certain regions which are predisposed thereto on account of their greater distance from the heart or of an unfavorable construction of their local venous and lymphatic plexuses, perhaps also of the local innervation.

Examples of the latter are the well-known stases in the lower limbs of females who have borne several children, or that condition which is termed acne rosacea of the face, etc.

We must also regard, as a further example, the local circulatory disturbance left over after superficial and also after deeply spreading inflammations of the skin, and which constitutes a transition to so-called chronic inflammations of the skin.

We have to deal, therefore, essentially with two conditions: either with a pre-existing venous disturbance or with a passive hyperæmia produced by an acute inflammation of the skin. We can bring these two conditions into closer relations if we assume that an active fluxion is added to the pre-existing disturbance, that an inflammatory irritant acts upon a part of the skin which is in a state of passive congestion. The differerence between both cases evidently lies in the question of priority.

We now have to settle the following points:

1. What produces the venous stasis itself, and what relation does it bear to inflammation of the skin? And further,

2. What results follow the combination of both factors in one or the other series of priority?

Experiments have shown that incomplete obstruction of the venous return flow uniformly produces a diminution of the rapidity of the current and coldness of the parts, then transudation of serum, either colorless or (in man) tinged with hæmatin, and finally, the passage of red blood-globules through the walls of the capillaries and small veins, the latter being recognized by numerous, dark red, punctate extravasations chiefly in the neighborhood of the ligature.

In the integument of the human arm the process in general results in the irregular propagation of the incomplete circulatory obstruction, produced by ligature of the veins, from the point of ligature gradually to the various capillary districts of the integument. The optical expression of this stasis is cyanosis. If the stasis continues for some time, the blood plasma passes through the walls of the capillaries, perhaps also of the smaller veins, but at first in very slight amount, so that the size of the limb is not strikingly increased. Diapedesis now begins; hemorrhages as large as the point of a pin develop, most numerous in the neighborhood of the point of ligature.

Around these ecchymoses, the blood plasma, which transuded at the same time, or even earlier, assumes a reddish color.

The vermilion spots, which develop below a ligature which has been applied for some time, are due to admixture of hæmoglobin with the blood plasma, and constitute a transition from serous transudation to the exit of red blood-globules in substance. Between these are noticed white spots, which are produced evidently by the irregular filling of the capillary districts with blood, in consequence of the incomplete stasis of the venous return flow.

These are the phenomena produced by ligature of the veins or stasis upon the human integument, and within a few minutes after the occurrence of the obstruction. If the latter is removed, in a short time all the results of the stasis disappear.

The question now arises, what happens when the stasis persists for a longer period?

In man, experimentation in this regard cannot pass beyond certain boundaries, but we have sufficient knowledge on this point from experiments on animals, and morbid processes in man.

Experiments have shown that the occlusion of the venous return flow, *a*, when it has continued too long, and is not relieved, finally results in complete cessation of the circulation and necrosis; *b*, when it is long-continued, but is relieved before necrosis can occur, produces acute inflammation, not that the stasis passes into inflammation, but that it acts as a vigorous inflammatory irritant; *c*, when it is relieved early, produces no further phenomena than the above-mentioned œdema and temporary cyanosis, perhaps here and there a few ecchymoses.

As in animals, similar results, from a clinical standpoint, are found with regard to venous stasis in man and its relations to inflammation. Venous stasis in a circumscribed territory, whether produced purposely or arising spontaneously, causes cyanosis, œdema, exudation of hæmatin and red globules, but not of white globules; it has nothing in common, therefore, with the inflammatory change in the walls of the vessels. As in animals, however, it is capable of producing inflammation when it has persisted for a longer period, and is then relieved.

The process just described is, however, less important with regard to the integument than the other case, viz., an incomplete stasis of long duration, such as is observed in general cachectic conditions, and in unfavorable local circulatory conditions.

My experiments have taught that the purely inflammatory phenomena which external irritants produce under such circumstances are usually less marked, and that the formation of pus occurs more slowly, but that the exudation of serous and bloody fluid, furthermore the formation of free ecchymoses, are more frequent; that an inflammatory granulation occurs with greater difficulty; that the entire tissue presents a livid infiltration and dark blue areolæ which cannot be made to disappear entirely upon pressure, and in which the elevation of temperature is less distinctly marked; but finally, that the inflammatory process rapidly terminates in necrosis.

These appearances are found in acne rosacea, in ulcers with a varicose foundation (ulcers of the foot), in eczema under such conditions, etc.

The phenomena of inflammation are thus, on the whole, concealed to a certain extent by incomplete stasis, they occur less distinctly and rapidly.

On the other hand, when inflammation is already present, the symptoms peculiar to stasis (cyanosis, œdema, exudation of hæmatin and ecchymoses) appear so much more distinctly after subsequent venous stasis, the more marked the inflammatory change has been.

Ligature experiments which I have made upon cases of scarlatina, etc., have shown that the vermilion spots around the ecchymoses occur most certainly and intensely in those positions in which the most marked erythema is situated.

From this fact and from experiments upon frogs and rabbits, it appears that, in moderate grades of inflammation, incomplete stasis is followed by slight phenomena (escape of serum and hæmatin); in higher grades, especially those associated with suppuration, by the enormous escape of blood-globules into the tissue of the cutis. But the stasis is by no means capable of producing more marked suppuration in an inflamed part which is already suppurating. There is no doubt, therefore, that venous stasis in itself has nothing to do with inflammation, *i. e.*, with the change in the walls of the vessels assumed therein. There is no doubt, on the other hand, that the stasis may act like another irritant, that phenomena of inflammation may be developed by stasis after some time.

These facts enable us to understand that inflammatory processes under disordered circulatory conditions, although not without a distinct difference from ordinary inflammations, nevertheless run their course without any change in the main type of the process.

With the aid of these physiologico-pathological data it will not be difficult to paint a picture of that chronic inflammation of the skin, of a superficial and deep-seated character, which so often prevents the resolution of the pure inflammatory process, and often leads to unfavorable terminations.

Previously, however, we must again recur to inflammation of the skin itself, and to some forms which constitute a transition from pure inflammations to the chronic forms of inflammation, developing at a later period during the cessation of inflammation. We will call them stasis-inflammations of the skin, and separate them into stasis-catarrhs and stasis-phlegmons, according as they occur superficially or are more deeply seated. To the former belong ecthyma and the superficial ulceration of the skin, to the latter, phlebitis and lymphangioitis cutis, and erysipelas.

The examination of ecthyma pustules upon the leg showed the well-known appearances of an ulcerated pustule: infiltration of the base of pustule, of the tissue of the corium beneath the pustule, and in the immediate neighborhood, with dense masses of cells and granular detritus giving rise to an obliteration of the boundary between the epidermis and corium.

In such cases the differentiation from pure forms of dermatitis lies in the fact that the termination in necrosis of the base, which is confined to the pustule itself, is caused by the unfavorable nutritive conditions of this base, while this termination is exceptional in other forms of pustular formations; for example, in small-pox, pustular eczema due to croton oil, etc.

This corresponds to the definition of ecthyma originally given by Willan:

"An eruption of large phlyzacical (*i. e.*, surrounded by a zone) pustules, each of which is situated on a hard elevated base, and terminates in the formation of a thick, hard, greenish, or dark-colored crust. They are separate, distributed over a slight area, and are not contagious."

Analogous to the superficial forms here described, certain transition forms between congestion and stasis also occur deep in the skin, viz., phlebitis and lymphangioitis of the skin and erysipelas, and in this complication of superficial arterial congestion with marked stasis symptoms more deeply, the direct relationship is made evident between these phlegmonous processes and the pure stasis diseases, in which arterial congestion is entirely absent or plays a subordinate or accidental part.

Erysipelas furnishes us with a typical picture of the stasis phlegmons.

Here the vessels extending into the subcutaneous tissue are filled with blood; the tissue of the cutis soaked with serum, its meshes dilated and filled with white blood-globules which are especially profuse around the deep venous trunks and almost conceal them, but are also accumulated between the fat-cells and around the sudoriparous glands. I have found the lumina of the larger lymphatic vessels gaping; their distention with lymph-corpuscles was not demonstrable. Renaut found the panniculus adiposus inflamed and swollen.

In the papillary layer of the corium, the cellular infiltration is also present, but less dense than lower down. In the cases of the development of vesicles, the corresponding changes may be observed as in the formation of vesicles in general.

The cellular infiltration and inflammatory œdema of erysipelas disappear as completely in those portions of skin which return to the normal, as they do in superficial inflammatory processes.

It is evident from the anatomical appearances and clinical signs that in true erysipelas we always have to deal with an affection of the lymphatic vessels (and blood-vessels) deep down and extending into the subcutaneous tissue. While a rosy redness is present in superficial erythema, erysipelatous redness shows a peculiar bluish-red glistening with a yellowish border, corresponding to a congestion deep down in the skin covered by a thicker layer of tissue, and the œdematous swelling corresponding to the serous infiltration of the connective tissue in the depth of the skin.

Erysipelas must be regarded probably as a capillary lymphangioitis and capillary phlebitis, which can only be distinguished by its superficial expansion from the band-shaped redness of phlebitis and lymphangioitis of larger vessels. As a matter of course, the process results secondarily in an affection of the upper layers of the skin, the papillary layers of the cutis, and the epidermis.

There is much to be said in favor of the view that the capillary lymphangitis and phlebitis, which give rise to erysipelas, must be attributed to infection by a microparasite. Very recently, Fehleisen produced erysipelas directly in man by the inoculation of cocci cultivated from erysipelatous skin (Verh. d. Würzburger phys.-med. Ges., 1882). The anatomical and clinical process in erysipelas may, indeed, be explained very plausibly in this manner.

We may now conclude the nosology of chronic inflammation of the skin. As previously explained, we have to deal with a case in which stasis is induced and gradually superadded to a previous inflammatory affection. It is evident from experiment that these conditions of stasis should not be regarded directly as inflammatory processes, but merely as incomplete phenomena of stasis secondarily caused by the latter, that accordingly the phenomena of the real inflammatory process actually have been extinguished in them. In the human skin the following changes occur as evidences of this improperly termed chronic inflammation, which should be really called "chronic stasis in consequence of previous inflammation."

1. The phenomena of inflammatory œdema give place to the œdema of stasis, *i. e.*, the escape of blood-serum from the veins. At the same time, the connective-tissue cells present in the connective tissue of the skin begin to grow and proliferate, and in their further course present the various stages of development into fibrous and elastic networks and bands, new formed blood-vessels, etc.

The clinical signs of this condition are: infiltration and thickening of the skin, at first with purely serous contents which may be forced away partially upon pressure; a dark-red, gradually passing more and more into a bluish tint, of the surface upon which a disturbance develops secondarily in the formation of new horny layers, inasmuch as a slow, branny desquamation occurs. This is the typical highest stage of chronic superficial inflammation of the skin, the so-called chronic eczema. As a rule, however, the process terminates in a return to the normal, although often not until after a long duration of the stasis.

2. Under specially unfavorable circumstances, such cases also may result in further degeneration, in those necrobiotic processes which occur ordinarily only as terminations of phlegmonous inflammations. Thus, chronic eczema terminates occasionally in sclerosis of the connective tissue of the skin, together with stasis processes in the sanguineous and lymphatic capillaries, *i. e.*, in pachydermie.

The pustules of acne and variola end occasionally in the formation of ulcers and cicatrices, while these terminations constitute the rule in deeply spreading phlegmons. Here they appear either as necrobiosis with softening and necrosis of the tissues in layers (in phlegmons affecting the layers of the skin, such as burns and congelation), or in

the form of circumscribed degenerative processes with ulceration and gangrenous degeneration (in localized phlegmons: furuncles, anthrax, and the like).

Under the same conditions, as in other regions, fatty and amyloid degeneration, mucoid metamorphosis, cheesy degeneration, and calcification play their part in the integument.

THE INFLAMMATORY NUTRITIVE DISTURBANCE OF THE SKIN IN ITS RELATION TO ANGIONEUROTIC AND NEURITIC PROCESSES.

It is now incumbent upon us to examine the large number of inflammatory processes occurring upon the skin, with regard to their connection with the entire organism, and with morbid changes in the latter. Attention has been called in various places to the close connection between inflammation of the skin and the condition of the entire organism, to the fact that many local fluxions are effected directly through the agency of other organs without an external irritant, and also to the reaction of inflammations of the skin upon the entire organism, its nutrition and diseases.

But apart from this general causal nexus, there are other special factors which are so important that they powerfully influence the course, even the character of such inflammations. This will be made clear by a comparison of a few distinct types of skin disease with one another, for example, acute eczema produced by croton oil, the variola eruption, and herpes zoster.

In the beginning, all three forms present hyperæmia of the skin, followed by the formation of nodules, vesicles, and pustules. As is well known, eczema nodules may pass into vesicles, and the latter may be converted into pustules; this is the case in variola, and in herpes zoster, a few vesicles develop into pustules.

The anatomical picture is always that of the inflammatory efflorescence, starting from the papillary body and its blood-vessels, with subsequent changes in the epidermis, swelling and formation of a network, inclosure of serous, then of purulent contents in their centre, finally drying of these contents, and development of new epidermis above the papillary border.

Despite the fact that the clinical course of the inflammatory process upon the skin develops under all circumstances according to the same anatomical and nosological pattern, nevertheless a separation of the three conceptions of the disease is afforded by other factors which do not depend upon the local effect of the inflammation.

If we retain the above illustrations, we will find that an intense acute eczema, produced, for example, by the prolonged action of croton oil, allows the local action of the irritant upon the structure of the skin to appear most markedly and exclusively; marked erythema, extensive and diffuse serous infiltration of the diseased parts, rapid formation of vesicles, occasionally leading quickly to excoriations of the upper layers of the skin, gradual transition into desquamation and recovery. On the other hand, we find in severe cases of variola, a typical, but slightly variable period of incubation, characterized by general symptoms of the disease, without any localization, then a prodromal febrile stage, and a diffuse prodromal erythema which is entirely independent of the later position of the efflorescences; tendency to ecchymoses (variola hæmorrhagica); development and progress of the individual pocks, not alone according to the types of inflammatory efflorescence formation, but also with a typical duration of the individual phases of development, and without diffuse serous infiltration of the skin between the pustules, as in eczema; finally termination of the process with the drying up of the individual pustules,

or formation of ulcers and cicatrization without persistence of a chronic inflammatory process, while this is not infrequently the case in eczema.

The peculiarity of this course is connected evidently with an influence foreign to the inflammatory process of the first variety, the point of attack of which must be sought in the circulation of the blood, in the walls of the blood-vessels, and the blood itself. The mere progress of the process in the integument is alone sufficient to show the difference between the variola process and that of acute eczema, despite the identity of the purely inflammatory processes.

If we pass to the third one of the illustrations chosen, to the so-called herpes zoster, we will find in it:

Frequent neuralgic pains, as the prodromal stage, in the distribution of the tegumentary branches of a certain nerve, then the coincident occurrence of a series of inflammatory nodules upon a hyperæmic basis, which soon pass into vesicles, partly also into pustules; then development and progress of one or more other groups of vesicles according to the same type and the same course, always along the branches of some nerve; finally, after the termination of the local inflammation, occasional continuance of the neuralgia. These forms of inflammation of the skin always depend upon a neuritis of the nerve-trunk, or an affection of its ganglion.

If we compare the three morbid processes just mentioned, we will arrive at the following conclusions:

1. The process of inflammation in the skin runs its course according to a pathological schema which agrees with the general symptomatology of the inflammation of organs in general. Hereafter we will describe this process as simple inflammatory process of the skin (dermatitis simplex).

2. This inflammatory process constitutes a nosological entity which cannot be destroyed either by the predominance of one or the other symptom of inflammation, or by the degree of development which the process reaches in individual cases.

3. In addition, there are inflammations of the skin the development, course, and terminations of which correspond to the nosological schema of this inflammation, but in which other phenomena are vividly presented, the origin of which cannot be found among purely inflammatory factors, or only when the latter have increased to such an extent as does not seem capable of furthering the inflammatory process. This occurs in smallpox and zoster. These forms of inflammation may be termed partly angioneurotic, partly neuritic dermatoses, according to the character of the most prominent factor. Their difference from simple inflammations of the skin resides in the fact that the character of the general constitutional influence is such that it essentially changes the form of the inflammatory process in the skin. And this alone is the reason why a clinical separation of these three forms of inflammatory dermatoses appeared advisable.

a. Changes in vascular tonus always occur in congestions produced by inflammatory irritants, and cannot develop without active or passive stimulation of the muscular coat of the vessels within and around the inflamed part. And furthermore as a consequence of, and during the course of the inflammatory process, changes in the vascular tonus readily develop and may find an expression in chronic disturbances of the circulation and absorption. But the change of vascular tonus, to which reference is made here, manifests itself first by its relative independence of the inflammatory process; secondly, by its direct connection with the etiological factors which have given rise to it, and thirdly, by the extension of its influence beyond the point of action of the irritant. The etiological factor in all diseases of the skin belonging to this category bears the peculiar

character of an agent which acts toxically upon nutrition. This implies at the same time that we have to deal with an irritant action which, starting from a certain central point, radiates in all directions, or, at least, which recurs and repeatedly attacks the periphery.

All the affections of the skin belonging in this category correspond to the following nosological conception: a peculiar irritant acting upon any part of the organism whatsoever, influences a centre of vascular nerves, either directly or in a reflex manner, so that a change occurs in the tonus of the vascular branches belonging to it. Whether this "centre" is identical with one of the large vaso-motor centres in the medulla oblongata or spinal cord, or must be sought in the small ganglionic nerve elements of the walls of the vessels themselves, can be determined in general with great difficulty.

Equally difficult is the solution of the question whether we have to deal with phenomena of irritation or paralysis. We cannot doubt that vaso-dilators as well as vaso-constrictors are active in the walls of the blood-vessels of the skin, and that an irritation of the dilators may thus effect the same object as paralysis of the constrictors, viz., dilatation of the vessels. The reverse holds true concerning contraction of the vessels. As the first series appears to correspond more to the picture of arterial active fluxion, the second series to that of passive congestion, and at the same time bears the stamp of a spasmodic contraction of parts of the vessels, such as we find especially marked in urticaria, it does not seem to be too bold an hypothesis to state that we have to deal in the first series with an irritant effect upon the dilators or paralysis of the constrictors, in the second series with irritation of the constrictors or paralysis of the dilators. Dilatation and contraction of the lumina of the vessels may be observed at the same time in different parts of the same vessels, one form not infrequently as a sequel of the other. It should also be taken into consideration that the contractility of the capillaries can scarcely be doubted.

Among the angioneurotic inflammations of the skin, I place the acute exanthemata in the first rank.

In their essence these diseases are poisons of general nutrition. Their symptoms appear in the most varied tissues and organs, also upon the integument. In the latter the effects are manifested always in the form of a peripheral angioneurosis, as dilatation of the vessels and hyperæmia of larger or smaller portions of the skin. Under the influence of the general febrile condition and the continuance of this change of tonus in the walls of the vessels, those changes in the latter begin which are produced otherwise by inflammatory irritants of a different kind.

In this manner local inflammations of the skin are begun, the further course of which is variable. In some acute exanthemata the inflammatory change is also a superficial catarrhal one and is restricted either to the development of erythema, or an inflammatory exudation appears more distinctly with formation of papules and patches of pigment, or, in a third series, the formation of vesicles and pustules occurs, but always retains the character of superficial inflammation.

The appearances in scarlatina are, indeed, exactly the same as in the roseola of typhoid fever or cholera: at first, simple hyperæmia without any change in the tissue of the corium; further, in some parts of the skin, occasional accumulation of red blood-globules, in certain places ecchymoses and suffusion with blood pigment (yellowish color of the skin) which latter phenomena may be attributed to the general morbid character of these infectious diseases. However, the inflammatory œdematous swelling of the skin is due to the inflammation of the skin proper, unless it has been produced by an early renal affection.

A greater implication of the follicles of the skin is evident in measles, and is manifested clinically by the formation of nodules at the site of the openings, microscopically by abundant accumulations of cells around the excretory ducts. We are justified in regarding the morbilli process as spreading more deeply in the integument than that of scarlatina, and this is further substantiated by the fact that distinct dark pigmentation remains behind after the morbilli spots and nodules have grown pale, and only a yellowish color remains after scarlatina. But that both processes play merely the part of catarrh of the skin is shown by the appearances upon the mucous membrane of the larynx and pharynx, which merit evidently the most complete analogy with the external integument. The increased secretion of the mucous membranes corresponds also to the desquamation after measles and scarlatina.

It is different in a second series of the infectious angioneuroses, in which deep-seated abscess formations of a diphtheritic, or, at least, phlegmonous character occur, and evidently bear the character of specific efflorescences (pustules of variola, glanders, and splenic fever) in connection with the continued circulation of the specific virus. The anatomical conditions in these forms of inflammation have been described above in their main features, and we may, therefore, regard them as known.

The prominence of the angioneurotic disorder in the acute exanthemata is evinced still more clearly by the undeniable relationship between them and the toxic angioneuroses of the skin, to which belong, in the first rank, the so-called medicinal exanthemata. Here also the symptoms of a vaso-motor neurosis are not alone prominent in the beginning, but apparently are often the sole ones until the termination of the process. In other cases they are associated with nutritive disturbances of the skin which bear mainly the character of inflammatory congestion in the most varied grades.

The apparent relationship of certain of these diseases with infectious exanthemata is so great that mistakes arise not infrequently (scarlatina and quinine eruption, between measles and certain maculo-papular eruptions produced by resinous substances, such as balsam of copaiba, etc.).

And finally a third group exists which I will call the essential angioneuroses of the skin. The cause of the diseases belonging to this class is to be regarded as a general increased sensitiveness of the vaso-motor nerves to irritants of all kinds, which appear either in a persistent or cyclically recurring manner, and the expression of which may be regarded as the tendency of the skin to respond to the slightest contact with dilatation or spasm of the vessels, in the beginning only at the point of irritation, but soon in a larger zone around the latter.

Upon such an integument sharply defined erythema or even distinct wheals with a white, anæmic border develop at every contact; they disappear only after a long time, and after they have spread upon the surface for some time.

This phenomenon is merely a morbid increase of the vaso-motor sensitiveness of the skin which is present also in the healthy condition.

This condition of increased vaso-motor sensitiveness is to be regarded as the real pathological change in the forms of disease belonging to this category, but which requires for its development either an increase of the cause of the disease itself, or an accidental irritant not standing in direct connection with it.

While, therefore, in the infectious and toxic angioneuroses of the integument, the poison circulating in the organism seems to be the excitant both of the central vaso-motor irritative conditions and those located in the skin, and the angioneurosis disappears with its disappearance, the general vaso-motor disturbance in diseases of the third

group is a persistent or cyclically recurring one. The addition of an internal or external irritant, however, is necessary to the development of the skin affection. It is, therefore, wrong to call these diseases purely vaso-motor. Not the inflammatory disturbance, but the tendency of the skin to respond with such a change to irritants of the most varied and often trifling character must be regarded as the real disease; this distinguishes these nutritive disturbances from the inflammations produced by inflammatory irritants upon normal skin.

Only when looked at in this manner does the vaso-motor hypothesis agree with the facts; the catchword "angioneurosis" alone, as is so fashionable at the present time, is in no respect sufficient.

b. The second category of inflammations of the skin which branch off from the simple forms of dermatitis, is that of the neuritic inflammations, *i. e.*, those produced by diseases of the nerve trunks or centres.

These affections, which are often termed "trophoneuroses of the skin," have not been excluded hitherto by dermatologists from the category of dermatitides in general. But they require an independent position, because the etiological factor has an essential influence upon the symptoms and course of the disease. Here stress need merely be laid upon the fact that I prefer the term "neuritic dermatoses," to that of "trophoneuroses," because,

1. The relation of the trophic processes in the skin to their innervation in general has been cleared up to but a slight extent from the physiological side;

2. Because the separation of this group was adopted by me merely for the special reason that a morbid process in the nerves can be constantly determined objectively as the causal factor.

Such neuritides may give rise to nutritive disturbances of the skin, which differ in their course from ordinary inflammations. The inflammation either appears and runs its course in a strictly cyclical form as in herpes zoster, or, with a less strict type of course, the inflammatory processes present a great variety of form and grouping (the neuritic erythanthems with their development of nodules, vesicles, pustules, and wheals), finally, thirdly, by their tendency to further nutritive disturbances as, for example, to disturbances of absorption (œdema) and quite often to a diminution (atrophy) or even complete extinction (necrosis) of the nutrition of certain portions of the skin. To the latter category belong neuritic lioderma (glossy skin of American authors), alopecia neuritica, leucoderma neuritica (morphœa, vitiligo), decubitus acutus of Samuel, etc.

The classical picture of dermatitis neuritica of a cyclical course is furnished by herpes zoster, the trophic tegumentary change in which corresponds to the distribution of sensory nerve trunks.

There are so many transitions between the symptoms of typical herpes zoster and those of an anomaly of development, for example, of the hairs in consequence of neuritic processes, that their complete description must be reserved for special consideration. I include all these forms—because they are always situated upon an inflamed, reddened base, and present the most different and variable efflorescences—under the same generic term erythanthema, but with the specific limitation "neuriticum" in order to distinguish it from the other variety

The symptomatology of the non-cyclical, neuritic nutritive disturbances of the skin with an atrophic character is furnished by glossy skin, an affection which occurs occasionally after injuries of nerves, but only when no complete solution of continuity in the nerve has occurred. It begins with erythema, which gives way to a smooth shining appearance of the thinned integument.

Finally, the purely necrotic process in the integument in consequence of neuritis may be studied most clearly in the so-called acute decubitus—a redness with formation of vesicles which occur suddenly upon the paralyzed or anæsthetic side in diseases of the brain and spinal cord, almost always in the sacral region without any demonstrable pressure, and rapidly passes into necrosis.

REVIEW OF THE INDIVIDUAL FORMS OF INFLAMMATION OF THE SKIN.

If we now review the pathological processes in inflammation of the skin in general and compare with them the symptomatology and anatomical appearances of the individual forms of dermatitis, we will find

1. The most superficial and mildest forms, the temporary erythemata produced by mild irritants, such as erythema solare and the like, and erythema of longer duration but slight intensity, like erythema neonatorum.

2. A somewhat more prolonged duration of the erythema and the occurrence of more or less intense œdema are found—

a. in various acute infectious diseases: measles and scarlatina, the prodromal rashes of variola, furthermore in the roseola of typhus and cholera. We have to deal here with erythemas which are impressed with the character of a severe general disease and, upon the skin, with the peculiar character of the angioneuroses, but which, if the tegumentary processes per se are considered, can only be interpreted as simple hyperæmia with more or less inflammatory œdema.

b. In the various forms of development of wheals (erythema papulatum, pomphosis, urticaria, cnidosis). Here also we have to deal with erythema associated with inflammatory œdema, but under certain peculiar modifications, viz., the combination with spasm of individual vascular tracts, which occurs most constantly in true angioneuroses, but also accessorily in simple inflammations and in inflammations of the skin produced by neuritis. Erythema papulatum and urticaria in toxic and the so-called essential angioneuroses of the skin, as well as in neuritic dermatoses, depend upon this process, which really constitutes merely a symptom of inflammation, but no real independent disease.

3. The complete development of the inflammatory process may be learned in various morbid processes which we may attribute, partly with entire certainty, partly with great probability, to an intensified form of the inflammatory irritant and its vigorous action.

Here may be included:

a. Some of the series of erosive superficial inflammations, for example, the efflorescences which are produced by the acarus; furthermore, the forms of diffuse superficial inflammations (eczema) and of follicular superficial inflammations (miliaria, acne, sycosis);

b. The final variola exanthem.

c. A series of inflammatory superficial affections of the skin, which are distinguished by the manifold character of their efflorescence development from simple hyperæmia to the most fully developed pustular formation: the erythanthemata eruptions upon an erythematous basis, to which belong, among the series of the angioneuroses of the skin, the "polymorphous" erythemas of Hebra and those produced by drugs (toxic), and, finally, neuritic skin diseases of an inflammatory nature.

At a later period we will have an opportunity of noting that the variability of the form as well as certain peculiarities of the development and course of the primary lesion in all

these cases do not arise from the mere inflammatory irritants themselves, but from other nosological factors. From an anatomo-pathological standpoint, however, there is no difference between these and other inflammations of the skin of a superficial character.

d. Certain inflammatory dermatoses, which are called "irritative forms" (Virchow) of dyscrasic processes, such as syphilis, scrofula, etc.; they appear as maculæ, nodules, vesicles, pustules.

The processes giving rise to them are of such a wide-spreading character that they are no longer restricted, in severe cases, to the production of superficial dermatitides, but also extend their action into the deeper parts of the skin and give rise to furuncular, ulcerative, and destructive forms, the main pathological sites of which are the deeper layers of the corium and the subcutaneous tissue. And, finally, all these irritative processes are associated with further processes which can scarcely be termed inflammatory, but rather developmental anomalies of the connective-tissue elements, as granulation-like new formations of an embryonal type, and which impart to syphilis, scrofula, and other affections their peculiar type. We were compelled to mention them at this time because that part of their symptoms, which is called irritative, presents in many cases perfectly sharp pictures of the inflammatory process. Too much stress cannot be laid upon the fact that it is not the cellular infiltration per se which constitutes the essence of one or the other process, viz., inflammation or granulation, but that the real feature is the anatomo-pathological course of this cellular infiltration, which is soon absorbed in inflammatory processes of a superficial character, in more deeply seated ones assumes necrobiotic terminations (degeneration, ulceration, destruction), but, in granulation processes, continues for some time as a more persistent infiltration without further development into higher forms of tissue but also without rapid retrogression.

If we associate all those forms of inflammation of the skin in which the inflammatory irritant has acted from the start upon the deeper layers of the corium of the subcutaneous connective tissue, *i. e.*, upon the layer of the larger nerve-trunks, the larger lymphatics, and the deeper glandular structures, it becomes evident that the appearances even in these cases differ from the varieties of superficial inflammation of the skin in those anatomical peculiarities alone which are furnished by the deeper anatomical situation. A second essential feature is the fact that, in consequence of the deeper point of attack of the irritant, disturbances in the circulation, venous and lymph stases, occur more readily and furnish a direct transition between certain of the forms of inflammation, belonging to this category and true stases-diseases of the skin; finally, for the same reason, viz., the circulatory disturbance, necrobiosis develops readily and rapidly in these deeply spreading inflammations. This is true of superficial phlegmons (burns, pseudo-erysipelas, or diffuse idiopathic phlegmon of the skin), of localized phlegmons (furuncle, anthrax, carbuncle, etc.), and finally of phlegmons directly complicated with stasis (phlebitis and lymphangioitis of the skin, and erysipelas).

THE NON-INFLAMMATORY NUTRITIVE DISTURBANCES OF THE SKIN DUE TO INDEPENDENT STASIS-PROCESSES.

There is a series of morbid processes in the skin, in which venoso-lymphatic stasis and the consequent tissue changes appear from the start in a characteristic manner, although they possess no necessary connection with inflammatory irritants or with an inflammatory congestion which inaugurates the disease.

Direct mechanical obstructions to the circulation are the most frequent causes of

such circulatory disturbances, and occasionally inflammatory processes in the walls of the veins and lymphatics (phlebitis, lymphangioitis). It cannot be denied that these diseases of the vessels are followed occasionally by a form of passive inflammatory congestion (erysipelas) and that, on this account, these acute hyperæmic processes are connected directly with the chronic forms of stasis and anomalies of absorption; for example, elephantiasis arabum is thus connected with erysipelas and acute lymphangioitis. But this connection is an etiological, not an essential one.

We know from experiment and experience that, under favorable circumstances, the venoso-lymphatic stasis may act upon the skin as an inflammatory irritant.

If this occurs, it results in the development of true congestive processes, even of an arterial character, of which erysipelas is an illustration. Whether such an irritant action occurs at the same time or later, the real character of the process does not reside in the inflammation, but in the vascular stasis and the anomalies of exudation (transudation) and the tissue changes (sclerosis of the connective tissue, etc.) immediately connected therewith, or finally in the development of a necrotic process (gangrene, etc.); and this is the reason for including such diseases in one group, viz., stasis-dermatoses presenting the characteristics of a passive disturbance of circulation and impaired venoso-lymphatic absorption.

The passive stases of circulation and absorption which characterize diseases of this kind produce, according to their form, partly incomplete, partly complete obstruction of the circulation. As has been shown above, the effects of the former vary according to the degree of stasis and the greater or less implication of the lymphatic apparatus. They are either:

Mere passive (stasis) hyperæmiæ which do not, however, present the vascular change necesssary to inflammatory processes, and must, therefore, be distinguished from inflammatory hyperæmias.

Or they lead to transudation of blood-serum though the walls of the vessels into the surrounding tissues. Pathology applies to these serous effusions the name œdema and recognizes various modifications of it.

To the class of stasis-dermatoses—apart from mere passive (venous) hyperæmias and local ischæmias and also complete stasis-processes with necrosis of the skin, as, for example, local asphyxia, traumatic decubitus, etc.—belong in the main two forms of disease, both of which begin with venoso-lymphatic stasis, and terminate in part in hypertrophy, in part in atrophy. The first form is represented by elephantiasis arabum, or pachydermia, the second by sclerema (scleroderma, scleroma of the skin). It appears to me proper that in this class should be included "myxœdema," first described by Gull as a chronic general œdema with pallor, dryness, atrophy of the skin and mucous membranes, diminution of temperature, and psychical disturbance. According to Ord and others, the œdematous infiltration presents a marked mucoid structure.

It must be reserved for the special nosology to give the clinical symptoms of the other diseases mentioned above. At this place the reasons for giving the above definitions will be briefly stated:

Elephantiasis arabum is not an inflammation of the skin and does not begin as such, but, in the endemic forms which constitute the most typical varieties, with a symptom of stasis, viz., with acute or chronic œdema of the subcutaneous connective tissue, followed by enlargement of the lymphatic glands, occasionally by inflammation of the larger deep veins and lymphatics, then by erysipelas, and finally thickening of the connective-tissue layer underneath and in the skin. The deep inflammatory symptoms which