necrosis of cells, and in certain other cases we can detect various forms of degeneration in cells; but there must be various degrees of injury which we cannot detect by changes in the cells and tissue. In the exposed mesentery of the frog all the phenomena of inflammation may be seen in a tissue which so far as we can tell is normal. The same thing is true of the action of mild irritants in this and other tissues. It is certainly possible that there may be alterations in tissues which we cannot recognize. Cohnheim has explained all the phenomena of inflammation by an assumed alteration in the walls of the vessels brought about by the injury. The primary condition, he thinks, is due to this, the vessels become more porous, allowing exudation to take place, while the accumulation of leucocytes in the vessels is due to an increased adhesiveness of the wall. He attributes the emigration of leucocytes and the diapedesis of the red corpuscles to the alteration in the wall and the effect of pressure. This theory attributes to the walls of the vessels a preponderance of activity which we are not warranted in assuming. It is the tissues which determine the character of the circulation in a part; a gland does not secrete because it receives more blood, but it receives more blood because it is secreting. It is much more reasonable to assume that it is the change in the tissue brought about by the injurious agent which determines the vascular phenomena. We cannot define how the interaction is brought about, but there are so many examples of the regulation of vascular supply by the demands of the tissue that we must believe in the relation. The dilatation and finally permanent enlargement of a collateral artery after ligature of the main stem is an example; the compensating endarteritis producing a diminution in calibre of a vessel supplying a part which has become atrophied is another example. We can see the vascular phenomena take place under conditions in which injury to vessels can be excluded. In the tail of the tadpole there is a very small margin which is not reached by the vessels and it is possible to cut away a portion of this without touching the vessels, and still the adjacent vessels dilate, the current becomes slow, and an abundant emigration takes place from them. The vessels here have not been injured by the trauma. The same thing is true of central injuries to the cornea. If the centre of the cornea be touched with nitrate of silver a local necrosis of tissue is produced. A brown mass is formed by the precipitation of the silver, but the necrosis extends a short distance beyond the brown eschar. All of the peripheral vessels are affected. There are congestion and abundant fluid and cellular exudation from these, while there may be only slight congestion in the conjunctival vessels above them. It is unreasonable to suppose that these vessels are affected by the extension to them of the action of the silver. How the tissues influence the vessels we do not know. It may be by a purely local nervous mechanism, for the phenomena can be observed in a part in which all connection with the central nervous system has been destroyed. The accumulation of leucocytes in an inflamed part seems to be due not to any change in the vessels but to an attraction for leucocytes which is exerted by the injured tissue. We know that chemical substances have the power of attracting to them micro-organisms which possess independent motion, and leucocytes may also be attracted or repelled by certain substances. The name chemotaxis has been given to this attraction, positive when it attracts, negative when it repels. It is certain that necrotic tissues exert a strong positive chemotaxis for neutrophile leucocytes, and usually wherever an area of necrosis is found leucocytes have invaded it and are in the vicinity. It seems clear, therefore, that to this chemotaxis the presence of the leucocytes in the tissue is due. Substances of chemotactic power may be produced even in the slighter forms of injury in which there is no actual necrosis of tissue produced. Certain of the micro-organisms exert a strong chemotaxis for leucocytes. We are ignorant how these substances exert their action. In the cornea, for instance, are chemical substances produced in the necrotic centre and diffused in the tissue until they reach the vessels; or how do they influence the leucocytes? It is just as possible to explain the emigration of the leucocytes from the vessels in this way as it is to explain their migration in the tissues to the necrotic area. Another thing is very striking. If the cauterization is produced not in the middle but at one side of the cornea the vascular changes and emigration take place from the nearest vessels. No leucocytes will be found in that part of the cornea most remote from the injury. Although it is possible that a chemical substance would reach first and affect with the greatest intensity the vessels which are nearest, it should still exert some effect on the more remote. It can be considered certain that the process of emigration is an active one, the blood cells creeping through the walls by means of amœboid movements. The passage takes place chiefly at the small openings between the cells composing the walls, which in consequence of the dilatation of the vessels are wider than normal. That these openings are wider has been shown by the injection of normal and inflamed vessels with nitrate of silver which stains the intercellular substance. Examination of specimens which show clearly the relation of protoplasm and nucleus shows that the nucleus is in that part of the blood cell which first passes through, following the same law that prevails in migration of leucocytes in the tissues. The adherents of the exclusively vascular theory of inflammation attribute the slowing of the blood stream and the close packing of the cells in the vessels to the alteration of the walls. It is not necessary to assume this. Wharton Jones first pointed out that it could be explained by the concentration of the corpuscles due to the passage of fluid into the tissues. This must increase the friction of the corpuscles in the vessels and at the same time the pressure within the latter is reduced. It is astonishing, in the explanations which have been given of the inflammatory phenomena, how little attention has been paid to the tissues. It is generally assumed that tissue pressure remains the same in all conditions and that the dilatation of the vessels shows a higher pressure on the part of the blood. The process of exudation is active and not a passive filtration under increased pressure. How much it is due to activity on the part of the endothelial cells, analogous to secretory activity, is uncertain. We do not know what changes are produced in the tissue rendering them more capable of taking up fluid from the blood nor how these changes act. We know nothing of the mutual relations of blood and tissue pressure in an inflamed part. It is certain, however, that the tissue pressure is increased; the rapid outflow of lymph from an inflamed part—the greatly increased tension—shows this. It also has been generally assumed that diapedesis is purely mechanical and is due to the red corpuscles being forced through the enlarged spaces in the walls by the increased pressure in the vessels. There can be no question that the process is a mechanical one, but it is much more reasonable to assume that the red corpuscles are carried through the small openings in the vessels by the fluid stream passing into the tissues.

Any injury to the tissues is accompanied by changes in the tissue cells which, taken *in toto*, must be regarded as reparative. In some cases these tissue changes are simple, in others they are extremely complicated. Slight injuries involving only partial loss of surface epithelium are easily repaired by proliferation of the cells adjoining the injured part. This is accompanied by congestion of the vessels and probably by a slightly increased transudation. In more extensive injury involving complicated structures, in which not only cells but intercellular substance must be formed, the changes are more complicated. This may be more easily studied in the cornea than elsewhere. If necrosis is produced in the centre of the cornea by the application of nitrate of silver, changes begin to appear in the corneal corpuscles adjoining the necrosis in the course of a few hours. The first change consists in an increase in the staining capacity of the nucleus due to an increase in the chromatin. The protoplasm also increases in granulation and in amount. The cell processes

become more numerous and are more plainly visible. The new protoplasmic projections are chiefly formed on the side of the cell adjoining the necrosis and extend in long lines into the necrotic territory. The nuclei divide rapidly by mitosis and the newly formed nuclei pass up into the cell process. These become separated from the parent cell and form new corneal corpuscles. Numerous nuclear figures may be seen in the cells eighteen hours after the injury. The proliferation is not confined to those cells immediately adjoining the necrosis, for nuclear figures may be seen in cells several rows distant and in scattered ones in the cornea elsewhere. There seems to be a general increased activity in all the cells of the cornea, for the cells in the entire tissue stain more actively. All the increase in cells takes place by indirect division of the nucleus. In certain of the corneal corpuscles adjoining the necrosis, where the injury was probably not sufficient to produce at once death of the cells, a process of direct division may be observed. This takes place in large swollen vacuolated corpuscles. There is no increase in the chromatin of the nucleus or in the protoplasm which stains rather more faintly. The nucleus becomes vacuolated and the chromatin collects along the periphery in crescentic masses. Constriction and division of both nucleus and cell follow. In the division of the nucleus the chromatin masses at the periphery remain unchanged. There is no increase in the amount of chromatin or in the interchange of its parts. The process is one of cell fragmentation and not of true proliferation. The various steps can be made out only in separate cells. This peculiar form of degeneration is seen more frequently in some cells than in others; it is especially common in the cells of stratified epithelium.

There is no active participation of the leucocytes in regeneration and it may take place without them. In very slight injuries the leucocytes may not reach the affected region; thus, slight necrosis may be produced by touching the cornea with chloride of zinc, yet all emigration may be absent. When leucocytes are present they usually are seen in greatest numbers immediately around the central eschar, where the tissue is filled with the precipitated silver, and when they are among the proliferating corpuscles they are taken up by these. All of the proliferating cells are phagocytic, and their protoplasm contains numbers of polynuclear leucocytes or their fragments. The enclosed cells or their fragments often lie in vacuoles in the protoplasm. Even the connective-tissue cells at a distance, which show increased activity only by the increase of chromatin in the nuclei, are actively phagocytic and take up the fragments of protoplasm which become separated from the travelling leucocytes. In these early stages the process is easily followed and the derivation of the new cells is plainly seen. Later, the new cells become entirely separated from the old and are found in the central area. Here they present little similarity to the old connective-tissue corpuscles. They are round and irregular in shape, the protoplasm is granular and stains easily, the nucleus is more or less irregular in form and contains an increased amount of chromatin. They are usually larger but may closely resemble mononuclear leucocytes. The same process of cell division and phagocytosis may be seen in the mesentery after various forms of injury.

In more complicated tissues, repair or rather tissue change is not so easily interpreted and a variety of cells are affected. In vascular tissues we almost constantly find plasma cells in the late stages of inflammation, even after twenty-four hours. These may or may not be normally present in the tissue. Considerable numbers of them may be found in the subconjunctival tissue around the edge of the cornea. They are not present in the normal mesentery of the rabbit. They are cells varying in size, somewhat larger than polynuclear leucocytes, with an abundant granular protoplasm which is stained with almost all reagents, but particularly with polychrome methylene blue. The nucleus is round or oval with abundant and brightly stained chromatin arranged peripherally. Larger cells may be seen containing two or more nuclei but otherwise of the same character as the smaller. There is much uncertainty about the origin of these cells. They more closely resemble the lymphoid cells than any others and apparent transitions may be seen between them, the protoplasm and the chromatin of the lymphoid cell increasing in amount and staining more actively. They are amœboid and may be found in the corneal spaces a considerable distance from the periphery. I am strongly of the opinion that they are derived from the lymphoid cells. Others believe that they are produced from the fixed cells of the tissue and take part in tissue formation. They are never phagocytic, but like the lymphoid cells they are taken up by other cells. In some cases they are certainly brought to the part by the blood and pass by emigration from the blood-vessels into the tissue. In the kidney I have seen them in the act of migration. It has not seemed to me that they take any part in tissue formation. They are most numerous in the chronic inflammations in which injury and repair are going on continuously. Nuclear figures are numerous in them and they certainly increase in number in this way. The lymphoid cells take no part in tissue formation. With regard to the large mononuclear leucocytes there is more doubt. It is certain that their presence in the inflamed part is due in part to emigration from the vessels. It is not certain whether this is their only source. I am disposed to regard these cells as endothelial in origin and they may be formed in the part from the endothelium of blood-vessels or other endothelium. It is difficult to differentiate them from young tissue cells. It is not probable that they take any part in tissue formation, but in the difficulty of distinguishing them from cells which certainly are tissue producers it does not seem possible to decide. In the regeneration of the destroyed tissue intercellular substance as well as cells must be formed. Fibrillæ are formed between the cells early. The newly formed intercellular substance may contain elastic fibres and the ordinary fibrillæ of white fibrous tissue. It is generally believed that the fibres are differentiated from an intercellular substance produced by the cells and not formed in them. When the tissue has been entirely destroyed the newly formed connective tissue is not of the same quality as the old. The fibrillæ are thicker and tend to adhere together, forming thick masses, and the tissue is more opaque and may be recognized long afterward as the cicatrix which remains.

The activity of regeneration depends upon the character of the agent which produces the injury. While it begins early and is active in the cornea after simple injuries, there may be no evidence of it twenty-four hours after inoculation with staphylococci. Of course there can be no laws governing the reaction of the tissues to other bacteria, for some of them may directly excite the tissue cells to proliferation.

Tissue proliferation of any extent is accompanied by formative activity in the blood-vessels belonging to the part, leading to a new formation of vessels. (Plate XXXV., Fig. 3.) No better place than the cornea can be selected for the study of this process. Evidences of this formative activity are seen first in a general increase in size and staining capacity of the endothelial cells. The large cells proliferate and form new vessels. The new vessel begins as a process from one of the endothelial cells which projects into the tissue, the process coming from a cell on the side of the vessel toward the injured area. The process enlarges, nuclei are formed by mitotic division and pass into the process, and new cells may be formed from the adjoining endothelium. The process of cells becomes hollow and communicates with the lumen of the vessel from which it arises. The process is pointed at the end and often several points are given off from it. Some of these processes unite with similar processes from other vessels, a loop is formed, and the circulation is thus established. Great numbers of these vascular processes arise, though but few of them form true vessels. It is uncertain whether the growing ends exert a positive attraction for each other or whether the junction is fortuitous. It is possible that the cell spaces of the tissue

have an influence in determining the direction of the growth. In the cornea I have found that new cells may line such a cell space which then becomes a part of the lumen of the new vessel. It is not probable that tissue cells are formed from the vascular cells. The new blood-vessels persist as long as the active cell proliferation is continuing. They finally atrophy and a cornea which showed a marked pannus may again become perfectly clear. The new blood-vessels are formed from that part of the periphery which is nearest to the injury.

The processes of repair are different in the different sorts of tissue. The repair is most perfect and simplest in character in tissues which are least differentiated and homogeneous. This is easily seen in sections passing through the skin and subcutaneous tissues. Laparotomy incisions have supplied us with an abundance of material in man illustrating every stage of inflammation and healing in different tissues. The process can also be studied experimentally by incisions into the rabbit's ear. In incisions of the skin there is less inflammation in the connective tissue than elsewhere. If the wound is not infected and the coaptation of the edges is perfect, healing may take place with almost an absence of leucocytes. New cells are formed from the connective-tissue corpuscles nearest the incision, and permanent union is first effected by the interlacing of the processes of these cells. Here also as in the cornea the cell proliferation is much more extensive than would be required solely for the purpose of regeneration. Nuclear figures may be met with in the tissues at quite a distance from the line of incision and injury. When the section in the rabbit's ear passes through the cartilage no regeneration takes place in this. The portion of cartilage immediately adjoining the incision degenerates and the nuclei break up into nuclear detritus. The ends of the cartilage become included in the connective tissue formed around them, and permanent union takes place by the metaplasia of the connective tissue, the cells becoming transformed into cartilage cells and the fibrillar tissue into the homogeneous matrix. Where the laparotomy wound passes through the subcutaneous fat, healing takes place only after this has been removed. This is accomplished by the presence of large phagocytic cells which appear around the fat droplets. The origin of these cells is uncertain. They suggest in their general characters endothelial cells. By these cells the fat is removed and connective tissue appears in its place. This fat absorption takes place at a considerable distance from the line of incision, and usually numbers of leucocytes are found in the tissues. The large cells are found in injured tissue which contains fat, and they are very numerous around necrotic areas in the central nervous system. The incision through the muscle leads to extensive necrosis which involves the muscle on either side for a considerable distance from the line of incision. The necrotic fibres are invaded first by leucocytes and then by large cells, probably of endothelial origin, by which the necrotic tissues are removed. There is very extensive proliferation of the nuclei of the sarcolemma which seems to take place by direct division and to be degenerative in character. Long rows of vesicular nuclei containing but little chromatin are found in the ends of the fibres and extending some distance along them. I have never found any nuclear figures or evidence of true division here.

The fluid exudation finds its way out of the tissues in various ways. A purulent exudation may produce gradual softening of the overlying tissue and thus provide a way for its discharge. In parts with such a thin covering over them as exists in the alveoli of the lung the exudation easily passes through to the surface. Even when the surface is a mucous membrane the exudation may readily find its way between the cells. The abundant discharge from an inflamed mucous membrane is principally composed of the exudation coming from the tissue beneath, but is mingled with an increased secretion from the mucous glands. The greater portion of the exudation is removed by the lymphatics. It has long been known that the lymphatics coming from an inflamed part are dilated and the flow of lymph is increased. This flow is proportional to the amount of fluid exudation in the part, but it may not be proportional to the amount of tension and swelling in the part, for this may be due to the presence, in the exudation, of material like fibrin which will not pass away by the lymphatics. On microscopic examination the lymphatics in a tissue adjoining an inflamed part are found to be dilated and easily recognizable. It is remarkable that they contain so few cells. The cells of the exudation are not removed, to any extent at least, by the lymphatics. The lymphatics contain an abundant granular coagulum and usually a small bunch of cells, among which may be a few red corpuscles and degenerated leucocytes which probably came from the exudation; but most of the cells within them are cells of endothelial character probably derived from proliferation of the lining cells. This seems to be true of the lymphatics in all tissues of the body and in various forms of inflammation. I have studied carefully the lymphatics of the lungs in various forms of pneumonia. They are dilated, they contain granular coagulum and often considerable fibrin, but they contain few or no cells from the exudation. It does not seem to me probable that any of the exudation cells return to the blood from the tissues either directly from the blood-vessels or indirectly by means of the lymphatics. No one has seen the emigration from the tissues into the blood-vessels in the living tissues nor can any evidence of it be seen in the hardened tissues. It is difficult to see, unless we attribute to them an independent volition, what influence could be exerted on the exudation cells to induce their return. Some of them are devoured by phagocytes, and they undoubtedly contribute in this way to the building up of the tissues, but the great mass of them is probably dissolved or digested by the tissue fluids.

A fibrinous exudation on a surface is removed by the process of organization. In this a highly vascularized tissue grows up into the fibrin and gradually removes and replaces it. How the removal is brought about is uncertain. The fibrin simply disappears before the growing tissue, becoming first more homogeneous and then hyaline. In the last stage of the process irregular, swollen, hyaline masses of fibrin are found on the surface and within the new connective tissue.

Before our recent knowledge of the formation of leucocytes and the conditions under which it takes place there was much uncertainty as to the origin of the great numbers of leucocytes found in an inflamed part. It could not be explained without the assumption of some increase in the number of leucocytes, for those found in the exudation in croupous pneumonia might exceed in number the leucocytes ordinarily contained in the entire mass of the blood. It has been found from blood examinations that in inflammation there is an increase in the number of leucocytes in the blood. In croupous pneumonia there may be eight times the normal number of leucocytes, the increase being only in the neutrophiles. The relation of leucocytosis to inflammation has recently been studied by Brinckerhoff in the rabbit. He has found that in the beginning of the exudation leucocytes are withdrawn from the blood more rapidly than they can be replaced and a condition of hypoleucocytosis is produced. This is but temporary and the number in the blood rapidly increases to the normal and beyond. Examination of the bone marrow at corresponding periods shows that in the beginning of the increase in the blood the amphophiles stored in the marrow rapidly leave it, producing a local hypoleucocytosis which is followed by a rapid new formation of amphophiles. The blood examination gives us a further proof that there is no return of the leucocytes into the blood. In pneumonia the number of leucocytes in the blood rapidly falls at the crisis and may reach the normal at the time when there is the most rapid absorption of the exudation. If the leucocytes did return into the blood there should be an increase at this time.

The forms of inflammation which we have considered so far may be termed acute. They were excited by an agent which produced an injury to the tissue and whose

EXPLANATION OF
PLATE XXXV.

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FIG. 1.—Muscle in Vicinity of Necrosis Produced by Croton Oil, Showing Absorption of Necrotic Muscle Fibres and Proliferation of Tissue Cells. 4 mm. Zeiss.

FIG. 2.—Single Muscle Fibre Undergoing Absorption by Endothelial Cells. The dark masses represent the remains of the necrotic tissue. All the fissures in it are filled by the protoplasm of the phagocytes. One-twelfth hom. immersion, Zeiss.

FIG. 3.—New Blood-Vessels Extending into Cornea from Scleral Margin Three Days after Inflammation Produced by Inoculation with Staphylococcus Aureus. 8 mm. Zeiss.

FIG. 4.—Granulation Tissue Showing Newly Formed Blood-Vessel from which Emigration is Taking Place. 4 mm. Zeiss.

FIG. 5.—Edge of Muscle Showing Ingrowth of Connective Tissue Five Days after Inflammation Produced by the Application of Croton Oil. 8 mm. Zeiss.

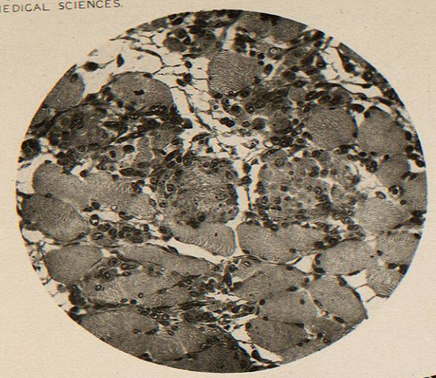

FIG. I.

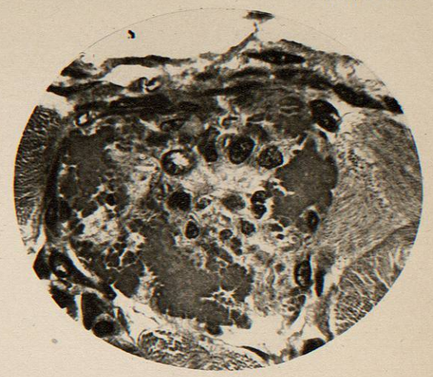

FIG. 2.

FIG. 3.

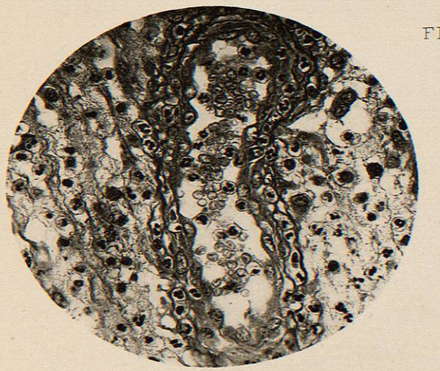

FIG. 4.

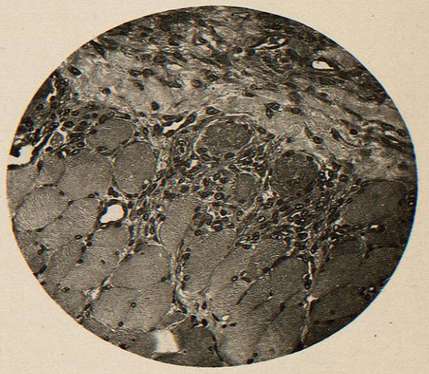

FIG. 5.

action ceased with the production of this injury. The tissue changes were the result rather of the injury produced by the agent than of the agent itself. It is possible in various ways to excite an inflammation in which all the phenomena which have been seen in acute inflammation develop much more slowly, or some of them may be entirely wanting, the whole process lasting much longer or even indefinitely. Such inflammations are termed chronic. It is of course impossible to assign any limits to the acute inflammations, and we cannot say that after so many days an inflammation ceases to be acute. The terms acute and chronic, therefore, while convenient, are difficult of definition. Chronic inflammations may be produced, first by the continuous action of an injurious agent, secondly by the repeated action of slight causes on a tissue which is not normal and not capable of resisting this action. In chronic inflammation, again, the changes in the tissue are marked and exceed in importance the exudation. The best types of chronic inflammation are seen in the inflammations around foreign bodies and in the ulcer. Inflammation surrounding foreign bodies may be studied by the introduction of various foreign bodies into the tissues of rabbits, and by the accidental material which is provided by surgery. Silk suture may remain in the tissues an indefinite time without undergoing absorption. The changes which take place are of two sorts: first, those which have for their object the absorption of the material (Plate XXXV., Figs. 1, 2); second, those which lead to the formation of an indefinite tissue which can resist the action of the foreign substance. Sections made across a silk suture which has been introduced into the muscles of the leg of a rabbit show in twenty-four hours around the suture an area of necrotic muscle. If the suture has been soaked in corrosive sublimate the fibres immediately adjoining it show no change. They have been killed by the corrosive and preserved in their normal state in the same way as muscle is hardened in a preservative. Such muscle fibres resist invasion by phagocytes. Farther out is an area where the nuclei of the fibres have disappeared and the fibres are swollen and hyaline. There is an abundant exudation of leucocytes which infiltrate the necrotic muscle and the suture. There is a slight new formation of cells in the surrounding tissue and the ordinary nuclear increase in the muscle nuclei. Sections made at late periods show first the degeneration and final disappearance of the leucocytes and the necrotic muscle fibres. The necrotic fibres are invaded by leucocytes, but it has not seemed to me that these are the true phagocytes. The final removal is effected by cells of the endothelial type (Plate XXXV., Figs. 1, 2) for which the way has been prepared by the leucocytes. The leucocytes all degenerate. The place of the exudation cells and necrotic tissue is taken by a tissue composed for the most part of spindle or irregularly shaped cells with large nuclei. Around the silk fibrils there are large protoplasmic masses containing a number of nuclei. These may surround the fibrils, and on longitudinal section are found to extend along them a considerable distance. These giant cells are formed from the cells of the tissue. They may be produced by coalescence of cells, or by the increase in size of a single cell with proliferation of nuclei but without succeeding division of protoplasm. They are formed about foreign bodies of all sorts. In wounds of the skin they are found with enclosed masses of connective tissue and elastic fibres. The silk fibres enclosed within them undergo no change even in the course of years. Sections made several weeks after introduction of the suture show that nearly all of the newly formed tissue has disappeared and the suture is surrounded by a thin mass of connective tissue, but which extends for some distance between the fibres of the surrounding muscle. The amount of the newly formed connective tissue varies greatly in different cases and according to the character of the material used for suture. It is very much greater around catgut than around silk suture. The catgut suture may remain unchanged in the tissue for a considerable time. It appears on section as a dense homogeneous mass surrounded by leucocytes, and later by newly formed tissue. Leucocytes do not invade it until cracks and fissures are formed in it. It appears rather to undergo slow softening and dissolution in the tissue than to be removed by phagocytes.

In the ulcer there is a combination of conditions. There is a constant trauma acting on the surface which has been deprived of its protective covering. The surface of a skin ulcer is always covered with a thin layer of necrotic tissue, within and below which there is an abundant infiltration with leucocytes. Extending to a variable depth below the surface there is a tissue composed of newly formed blood-vessels and of young connective tissue rich in cells. Between the cells of the connective tissue there are a few connective-tissue fibrillæ. The whole tissue is loose and œdematous, it contains great numbers of lymphoid and plasma cells, principally in groups around the vessels. The newly formed blood-vessels are large (Plate XXXV., Fig. 4), their walls are composed of large endothelial cells, with large nuclei, projecting into the lumen, and from them there is abundant emigration. This tissue remains until the surface is covered with a regenerating epithelium, and is finally replaced by dense connective tissue containing few cells or vessels. Such a tissue is made less resistant to traumatic influences than is normal tissue. In the first place it probably receives more injury from a blow or from pressure owing to its density and consequent inability to distribute the influences of the trauma. The blood-vessels in it are few in number and owing to the density of the tissue are less capable of quick dilatation and the increased nutrition necessary for repair. This condition is always to be considered in chronic inflammation. The repair of a tissue after an injury involving destruction of complicated structure is never perfect. Repair involving cells alone easily takes place and is perfect, but the tissues seem to have lost the power of forming again tissue of the same character as that produced in the embryonic condition. Chronic inflammation is frequently the result of repeated separate injuries produced by influences which come within the normal, acting on a tissue whose capacity for resistance and repair is low and which is continually lowered by each succeeding attack.

Healing of the abscess may take place in a number of ways. The exudation and with it most of the infectious agents which have been produced may be removed in the ways we have spoken of. There remains a surface which is very similar to the surface of an ulcer. It is infiltrated with leucocytes and contains numerous vessels and young tissue cells. The bacteria are partly removed, many of them are enclosed in phagocytic cells, the tissue has become resistant to the action of those that remain, and thus a local immunity is produced. The cavity becomes obliterated by the contraction of the tissue and the growing together of the walls.

It would be impossible within the limits of this article to consider further the special forms of inflammation due to the character of the tissue or the nature of the injurious agent. In certain organs the processes of inflammation may differ in a marked degree. In the kidney, for instance, there are lesions which come under the broad interpretation of inflammation which are not found in any other situation. In general, however, all the differences which are met with are but variations of the processes which have been described. *W. T. Councilman.*

INFLUENZA.—*Influenza* is an acute, self-limited, infectious fever, occurring in widely distributed epidemics, and characterized by catarrhal inflammation of the respiratory and gastro-intestinal mucosa, by profound nervous disturbances, and by extreme debility.

Synonyms: Febris catarrhalis; Epidemic catarrhal Fever; La Grippe; Grip; Tac; Horion; *La Dando; Ziep; Epidemischer Husten; Epidemischer Schnupfen; Schafhusten; Blitz-Catarrh; Modefieber; Mal Russe; Snufsjuka* (Swedish); *Qual-Tong* (Chinese).

Many other synonyms, grave and humorous, might be listed which have been suggested by the peculiarities of various epidemics, the national characteristics of the