophy*. Fatty and pigment atrophy are so closely related to simple atrophy that they are to be considered as simple atrophy followed by or associated with fatty infiltration and pigment formation. They should be carefully distinguished from the true degenerative atrophies in which changes in the nature of the protoplasm occur from the very beginning. As a result of these changes new substances are formed in the cells or about them (mucous, fatty, hydropic degenerations; amyloid, etc., deposits). In these conditions, especially in the case of the pathological deposits and infiltrations, the atrophy of the cells must be regarded in many cases as a secondary process.

As a rule the more highly specialized portions of the tissues suffer to a greater extent than the connective-tissue framework. This may be unchanged or, as most frequently occurs, increased in amount. In the newly formed connective tissue there is usually a greater or less degree of fatty infiltration. Through the increase of connective tissue and the fat deposit the normal pressure upon surrounding structures may be preserved unchanged. The fatty infiltration may, therefore, be regarded as being of the nature of a compensatory process.

In atrophy of striped muscle the contractile substance disappears while the nuclei of the endomysium proliferate to a greater or less extent. In atrophy of the lung the alveolar walls become greatly thinned, the capillaries disappear, and the air spaces become increased in size or

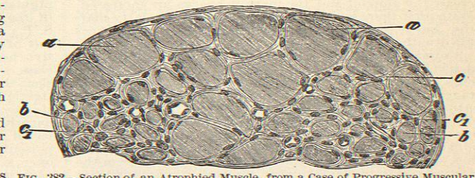

FIG. 382.—Section of an Atrophied Muscle, from a Case of Progressive Muscular Atrophy. (Müller's fluid; Bismarck brown.) *a*, *a*, Normal muscular fibres; *b*, atrophic muscular fibres; *c*, perimysium internum, the nuclei of which, at c_1, seem to be increased in number. Magnified 200 diameters. (After Ziegler.)

confluent through the disappearance of the wall. As a result of the obliteration of many of the smaller capillaries the larger vessels show a state of chronic congestion. The liver lobules become very much smaller in atrophy of that organ, the connective tissue of Glisson's capsule is relatively or actually increased, and is more hyaline in character, resembling scar tissue. The liver rods and cells are decreased in size, and there is a great variation

in the size of the individual liver nuclei, many of which show a compensatory hypertrophy. The central veins and capillaries are congested, and the liver cells of the central zone of the lobule contain much hæmatoidin, while those of the peripheral zone show an increased amount of fat.

In the atrophic kidney there is a decrease in the size of the tubules due to a decrease in size and to a diminution in number of the epithelial cells. Many tubules may be found containing few cells or completely collapsed. As a result of the loss of intervening tissue the glomeruli are brought closer together, so that from twenty to forty may be found in one low-power field. The epithelium and capillaries of the glomeruli also disappear, and as a result numerous obliterated glomeruli are found. In atrophy of the central nervous system the ganglion cells disappear or become smaller, while the neuroglia remains in normal amount or becomes increased. Atrophy of the lymph glands and spleen is shown by a disappearance of the follicles and a diminution in the number of the lymphadenoid cells. The trabeculæ are brought more closely together, and the finer stroma is increased in amount. In atrophy of bone the bone substance is decreased in amount and the marrow spaces are increased. With this there is usually an increase in the fatty marrow, but it occasionally disappears, leaving cystic spaces filled with fluid.

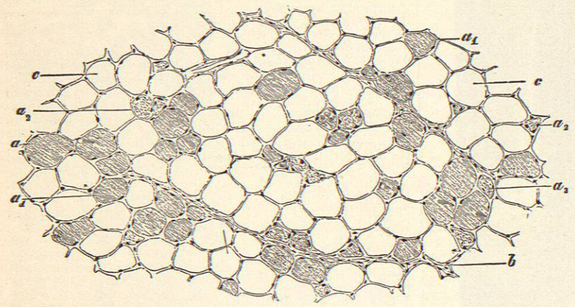

FIG. 383.—Lipomatosis of the Muscles of the Calf of the Leg, Together with Atrophy. (Müller's fluid, carmine.) Transverse sections of a normal (*a*) and an atrophied (a_2) muscular fibre; a_2, transverse section of a tubular sarcolemma containing contractile substance in a condition of disintegration; *b*, bands of connective tissue; *c*, fat tissue. Magnified 60 diameters. (After Ziegler.)

COURSE.—The course of the various forms of atrophy depends wholly upon their nature. Total atrophy occurs as the result of the exhaustion of the inherent histogenetic energy, as in the case of many of the fetal structures, the thymus, etc. In partial atrophies due to other causes, such as disturbances of nutrition, pressure, etc., a greater or less degree of restoration is possible in all structures in which the histogenetic limit has not been reached. If the causes leading to atrophy operate in the early periods of development, agenesia or aplasia may result. Certain organs, as the thyroid or sexual glands, may be thus affected and their lack of development may lead to retarded growth of other tissues. As stated above, these processes are not of the nature of true atrophies, but it is difficult in all cases to make sharp distinction. The atrophy of certain fully developed organs likewise may affect the growth of other organs or even of the whole body, as in progressive muscular atrophy where atrophic changes in the bones follow those in the muscles.

In so far as the function of the organ is concerned, the atrophy of its elements is of the greatest importance. Atrophic muscles lose their contractile power, atrophic glands their secretory function, osteoporotic bones are easily broken, and atrophic skin is easily injured by a very slight trauma. Further, the atrophy of one organ or set of tissues disturbs the function of other organs and leads to a general diseased condition of the organism.

The prognosis in atrophy is favorable only in those pathological conditions in which the cause of the atrophy may be removed, and in tissues in which the physiological limit of growth has not been reached. Atrophy of the vital organs, heart, medulla oblongata, kidneys, respiratory muscles, etc., not infrequently leads to death. There may also result a complete disappearance of certain structures caused by the atrophy of the matrices which form them. In atrophy of the periosteum the bone disappears, in atrophy of the skin there is a loss of the hair and nails, and in atrophy of the lymph glands there is a decrease in the formation of leucocytes.

It is evident that only the purely passive forms of atrophy admit of treatment. The removal of the cause and the restoration of the normal nutrition are the chief indications.

Aldred Scott Warthin.

ATROPIC POISONS.—The natural order *Solanaceæ* comprises many plants possessing actively poisonous properties. Among these the more important are: *Atropa belladonna*, deadly nightshade; *Hyoscyamus niger*, henbane; *Datura stramonium*, thornapple or Jamestown weed; *Mandragora officinale*, mandrake; *Scopolia Japonica*, Japanese belladonna; and several species of *Duboisia*. It is worthy of note that several of the most widely used foods—*e.g.*, potato, tomato, and egg plant—are derived from plants of this order.

The poisonous qualities of the *Solanaceæ* depend on well-marked alkaloids, analogous in composition and properties, notably as regards the power to produce dilatation of the pupil, for which reason the term *mydriatic alkaloids* has been applied to them collectively. Since the toxicology will include the effects of both the plants and the active principles, it will be more satisfactory to consider them under the title of the "Atropic Poisons." The deadly nightshade, *Atropa belladonna*, is the most important of the group, and its alkaloid, atropin (atropina, U. S. P.), has been extensively used by ophthalmologists, but of late years it has been often replaced by a derivative, homatropin, and by another natural alkaloid of the class, scopolamin; the effects of these being more transient than those of atropin. According to recent investigations, the so-called daturin—derived from *Datura stramonium*—is identical with atropin, but commercial daturin often contains some hyoscyamin. Statistics collected by Witthaus show that of the reported cases of poisoning by the plants of this class or their alkaloids, over sixty per cent. are by belladonna or atropin, and next in order by *Datura stramonium*. The majority of cases were accidental.

The *symptoms* produced by toxic doses of the preparations of belladonna usually manifest themselves within an hour, and are marked and characteristic. They are heat and dryness of the mouth and throat, increasing to a feeling of burning or constriction, difficulty of swallowing, giddiness, nausea and vomiting frequently, but not invariably; great mental excitement, delirium, and hallucination, often decidedly maniacal or hysterical. The circulation is decidedly affected, the pulse being quickened, the face becoming red and turgid, and in some instances a scarlet eruption has appeared over the body.

Dilatation of the pupil with insensibility to light and impairment of vision is a usual and important symptom. The mental symptoms often take the form of wild and uncontrollable laughter. The following case, detailed by Taylor ("Treatise on Poisons"), illustrates the clinical history of belladonna poisoning. A boy, aged fourteen, ate about thirty belladonna berries. In about three hours it appeared to him as if his face was swollen, his throat became hot and dry, vision impaired, objects appeared double, and seemed to revolve and run backward. His hands and face were flushed and his eyelids swollen, and occasional flashes of light were experienced. He tried to eat, but could not swallow on account of the state of his throat. In endeavoring to walk home he staggered, and felt giddy whenever he attempted to raise his head. He was incoherent, frequently counted his money, and did not know the silver from the copper coin. His eyes had a fixed and brilliant look, he could neither hear nor speak plainly, and was very thirsty; he caught at imaginary objects in the air. There was headache, but no vomiting nor purging. These symptoms were much the same nine hours after the taking of the poison. The pupils were so strongly dilated that there was merely a narrow ring of iris; the eyes were quite insensible to light, the eyelids did not close when the hand was passed suddenly over them, but the nerves of common sensation were unaffected. The pulse was 90, feeble, and compressible. He continued in this state for two days, but gradually recovered.

Fatal cases usually terminate in coma, less frequently in convulsions.

The *treatment* of belladonna (or atropin) poisoning is both direct and physiological. Free evacuation of the stomach by means of emetics or the stomach pump should be resorted to; as antidotes, animal charcoal or tannic acid may be given. The former has been found efficacious by actual experiment on the human subject: it acts by absorbing the alkaloid; tannic acid renders it insoluble. Both these agents are, however, only temporary; they do not set aside the necessity for evacuating the stomach or using other remedies. Since physostigmin—the active principle of the Calabar bean—and also morphin cause contraction of the pupil, they have been naturally suggested as physiological antidotes to atropin, and clinical experience has borne out this view. The hypodermic use of these agents should therefore be cautiously employed, the condition of the pupil and the general nervous symptoms being used as guides to the medication.

Many of the recorded cases of belladonna poisoning have recovered under treatment. When a fatal result takes place, no special or characteristic post-mortem appearances are discoverable.

There are no striking or easily applied chemical tests for atropin. It may be identified by its physiological action, dilatation of the pupil.

Henry Leffmann.

ATROPINE. See *Belladonna*.

AUDITION.—Audition or hearing is the result of processes by which certain vibrations of physical media are taken up by a peripheral sense organ, the ear, and transformed into nerve stimuli which excite in consciousness sensations of a peculiar kind. These sensations we know as *sound*. They are a function of a certain part of the cerebral cortex. Unfortunately, it is also the rule to designate by the name of "sound" those physical vibrations which are the objective cause of sound sensation but which have nothing in common with it.

This article will be chiefly limited to the treatment of objective or physical sound and the manner in which it affects the mechanism of the ear in giving rise to impulses in the auditory nerves.

Physical sound, as contrasted with sound sensation, is due to vibrations of particles of solid, liquid, or gaseous media. In its vibration a particle may move back and forth in a straight line, as in the transmission of an air wave or in the propagation of a tremor longitudinally along a rod of metal; or it may describe a more or less elliptical or circular path as in a wave of water, and tranverse to the direction of wave movement. When a particle is forced from its position of equilibrium, the force with which it tends to resume that position is a measure of the elasticity of the substance of which it forms a part. Elasticity is the property by virtue of which vibrations are propagated.

Transverse and Longitudinal Vibrations. Waves of Sound.—The vibration of particles gives rise to waves having definite form, length, and rate of progress. The waves most familiar to us are those seen on the surface of water formed by an up-and-down motion of the particles in an elliptical curve. The vibration is *transverse* to the direction and length of the wave, and the amplitude or depth of the wave and that of the vibration correspond. The wave length is the distance measured from crest to crest or hollow to hollow; it has no necessary relation to the amplitude of vibration. A stretched string or a tuning fork also executes transverse vibrations, when plucked or rubbed in the usual way. But if a stretched string or a rigid rod be scratched at one end the particles there will be set in oscillation back and forth (longitudinal vibration), and this vibration will be transmitted as a wave along the string or rod and may be heard as sound at the further end, but will make no visible movement. It is important to consider how these longitudinal waves are formed. The particles which are struck or scratched at the end of the string or rod are forced against those immediately adjoining, these in turn crowd upon the particles in front; the energy of the first displacement measures the degree of crowding or condensation thus produced. The particles unnaturally strained together spring away from one another by virtue of their elasticity, and their energy of movement carries them beyond their position of rest, making the number of particles at the previous point of condensation less numerous than normal, thus producing a condition of *rarefaction* as a result of the oscillation. The forward vibration of the particles is thus transmitted as a phase of *condensation* to successive layers of the substance traversed, each condensation being succeeded by a complementary *rarefaction*. The two phases of condensation and rarefaction make up the wave of sound. The length of the wave is the distance measured between two points of extreme condensation or rarefaction. This wave length has no necessary connection with the length of the path described by the individual particles whose motion gives rise to the sound wave.

Aërial vibrations resemble the longitudinal vibrations of solids and liquids in that the sound waves move in the same direction as the particles whose movement produces the waves. The amplitude of movement of the individual particles is very much less than the wave length produced by that movement. In the mobile air the path described by the particle is relatively great, but in a rod of wood or metal or under water, the excursion of each particle in its vibration is infinitestimal in its relation to the wave length or distance between successive condensations or rarefactions. The amplitude of movement of the vibrating particle is a measure of the energy which has been imparted to it, and it has extraordinary physiological significance, for upon it depends the *loudness* of sound. In exact language, the intensity of sound sensation is proportional to the square of the amplitude of the vibrations which cause it. Tyndall in his work on sound illustrates by a diagram the method by which a "sound pulse" is transmitted (see Fig. 384). The apparatus consists of a series of wooden balls separated from one another by spiral springs (the balls represent

FIG. 384.—Illustrating Passage of an Air Wave. The balls, E, C, B, etc., represent air particles and the springs the elastic force restoring them to position. (After Tyndall on "Sound.")