occur (erysipelas) are not a primary part of the process, but the result of a nutritive disturbance.

Nor is elephantiasis arabum a primary hypertrophy of the skin, a diffuse fibroma in Virchow's sense.

The hypertrophy of the connective-tissue layer is evidently the result of a disturbance of absorption in the tissues of the integument, as is distinctly demonstrable by the typical occurrence of œdema and the increase in thickness of the connective tissue after every relapse of acute phlebitis, lymphangioitis, and erysipelas.

These remarks hold good also with regard to scleremata of the skin. The first symptom of every scleroderma is lymphatic œdema, circumscribed or diffuse swelling of the tegumentary tissues. This condition corresponds clinically to increased tension of the skin, diminished temperature, loss of sensibility. The immediate cause of this change is entirely unknown, but the œdema, which is always present at the onset, places it beyond a doubt that we have to deal with stasis and an anomaly of absorption. On the other hand, all symptoms of phlebitis and capillary lymphangioitis are absent; there is no erysipelas or accumulation of lymph in the tissues, even in those forms which run an acute course (sclerema neonatorum), so that the difference between sclerema and elephantiasis arabum is at once evident.

As opposed to elephantiasis, which always ends in hypertrophy, the termination of all forms of sclerema in atrophy of the connective tissue must be regarded as typical. The majority of the cases observed have been described by authors merely during this last stage.

But sclerema is not a pure atrophy of the skin. It must be distinguished also from the "general atrophy" of Wilson, a probably congenital progressive atrophy of the skin associated with pigmentation and teleangiectases, which I have called essential lioderma.

In its further course, however, the termination of sclerema in atrophy occupies the foreground and its symptomatology finally becomes very similar to that of pure atrophy of the skin.

HEMORRHAGIC NUTRITIVE DISTURBANCES OF THE SKIN.

In the series of dermopathies last discussed, we found that the point of departure and chief characteristic are the mechanical stases in venous and lymphatic vessels, together with an absence of the inflammatory change in the walls of the vessels. Here, as in inflammatory diseases of the skin, it has often occurred that, in addition to the other phenomena of inflammation or mechanical stasis, an exudation of red blood-globules greater than normal has taken place through the walls of the vessels on account of a simple intensification of the pathological process. This experience offers a transition to a further group of diseases in which the primary inflammatory change in the vascular walls and venoso-lymphatic stases are likewise absent, but the chief feature of which is also the increased passage of red blood-globules through the walls of the vessels.

This group includes in part certain traumatic hemorrhages (ecchymoses) independent of external irritants of the skin, in part those forms (essential hemorrhages) which are connected with general affections or other organic disturbances. They include purpura, morbus maculosus Werlhofii, and scorbutus.

THE DISTURBANCES OF INNERVATION (IDIONEUROSES) OF THE SKIN.

The term idioneuroses of the skin is applied exclusively to those disturbances of function in the distribution of the cutaneous nerves to which no trophic disorders are peculiar, except that such nutritive disturbances may be associated as secondary processes. These affections are therefore readily distinguished from the neuritic dermatoses, as well as from the angioneuroses. The idioneuroses include sensory as well as motor neuroses.

The sensory neuroses are subdivided into two groups.

Occasionally the sensory disturbance consists of an increase, diminution, or alienation of the normal tactile sensation. The diseases belonging to this category, which are associated usually with central nervous affections, are called hyperæsthesia, anæsthesia, and paræsthesia of the skin.

The neuroses of the second group have nothing to do with sensory impressions from the outside, but merely reflect the impression which the condition of our integument or of individual portions of it *per se*, produce upon consciousness. This form of activity of our consciousness is called "general sensibility," and with regard to the skin "cutaneous general sensibility."

Disturbances of cutaneous general sensation may be presented to consciousness under two forms:

First as sensations of pain. Those changes of the tegumentary nerves which simulate in consciousness the impression of a uniform, constant irritation of a nerve-trunk and its region of distribution, are called neuralgias.

Secondly as the sensation of pruritus. This includes sensations which are interpreted in consciousness as if simultaneous, very slight irritations of the extreme terminal ramifications of the nerves had occurred. Pruritus is evidently closely associated with the sensation of tickling. The term pruritus cutaneus is applied to that disease which manifests itself by violent itching without any other disturbance, *i. e.*, in the form of a pure sensory neurosis.

This constitutes a transition to another sensory neurosis which differs from it only in the simultaneous implication of the muscular fibres of the skin, and represents the second form of diseases of cutaneous general sensibility. This form of disease is known as prurigo.

Pruriginous skin presents all the well-known histological changes found in any cutis which is in a condition of chronic irritation.

Derby and Gay found in the hair follicles and root sheaths such appearances alone as are common to other chronic processes of the skin with or without pruritus and formation of nodules, for example, the papillary out-growth of the root-sheaths and the thickening of the smooth muscular fibres, the arrectores pilorum, which occurs prominently in prurigo, but also in other processes connected with the hair follicles, for example, lichen ruber and lichen scrophulosus.

Thickening of the horny layer above the nodules in prurigo has been noted by all observers. The color of the nodule—if it has not been scratched—is like that of the healthy integument. These facts favor the view that the nodule of prurigo is nothing more than a form of lichen pilaris.

Goose-skin is a constant attendant of prurigo nodules and naturally occurs more markedly than usual when the arrectores are hypertrophic. As goose-skin often is produced suddenly by emotional disturbances or changes of temperature, it is regarded as a spasmodic muscular contraction, and the process which lies at its foundation as a motor neurosis of the skin.

The absence of signs of inflammatory fluxion, the occurrence of pruritus, etc., justify the following conclusion:

Prurigo, like pruritus, is a sensory neurosis of the skin. It differs from the latter in the primary development of nodules, and is characterized anatomically by the hypertrophy of the smooth muscular fibres, physiologically by the coincident goose-skin.

The interpretation of prurigo is rendered clearer by a few additional factors:

First, by the constant occurrence of urticaria (which is eminently a vascular spasm of the skin) in the prurigo of children, *i. e.*, in the first outbreaks of the disease.

Secondly, by the fact that the specific nodular eruption of prurigo is not absent in any case—otherwise the diagnosis is not made—but that there is scarcely a case in which the nodular eruption during one or another attack is either very slight or entirely absent, while the pruritus and eczematous symptoms do not suffer the slightest diminution compared with other attacks.

THE DEVELOPMENTAL ANOMALIES OF THE SKIN IN GENERAL.

The nutritive disturbances which we have discussed are often followed by more or less permanent changes in the quantity and quality of the tissue elements, as well as in the form of their restitution. The skin furnishes, in this respect, a distinct example in that secondary change of the upper layers of cells which are destitute of vessels, in the form of chronic desquamation often left over after erythematous and eczematous processes.

We will now make use of the illustration just adduced for a few further considerations. Desquamation of the epidermis may also occur in another manner, as in that disease which develops soon after birth and is known as simple ichthyosis (with diffuse desquamation or in slighter grades branny desquamation). In both cases, in desquamation after inflammations and in ichthyosis, we have to deal with an immoderately rapid and profuse restitution of the horny layer which is cast off; in the first case as the remains of a nutritive disturbance, in the second case as the expression of a developmental anomaly of the tissue elements. The latter disease is a primary one, but it resides inherently in the type of tissue development, *i. e.*, in the laws of development of certain tissue elements.

What has been said of ichthyosis, also holds good of acquired diseases.

In like manner, the distinction between nutritive disturbances and developmental anomalies may also be maintained with regard to those tissues which, on account of their anatomical structure, are the direct primary site of the nutritive disturbances, in our case accordingly the corium. In the case of the epidermis, indeed, the nutrition of which depends entirely upon the vascular underlying structures, the distinction depends upon the structure of the latter. If the corium is inflamed, the change in the epidermis must also be regarded as dependent upon the inflammation and is therefore a process belonging to it. But if the corium is normal and the epidermis elements are alone in an abnormal condition, we have to deal merely with an anomaly of development. This is different in regard to the corium, which, having arisen from the mesoblast, includes all forms of connective substance, and therefore appears polymorphous in its deviations of development.

The anomalies of development of the skin, therefore, may be differentiated in two directions, according as they depend upon the anatomical and physiological laws of development of the epithelial or of the connective-tissue layers of the skin.

Furthermore, as all the accessory and glandular structures, the sebaceous and sudoriparous glands, the hairs and nails are developed from the layer of epidermis, it is evident

that all anomalies of these structures must be included among the developmental anomalies of the first-mentioned epithelial parts.

ANOMALIES OF DEVELOPMENT OF THE SKIN OF AN EPITHELIAL ORIGIN AND TYPE (EPIDERMIDOSES).

Epidermidoses may be divided into three groups:

The first contains those diseases in which the developmental anomaly is, in the main, an anomaly of the cornification process of the upper layers (keratonoses).

The second includes the changes of pigmentation, which, in the normal skin, are also situated in the upper layers (chromatoses).

The third group embraces those diseases of the upper layers the characteristic of which appears to be an abnormal process of growth of the younger, not yet cornified elements of the upper layers, the so-called prickle layer (akanthoses).

Little as we know concerning the nature of the cornification process, there is no doubt with regard to its anomalies that we have to deal with an essentially chemical change (the formation of a peculiar substance, keratin) which old epithelial formations undergo: that the boundary between the prickle cells of the Malpighian layer and the horny layer is formed by a few strata of cells containing granules (Langerhans' layer), which are formed from the prickle cells proper by the loss of their processes and their more markedly lateral juxtaposition, and that this layer must be regarded probably as decisive with regard to the cornification process, as it is followed immediately by the youngest horny layer.

Closely related to cornification is the physiological process known as secretion of sebum and perspiration. As is well known, the glandular follicles are involutions of the epidermis in the corium, and the epidermal portion of the sebaceous glands appears in the form of the epithelium (one or more layers) which lines the duct of these glands. In sudoriparous glands of larger calibre, the process is evidently the same. Whether the perspiration should be regarded as a product of the transformation of the parenchyma of these glands or as a secretion furnished directly from the blood-vessels has not been decided hitherto. But we will not go astray in associating anomalies of the secretion of the sudoriparous glands, like those of the formation of sebum, with anomalies of keratin formation.

In accordance with these remarks we must consider the abnormal cornification process and the anomalies of secretion as physiologico-chemical processes, which are not necessarily caused by or complicated with an affection of the young layers of epidermis.

On the other hand, the opposite state of affairs sometimes occurs occasionally, *i. e.*, a disturbed cornification process sometimes results in secondary morbid phenomena in the prickle layer, and which may often be explained by the increased pressure of the hypertrophic horny layers upon their basis, for example, in the formation of callus.

In addition to the pure cornification anomalies (keratoses), the keratonoses also include anomalies in the development of the hairs (trichoses), the nails (onychoses), the secretion of sebum (steatoses), and the secretion of sweat (idroses), and, according as an exaggeration, a diminution, or a deviation from the type of development predominates, they may be termed hyperkeratosis, keratolysis, or parakeratosis, etc.

It will now be easy to illustrate the general considerations furnished above by a brief resumé of the anatomo-pathological appearances in the principal diseases belonging to this category.

For this purpose we will call attention, among the hyperkeratoses, to ichthyosis.

Ichthyosis is a pure hyperkeratosis, both in the flat as well as the papillary form.

A massive, fatty, often pigmented horny layer, either flat or in cones like the layers of an onion,

is spread above the prickle-cell layer, which is never thickened, but often diminished in size. The interpapillary rete cones and the papillæ of the flat form of ichthyosis are but little elongated, in cases of ichthyosis cornea often very considerably elongated: they do not branch dendritically, nor do the rete cones ever present a branching growth into the corium. In addition, no inflammatory cellular new-formation or thickening of the tissues is present in the corium and papillæ; on the other hand, the vessels of the papillæ are coiled and twisted here and there; in the sweat glands here and there occlusion and formation of cysts or hyaline cylinders, in the hair follicles pearly formation of cavities from the horny layers, out-growths from the root sheaths towards the hair follicles (these occur also in other chronic affections of the skin and even in normal integument), thickening of the arrectores pilorum.

All the appearances may be attributed to pressure exerted by the immoderate horny growth upon the underlying structure. The structure of the papillary body, for example, points clearly to the merely mechanical development of the changes in it, if they are compared with affections in which active processes are present in the young epidermis layers of the skin.

In like manner the twisting of the papillary vessels and the thickening of their walls must be attributed to the mechanical action of pressure.

With regard to the affections of the glandular and hair follicles in ichthyosis, it is evident that the anomaly of cornification has extended into the main involutions, and thus all the appearances connected therewith may be readily explained.

The hyperkeratoses also include lichen pilaris, a cornification anomaly confined exclusively to the excretory ducts of the hair follicles, and which occurs around the latter as a moderate and usually temporary thickening and accumulation of scales; it has nothing to do with the secretion of the sebaceous follicles, or with the hairs themselves, but with the lamellæ of the horny layers forming their circumference.

When this formation of scales around the follicles is congenital, and is at once renewed after each removal of the accumulation, so that the skin constantly looks like a grater, this congenital form of lichen pilaris must be regarded evidently as a moderate grade of ichthyosis around the follicles, and therefore I apply to it the term ichthyosis follicularis.

In addition to callosities and corns, horns should also be included among the localized hyperkeratoses, which develop free in the tissues but not around the follicles.

Among keratolyses the principal part is played by pityriasis—not that which remains after congestive processes, but the pityriasis alba of the hairy scalp and the pityriasis essentialis rubra of Devergie, the reddened base of which is due to the laying bare of the deeper layer of the Malpighian network, and which is evidently the expression of a cachectic process of the integument in general.

The paratypes of the cornification process, finally, are represented by two diseases which I regard as closely associated, viz.: psoriasis and lichen ruber.

The clinical symptoms of psoriasis prove that this is not an inflammatory process in the skin, as we are generally taught by the text-books.

The redness of the newly-appearing psoriasis nodule can be recognized as a hyperæmic redness, it may be pressed away underneath the finger and is lost with the increase in the scaly deposit, so that no trace of a red inflammatory zone can be observed around the constantly developing plaques, as in other inflammatory affections of the skin. The hyperæmic redness of the base gives place to a venous stasis in the vessels, upon which the hillock of scales rests, and this it is which gives rise to the hemorrhages after the removal of the scales.

The anatomical appearances in psoriasis, so far as they are constant, correspond to the clinical symptoms.

The following are the constant lesions:

Increase in thickness of the horny layer, consisting of opaque dry lamellæ, which are being continually desquamated.

The so-called granular layer, the prickle layer proper, and finally the cylindrical cells at the base of the epidermis present more vigorous and rapid developmental changes; increase of nuclei and nucleoli in the deeper layers of the prickle layer, more marked granular development in the upper cells, which are changing into the true granular cells, in addition to more rapid loss of the prickles, and more close juxtaposition of the cells from the cylindrical layer upwards. Finally, a distinct fibrillation of the prolongations of the cylindrical cells which reach downwards towards the cutis in such a manner that apparently a broom-like appearance of the interpapillary cones of the rete develops at the boundary of the papillæ, and create an impression as if several layers of cylindrical basal cells were situated upon the papillæ.

A real increase in thickness of the prickle layer cannot be observed, nor is there any noticeable thickening and elongation of the papillæ of the cutis.

Finally a constant appearance is the distention of the papillary vessels with blood, here and there more marked sinuosity, and in the vicinity of the vessels—but only after the psoriasis plaque has lasted a long time—an increase of the round and spindle-shaped cells in the papillæ, and œdematous separation of the connective tissue.

All these changes may be observed in psoriasis, but they occur more or less in all keratoses.

The main processes occurring in the epidermis in psoriasis, apart from a moderate increase of the growth of the young epidermis cells, is evidently an anomaly of the cornification process, inasmuch as a more rapid, but at the same time more imperfect cornification of the epidermis cells may be recognized, associated with the increased nutritive changes in the prickle layer. This is expressed clinically by the formation of hillocks of scales out of dry opaque, torn and lamellated, slightly adherent masses of the horny layer. At the same time the observation may be readily made that under these scales, which can be easily removed, the young layer of cells of the epidermis manifests a want of cohesion, so that beneath these cells we reach at once the cylindrical layer of the rete which allows the vessels of the papillæ to shine through, and, being itself readily detached, lays bare the bleeding papillary vessels. The cornification anomaly in psoriasis is associated accordingly with a moderate developmental anomaly of the younger layers of the epidermis, and both combined give rise to the plaques of scales which lend to the disease its peculiar stamp.

Related to psoriasis is a second form of disease, lichen exudativus ruber of Hebra with its variety, lichen planus Wilson, which I also include among the paratypical keratoses. The reasons for this are as follows:

From a clinical standpoint there is an evident similarity with nodular formations of various kinds, which must be certainly regarded as keratoses. But while the initial form of lichen ruber always presents that accumulation of epidermis around the mouths of the hair follicles, known otherwise as lichen pilaris, the nodules in psoriasis soon pass into a diffuse formation of disks. But when the lichen has attained a higher grade of intensity, so that the originally separate red nodules become more closely approximated, then the surface of such groups of nodules becomes covered with a layer of adherent whitish scales, which again gives the appearance of psoriatic skin; the palms of the hands and the soles of the feet especially become covered with a thick scaly fissured cal-

losity of the upper layers of skin, the nails become brittle, thickened, destitute of gloss, the hairs become thin, and gradually fall out.

In fact, the diffuse, inveterate form of psoriasis also approaches this clinical picture of lichen ruber, from the fact that a hyperæmic condition of the papillary layer may develop in both.

The clinical differences between psoriasis and lichen ruber appear to me to consist in this, that the former disease starts from the continuity of the skin, the latter from the epidermis layers of the hair follicles. In psoriasis, therefore, the process spreads towards the periphery in segments of a circle, while the centres of the plaques grow pale and heal. In lichen, however, the disease spreads along the root sheaths of the hair, *i. e.*, perpendicularly to the surface of the skin in an upward or downward direction. Connected therewith may be the fact that in the first case a more marked accumulation of epidermis develops in the form of a pointed nodular mass; in the latter case, upon advancing downwards, a central depression of the nodule of epidermis, a sort of umbilication is produced.

It is evident, then, that the primary affection in lichen ruber as in lichen pilaris and the beginning of psoriasis, is not a true inflammatory efflorescence, but merely consists of accumulations of epidermis the development of which has continued, and which, by pressure upon their base, produce hyperæmia and a secondary fluxion, and thus the red, brownish-red color of the nodules, while the continued predominance of the affection of the horny layer gives rise to the final extensive scaly surfaces.

I may omit further details concerning the nature of those anomalies of secretion of the growth of the hair and nails which are closely related to true keratoses, and also of chromatoses, and must restrict myself, so far as regards the character of the diseases of the prickle layer of the epidermis, with illustrating their nosological character by a few examples.

The type of simple proliferation of the prickle layer (hyperakanthosis) is observed in warts and condylomata; atrophy of this layer (akantholysis) in pemphigus; paratypical growth (parakanthosis) by those forms to which I have applied the term akanthomata (cancer of the skin in its various forms).

In all these anomalies, the chief importance must be attached to the relation of the basal cylindrical layer, the germinal-cell layer of the rete, to the corium, and, in a corresponding manner, the changes produced on the level of the papillary formations of the epidermis and corium acquire the most marked development. In warts and condylomata, as in pemphigus and epithelioma, marked desquamation or accumulation of scales never occurs, the development of the horny substance always takes place in an entirely normal manner.

So much with regard to the relation between the germinal layers of the horny layer on the one hand and the prickle layer on the other. The question further arises, what is the relation between the prickle layer of the epidermis and the upper layer of the corium; whether, as has been so often believed, all changes in the former should not be regarded as merely secondary, as a result of the condition of the papillæ of the corium.

The histological appearances have been interpreted usually as meaning that all the superficial morbid processes of the integument not alone take their departure from the papillary layer, but are also associated with an elongation of the individual papillæ themselves. In fact, the objective appearances teach us that an increase in the volume of the papillæ does really occur (though not produced actively) to a slighter extent in the keratoses, to a greater extent in the akanthoses, and it may be disputed whether the

papillæ do not actually grow. But this statement is made also with regard to all other processes, even when they produce very slight manifestations in the epithelium layer; the "elongated"[1] papillæ constitute the inevitable refrain of the histological melody, and estimates of their increase in length are taken without any regard to the length and structure of the papillæ of the normal skin at the same places.

But it is clear that the papillary layer differs physiologically from the remainder of the corium only in the fact that epidermis cones have grown into it. In the papillary layer, an inflammatory process runs exactly the same course as in the deeper part of the corium.

But the compression of the connective tissue by masses of cells, or an accumulation of exudation is a process which does not give rise to enlargement in the volume of the tissue *in toto*, until the limits of the elasticity of the fibrous tissue have been exceeded and their further compression is no longer possible, and when, on the other hand, a migration of this accumulated mass into the vicinity—into the epidermis on the one hand, and the subcutaneous connective tissue on the other—is not possible. But we are well aware that the vitality of the epidermis depends upon the passage of juices from the vascular corium into the former. We see daily in inflammatory processes that the most varied efflorescences containing serum and pus are formed in the tissue of the epidermis by exudation passing to them from below, and thus there can be no question of an increase in volume of the corium or of its papillary layer alone, so long as the tissues lying upon and around it have not lost the capacity for the absorption of fluid.

But granted that this has happened, what will occur next? The fluid will produce œdema of the subcutaneous connective tissue and corium by which an increase in the volume of the tissue, as well as of the papillary layer of the cutis, may be produced. But in this event the increase in the size of the papillæ must occur uniformly in all directions, not alone in their long diameter.

Let us assume the second case, viz.: distention of the tissue of the cutis with masses which are chiefly cellular. Experience teaches that a passage of the cell masses into the rete occurs in such a manner that the boundary between the cutis and epidermis is concealed and finally presents to the observer the appearance of an apparently homogeneous tissue filled with cells or nuclei. But the papillæ never increase in size on account of the cellular infiltration of their tissue, unless some other factor is active.

From these considerations we must infer furthermore that the growth of the papillæ of the corium may be interpreted only as an abnormality in the growth of the connective-tissue framework of the cutis with the vessels embedded in it, etc. But such an anomaly of development would not be confined to the upper layer of the corium, but would probably appear much more markedly in the layers lying underneath, since the larger vascular trunks are situated in that position.

We may now formulate the following statement: an increase in the volume of the individual papillæ of the corium only occurs simultaneously and proportionally with a corresponding increase of volume in the layers of epidermis corresponding to them. This is a constant appearance in the varieties of the class "epidermidoses," in the "chorioblastoses" with secondary proliferation of the epidermis, and in those conditions of irritation which terminate in hypertrophic condition of the papillary layer, for example, in chronic eczema.

[1] The expression "elongation" in ordinary use is improper, as it refers to growth in all directions.

Which of the two forms of tissue of the skin, the corium or the epidermis, plays the active or primary part in this increase of size is a question which has hitherto not been definitely settled.

I will not dwell longer upon the hyperakanthoses, since the most important points with reference to them have been mentioned above. However, a few remarks must be made to substantiate the description of pemphigus as akantholysis. For this purpose, the difference between the inflammatory and akantholytic formation of vesicles must be pointed out.

Experience teaches that vesicular formations in general occur upon the skin in two ways:

First. As a gradually developing change in the epidermis produced by a superficial or deep inflammation of the cutis, which leads to the formation of meshes and chambers from the epithelial cells in a circumscribed district, and to the filling of these chambers by a serous exudation, *i. e.*, the inflammatory vesicle or vesicular phlyctenula, which may be regarded merely as a large vesicle or confluence of a number of vesicles.

Secondly. As a sudden destruction of the younger layer of epidermis (the prickle cells), which is not due to a typical inflammatory process, but is merely accompanied accidentally or secondarily by congestive phenomena; this is effected by fluid which, escaping *en masse* from the vessels of the corium, lifts up the epidermis as a whole over a circumscribed spot, and effects its separation from the surface of the corium.

The cavity which has been produced in this manner and the fluid accumulated in it are bounded above by the compressed granule layer, over which the stratum lucidum and horny layer are adherent, and below by the papillary layer of the corium, still covered more or less with the remains of the cylindrical layer.

The formation of such vesicles is only conceivable if the Malpighian network has lost the capacity of withstanding the mechanical pressure of the blood serum which escapes from the vessels, and accumulates more markedly in various places in the tissue of the corium. I have therefore applied the term akantholytic to this variety of vesicular formation.

In addition, Unna has described in this form of vesicles (*Vierteljahresschrift für Dermat.*, 1878) a degeneration of the prickle cells (called by him fibrinoid) and their transformation into sausage-like, swollen bands and membranous flat masses, which adhere to the lower surface of the covering of the vesicle (G. Simon), and are traversed by degenerated nuclear formations.

The conclusion may be drawn readily that the form of the vesicle, as it was described in contrast with inflammatory vesicles, only is produced when there is a rapid removal of the epidermis layers from their base over circumscribed localities, during which complete destruction of the elements of the prickle layer and their gradual transformation occurs. This destruction may be produced by blistering remedies, by a burn of the second degree with rapid formation of vesicles, etc.

The destruction of the prickle layer and the vesicular formation may also be effected by a pre-existing diminution in the resistance of the prickle layer, and a series of factors favor the view that in pemphigus we have to deal with the latter form of cachectic condition of the epithelium.

These factors are, in brief, the following:

The clinical signs of inflammatory fluxion are either entirely absent or only auxiliary. The slight redness observed here and there around the pemphigus vesicles or upon their base creates merely the impression of a reactive or collateral fluxion. The process always runs its course with desquamation of the roof of the vesicle and formation of young epidermis without spreading deeper into the papillary layer. This is even the case when the skin affection extends over continuous parts of the skin (P. foliaceus Cazenave), and only the prolonged exposure of the surfaces, devoid of epithelium or other injurious influences, may effect here and there deep suppuration, and then the formation of ulcers and cicatrices.

This variety of vesicular formation evidently can be explained alone by the fact that the nutrition of the younger layers of the Malpighian network is impaired, that they offer but a feeble resistance to the fluid escaping through the walls of the vessels and, in unfavorably situated places, these younger layers are pushed away by the fluid and partly destroyed, partly pressed against the horny layer, and finally are thinned and ruptured by the constantly increasing pressure due to the accumulation of fluid. This explanation is supported by the fact that pemphigus is usually met with in cachectic or dyscrasic individuals; that children suffering from congenital syphilis present these vesicular formations most frequently.

Finally, if we examine the relations of pemphigus foliaceus to ordinary pemphigus, it becomes evident that in both cases there is a lesser or greater degree of capacity for resistance on the part of the epidermis, and a mechanical separation of it down to the horny layer. In this manner may be interpreted all statements concerning vesicular formations after long-standing skin diseases of another kind, for example psoriasis, chronic urticaria, chronic eczema, etc.

This does not militate against the fact that such vesicular formations occasionally develop acutely, if the causal factors arise suddenly or spread suddenly to the skin.

Acute vesicular formations, which present either the purely inflammatory type or that of angioneurotic fluxion (the bullous forms of erythema and herpes iris, and the like), do not constitute pemphigus. However, there may be some forms of vesicular formations which distinctly possess the mixed characters of inflammatory and akantholytic vesiculation, *i. e.*, of inflammation of the skin and pemphigus.

Under the term parakanthoses I include those anomalies of the prickle layer which present an atypical (paratypical) growth, in contradistinction to the simple hyperplasiæ.

Two groups of diseases may be included in this category: first, the so-called "molluscum contagiosum," a transformation of the prickle-cells into peculiarly constituted bodies (molluscum bodies), homogeneous and vitreous in the centre, horn-like at the periphery, which according to the most recent investigations have nothing in common with colloid or amyloid degeneration.

We have to deal in the second group with the extension of the atypical condition of the prickle-cells to the connective-tissue layer of the skin, inasmuch as foci of an epithelioidal type develop in the latter, in the form of irregular cell groups, which differ from the interpapillary cone forms, traverse the connective tissue of the corium in all directions, and find new centres of growth in the involutions of the epidermis in the corium. Nevertheless, the standpoint of Thiersch and Waldeyer must be retained, that these atypical epithelial new formations owe their origin to the epidermis, the pre-existing epithelium, or its continuations in the corium.

This form of epithelial new-formation of an atypical character is also distinguished by the formation of nests (alveoli) which may traverse the entire tissue. I have, therefore, applied to this second group of parakanthoses the term alveolar akanthomata.

As an independent and definite morbid appearance, it is peculiar to the various

forms of epithelioma and to carcinoma of the skin. As an auxiliary phenomenon it occurs in various other processes, especially the so-called granulations, which we will discuss later among the chorioblastoses.

The term "papilloma" should be dropped altogether, since neither the warty nor the alveolar forms of akanthomata, individually or combined, can be designated by it. But we need not be too rigorous, and may apply the term papilloma in the future to cauliflower tumors.

In like manner, the term "carcinoma" is superfluous with regard to the skin, both from a histogenetic and histological point of view.

Moreover, the conception carcinoma, in general, is not a definite one, despite the sharp differentiation from sarcoma which Virchow attempted to make.

However, we may employ the term "carcinoma of the skin" when we wish to designate merely a certain group of alveolar akanthomata. These may be divided into two groups, one of which is characterized by marked cornification of the proliferating epithelial cells in the alveolar bodies (the cancer bodies of Waldeyer), in which manner the well-known laminated epithelial nests, horny layer nests, pearly bodies, cholesteatoma globes, etc., are produced. These should be called the true epithelioma of the skin (epithelioma keratodes of Waldeyer), while the other group in which this cornification is very slightly or not at all apparent, retains the name "carcinoma of the skin."

THE DEVELOPMENTAL ANOMALIES OF THE SKIN OF CONNECTIVE-TISSUE ORIGIN AND TYPE (CHORIOBLASTOSES.)

The developmental anomalies of the corium are characterized by the fact that the growth of the connective tissue in the diseases of this class does not reach the development of really higher types, but remains in an embryonal (cellular) stage, *i. e.*, presents a profuse proliferation of cellular connective-tissue elements, which persist as such (granuloma of Virchow).

These forms of developmental anomaly have a great similarity with a process which develops in the corium after inflammatory irritants, and also depends upon an abundant development of young connective-tissue cells, and is usually termed inflammatory granulation. This process, which terminates every inflammatory process in the connective-tissue layer, and constitutes the first stage in the restoration of wounds and ulcers, also presents some similarity clinically with granulomata. In the granulation of wounds we have to deal also with the formation of nodules from the connective-tissue elements, viz., the granulations of proud flesh, but this is a formation of atypical character with a regular outgrowth of the embryonal elements into bands of connective tissue, vessels, and nerves, and finally the formation of an epithelial covering over the corium which, at the most, has become more tense and devoid of papillæ. On the other hand, the diseases to which we refer now show similar processes, but in an atypical manner, inasmuch as the continuance of the embryonal character of the young new-formation becomes the prominent characteristic.

The diagnosis is evident if the other symptoms of the inflammatory process are sharply outlined, and, on the other hand, resolution of the inflammation occurs. But it has been mentioned heretofore that occasionally the inflammatory irritant is not recognizable distinctly, that furthermore the clinical signs of inflammation appear less sharply defined, that finally its course occasionally is a tedious one, inasmuch as the accumulated embryonal cells persist for a longer period in the tissues, and are only made to disappear by necrobiosis; in such cases the differentiation is indeed more difficult from the true granulations, as developmental anomalies of the connective tissue.

But another question arises: as is well known, the presence of bacilli in some of the

granulation diseases (as in leprosy) has been demonstrated with tolerable certainty; in others, as in syphilis, the infectious nature of the process and the probability of the circulation of a toxic substance in the economy cannot be denied. Under such circumstances, would it not be more logical to regard these features as the essence of the process, instead of the granulation due to the infection? Indeed the question might be raised further whether syphilis and leprosy should not be included rather among the acute infectious processes, such as scarlatina, measles, variola.

This may be answered as follows: Whether the infection is regarded as the essential feature of these processes or not, this much is certain that the injurious influence of the cause of disease must be sought in the fact that the development of the tissue elements occurs in an atypical manner. This it is which is known as syphilitic new-formation, etc. Irritative processes in Virchow's sense also occur, *i. e.*, those which bear the stamp of nutritive disturbances due to inflammatory irritants, but they occur only temporarily or constitute merely an introduction to the formative changes which develop gradually.

An anomaly of development is therefore the essential change which results from syphilitic and leprous infection.

I believe accordingly that we may retain these diseases in the class of developmental anomalies.

I now return to the general nosology of these granulomata, the first group of chorioblastoses, and will consider first those forms of disease included therein.

These are:

Lupus essentialis (idiopathicus) tuberculosus and erythematosus, scrophuloderma, tuberculosis of the skin, leprosy, syphiloderma, rhinoscleroma, granuloma fungoides; if the latter, as French writers now claim, should not prove to be a lymphadenoma.

The following features are common to all these granulomata:

1. They consist of small cellular elements, in an embryonal stage of development.

2. The point of departure and chief site of the granulation new-formation are exclusively the tissue of the corium.

3. The small-celled granulomata appear in more or less sharply defined spots which may be recognized externally by the formation of nodules. These nodules have a dark brownish-red color, firm consistence, often coalesce into flat elevations.

4. They undergo retrogression, inasmuch as they either break up, become cheesy, suppurate, ulcerate, or atrophy without having undergone a loss of substance, and then leave cicatricial depressions.

5. These processes are generally chronic. Many granulomata are distinguished by the high degree of resistance of their cellular elements to necrobiosis. These qualities are not possessed by all granulomata at every period; but they may be possessed or acquired by all, whatever the source of the infiltration has been.

The description of the histological appearances must be reserved for the special part of this work, and I will confine myself here to the following points.

Upon a section of lupoid infiltrated parts, there are found in the tissue of the cutis—embedded between the bands of connective tissue, the vessels and glands—accumulations of cells (granulations), partly in the form of isolated islets, partly scattered over larger surfaces, chiefly around the vessels and lymphatics. Some of these masses frequently present the giant-cell arrangement, *i. e.*, one or more layers of wandering cells which inclose epithelioid-cells in a reticulum which probably starts from the vessels, and in which are situated one or more giant-cells.

Various views obtain concerning the interpretation of the above appearances, *i. e.*, concerning the origin of the cellular elements in general and the giant-cells in particular.