ANOMALIES

OF THE

SUDORIPAROUS GLANDS AND THEIR FUNCTION.

BY

PROF. E. GEBER, M.D.,

KLAUSENBURG.

THE sweat glands have an independent ample vascular system surrounding them, and specific nerve-fibres influencing their activity. Each epithelial cell of the convolutions represents an elementary secreting organ. On account of their difficult accessibility, our knowledge of the pathological processes of the sweat glands is in many respects defective. Still there is no doubt that they are exposed to hyperæmia, inflammation with its various terminations, hyperplasia and hypoplasia, hypertrophy and atrophy, the manifold neoplastic processes, to the same extent as the larger glandular formations. We shall first deal with the diseases of the sweat glands in the most restricted sense, and then with their abnormal activity.

I. INFLAMMATION OF THE SWEAT GLANDS.

Inflammation of the sweat glands is one of their most frequent diseases. As in other glands, it occurs in an acute, subacute, or chronic form, and, when the inflammatory process has not advanced too far, it may resolve, or else change into suppuration, abscess formation, hypertrophy, atrophy, fatty, hyaline, and other degeneration.

Etiology.—Inflammation of the sweat glands may appear primarily, idiopathically, or secondarily by extension from the neighborhood, or as a sequel of other general diseases. The former is possible when the pore is occluded, or when mechanical irritations, as by eczema, prurigo, scabies, pediculosis, etc., continually affect the skin. The latter may be the case when the last-described and other similar factors produce inflammation of the skin, and the inflammation extends by contiguity from the surrounding connective tissue to the gland (*periadenitis sudoripara*); when the affection arises after grave

febrile diseases—typhus, polyarthritis acuta, cholera, pneumonia—and in the train of marasmic conditions—cachexia, scrofula.

Anatomy.—In the simplest form the cells are traversed by a fine dust-like protoplasm, and appear slightly distended. The canal becomes narrow in proportion, and the gland, as a whole, seems enlarged. When the process has gone further, the cells lose their normal outline, the contents become opaque, the nuclei divide, and the nuclear corpuscles are increased. In the adjoining connective tissue a large number of leucocytes are noticed, chiefly around the vessels. Later, the greatly altered cells enter the canal of the gland, where they disintegrate and are pushed forward and displaced by the continually following pus-corpuscles. The final result of an exudative inflammation is either complete disappearance of all trace of a sweat gland (abscess of sudoriparous gland), or else we find in the loosened, wide-meshed connective tissue corresponding to the region of the pars reticularis cutis, partial outlines, whose centrally directed lumen and possibly still remaining epithelioid cells permit their interpretation as the remnants of glands.

Symptoms, Course, and Termination.—We shall take for our starting-point one of the relatively most frequent localizations which presents a more pronounced clinical picture. I refer to those inflammations of the sweat glands which are not unfrequently met with at a distance of one and a half to three centimetres from the anus.

The first symptoms are usually insignificant. Some occasionally recurring itching is present to which but little importance is attached. By-and-by, however, it becomes more frequent and changes to a burning, and the skin feels rather warm. As the inflammation increases, more violent pains and heaviness are experienced, and on careful examination a nodule may be demonstrated in the depth. In this stage, it is possible to cause dissipation of the tumor and resolution of all the inflammatory symptoms, by the avoidance of every movement and the employment of ice applications. If this improvement is not soon brought about, the skin will become much reddened, infiltrated, painful, and hot; in the centre of the patch may be felt a perpendicular hard cord, starting from a broader base—the enlarged nodule—toward the surface where it terminates either in a roundish or flattened elevation. At this time suppuration is usually present, though it is not easily demonstrable because so deeply seated; when we, however, insert a bistoury as deep as possible into the tissue, there is evacuated some thin fluid, occasionally more viscid pus mixed with crumbly particles. If the evacuation has been early enough and the patient takes care of himself, a few days' treatment often suffices to arrest the suppuration. In the opposite event, the suppuration extends far, and deep suppurating wounds may arise with sinuous tracks and perforation of the rectum (fistula ani). In marasmic individuals the course is particularly slow. The melting down of the surrounding tissue then appears in the form of cold abscess, and fistulous tracks are relatively frequent. Sooner or later, cicatrization takes place, the characteristic of which is that the surface is drawn in. The deep infiltration usually persists the longest, and with rare exceptions gives rise to frequent relapses.

Inflammations and abscesses of the sweat glands are also met with more frequently on the labia majora, in the axilla, and on the scrotum.

Differential Diagnosis.—It will not always be easy to distinguish inflammation and abscess of the sudoriparous glands from circumscribed phlegmons, furuncle, syphilitic gumma, etc. But the former affection begins as a deep movable nodule; is never associated with fever; is especially painful on pressure; permits the recognition of a firm cord extending upward; on the surface, according to the state of the overlying cutis, it

appears sometimes as a roundish projection when the cutis is loosely joined to the subcutaneous tissue, sometimes flattened when those structures are firmly connected; it commences to suppurate at the base; the suppuration often ceases remarkably soon after being punctured; it leaves a retracted cicatrix and a deeply imbedded infiltration which, by reason of its slow absorption, often gives rise to repeated relapses.

Treatment.—The treatment depends on general surgical principles. The main thing is to remove every injurious influence, and to provide absolute rest of the part in question. As long as the inflammation is not yet circumscribed, we may restrict ourselves to cold or ice applications. When the nodule becomes distinctly perceptible and the pains are continuous and serious, puncture should not be delayed, even if fluctuation cannot be demonstrated; when an abscess has formed, nothing remains but to expose the wound by a free incision, and to cause it to heal by appropriate dressings and attention. Even then relapses will be relatively frequent.

II. HYPERTROPHY AND ATROPHY OF THE SWEAT GLANDS.

A. *Hypertrophy.*—Enlargement of the sweat glands is sometimes congenital, in the case of general excessive development of the body, or gigantic growth. More usually, however, it is acquired and forms either autochthonously or is produced secondarily.

Etiology.—The simplest form of the former mode of development is represented by those idiopathic hyperplasias occurring in connection with those of the epithelium and the papillary body of the cutis. Under the same head belong also the soft warts (acrothymion) mentioned by Bärensprung, in the substance of which, as well as beneath, we may often find heaps of sweat glands enlarged up to one millimetre. The same condition may frequently be demonstrated in ichthyosis. These occurrences, however, are merely of an anatomical interest; while the hypertrophies, whose elements extend far beyond the normal measure, are in every way of greater moment and deserving of a more detailed consideration. Without any demonstrable cause there occurs, in the cases belonging under this head, a proliferation of the glandular epithelium which shows no other change than possibly enlargement, so that the wall at first is made to bulge out and later to project in the shape of a finger-like prominence. If we imagine the process continuing, every single spur again throwing off others, etc., we obtain a number of smaller and larger tubuli, like a dendritic anastomosis. As long as the gland is capable of performing its secretory activity, we can only look upon it as simply hypertrophic. But whenever it begins an independent existence no longer adapted to its previous function, it acquires the importance either of an adenoma when its outgrowths retain the glandular character, or that of a destructive neoplasm when epithelium and stroma proliferate in favor of the former.

Besides the genuine hypertrophy of the sweat glands, we must enumerate the following causes for their development: 1. Increased activity. In a number of grave diseases, it is certain that the profuse perspiration is attended by enlargement of the glands, as in cachexia, scrofula, articular rheumatism, phthisis, etc. 2. Mechanical, chemical, thermic, and electrical irritations. All of these factors, if continued for some time, cause a greater supply of nutrition either exclusively in the glands or in the cutis with its adnexa, and thereby increased formation of elements. 3. Inflammatory processes. Acute inflammations of the cutis, when the sweat glands participate, provoke only a temporary increase of the cells. On the other hand, after chronic inflammations they are almost always altered, most frequently enlarged. In skin thickened by elephantiasis, the excretory duct

of the gland is lengthened, the secretory or convoluted duct enlarged, the lumen dilated and the wall broadened in consequence of augmentation of the epithelial layers and increase of volume of the elements. Similar results appear wherever like conditions prevail, as in prurigo, eczema chron., sclerema adultorum, etc. 4. Proliferative processes of pronounced cellular character, *e. g.*, lupus vulgaris, lepra tuberosa, carcinoma, sarcoma, etc. The process here is the following: Either the specific proliferation of the neoplasm excites the elements of the sweat gland to homœoplastic production, or else it penetrates the membrana propria, infects the glandular contents, and its peculiar cells becoming ever more numerous in the gland, a heteroplastic proliferation is finally established. In the former case there result, in the beginning, purely hypertrophic sweat glands with extensive formation of spurs. As a rule, however, this does not continue. Gradually the lupus and other cells continue to advance and cause the glandular cells to perish, so that lupus, for instance, spreads at the expense of the sweat glands and fully replaces the latter unless the specific proliferation is arrested before. If infection of the glanular epithelia has taken place early, hypertrophic parts of the sweat glands may still occur, but they soon succumb to their fate. Accordingly we finally find long-drawn cones and anastomoses formed of heteroplastic elements, which could only lead us to suspect their origin unless perhaps remnants of the gland substance have remained on the terminal portions.

Symptoms, Course, and Termination.—If the activity of the sweat glands were to keep equal pace with its enlargement, and the quantity of the product secreted were exclusively dependent upon it, hypertrophy and hyperhidrosis would be equivalent. However, this is by no means the case, because the secretion is directly subordinated to nervous influence. Thus hypertrophic sweat glands may exist for a long time without becoming clinically at all perceptible. Enlargement of the gland will coincide with increased secretion only when both are based on the same etiological factor (phthisis), or when the conditions are given for both independently of each other. In the contrary case, enlarged sweat glands may even be associated with diminished perspiration. This is true also when isolated parts of glands are hypertrophic while the gland is affected with infiltration or heteroplasia.

The course and termination of hypertrophy of the sweat glands are manifold. It may persist for years without essential alteration; but it may also change into unlimited growth and, in that case, either maintain its typical character (adenoma), or undergo a thorough transformation (carcinoma). It may happen with equal frequency that, in consequence of the exhaustion, atrophy occurs; or by occlusion of the duct or opening, cyst-formation; or, by regressive metamorphosis of the glandular elements, fatty, hyaline, colloid, and other degeneration.

B. *Atrophy of the sweat glands.*—Diminution of the sweat glands is usually effected by decrease of the cellular elements; more rarely by diminution of volume of these elements.

The causes of the atrophy may either date from fœtal life, a defective development of the body and the cutis being associated with insufficient formation of these glands; or they may be post-natal, acquired. The latter again may be attributable to their physiological course of development, *e. g.*, senile degeneration, or be brought about by partly general, partly local pathological conditions. In persons affected with diseases of the digestive organs, and especially in those suffering from defective nutrition or marasmus, sweat glands in process of involution will be found almost without exception. Should the nutrition be insufficient only on some parts of the body, the glandular atrophy will

affect corresponding larger or smaller regions. Wheals, corns, cicatrices, etc., which diminish the blood supply by pressure on the underlying tissue and favor absorption, always cause atrophy of the sweat glands. Among etiological factors we must also enumerate the diminished activity of the nerves. For instance, on paralyzed parts of the body we find atrophic sudoriparous glands attributable not only to the cessation of active mobility, but also to the lesion of the specific nerves. This will also be the case with other nervous affections when these fibres have thereby been placed out of function. Finally atrophy occurs after inflammatory processes, degenerations, and metamorphoses of the glandular substance. It is mainly after the various inflammatory processes of the skin that hypertrophy is not rarely seen side by side with atrophy of the sweat glands.

Atrophic sweat glands, unless connected with anidrosis, are altogether of no moment.

III. FUNCTIONAL DISTURBANCES OF THE SWEAT GLANDS.

The secretion produced by the activity of the sweat glands may deviate from the normal in two ways. It may be changed in amount (quantitatively) or in composition (qualitatively), and possibly in both ways. The former manifests itself in three degrees: by the quantity exceeding the physiological measure (hyperhidrosis, ephidrosis), falling short of it (hypohidrosis), or being completely absent (anidrosis). Qualitatively the secretion is altered by peculiar relations of the normal constituents (albumen, sugar, bile pigment, etc.), various chemical influences (decomposition products: ammonium carbonate, etc.), whereby its color, odor, taste, chemical reaction, etc., are changed.

A. Hyperhidrosis—Ephidrosis.

By the term hyperhidrosis we mean habitually increased sudoral secretion, profuse sweating, extending over the whole body. If it affects only a single part it is called hyperhidrosis localis or, shortly, ephidrosis. To constitute hyperhidrosis, therefore, it is necessary that within a certain period of time, under all circumstances, more than the normal quantity of perspiration is produced. Nor are we justified in including under hyperhidrosis the profuse perspiration occurring in intermittent fever with the defervescence. The same may be said of the profuse, so-called critical perspiration in remittent fever, typhoid fever, pneumonia, etc.

Etiology.—Hyperhidrosis may be congenital, manifesting itself sometimes even in very young children. Er. Wilson mentions a family in which the husband and suffered from hyperhidrosis from the ninth to the fiftieth year, and the mother and two brothers were affected, while the two sisters were free from it.

In by far the greatest number of cases hyperhidrosis is acquired and may appear at any age. Although it is beyond question that the nervous influence is the determining factor in hyperhidrosis as in the normal secretion, we shall nevertheless not always be able to ascertain the cause inasmuch as it cannot be determined whether only one kind of fibres effects the secretion or whether, as Vulpian assumes, there are inhibitory besides the excito-sudoral nerves in the sympathetic. Moreover, there is still some difference of opinion as to whether the sudoral nerves are merely of sympathetic or also of spinal origin, whether they run isolated or only in company of sympathetic and not also with motor and mixed fibres, etc.

But far from being able to point to the connection with the nervous irritation as the immediate cause for all, *i. e.*, regional, unilateral, and general, hyperhidroses, there

is barely a single pathological process which always produces the same phenomena under equal conditions. In this respect I call to mind lesions of the cervical part of the cord which are followed not only by hyperhidrosis changing with the locality, but often by normal secretion or even anidrosis. Therefore, instead of grouping the causes of hyperhidrosis under special points of view and separating them from the ephidroses, we shall have to remain content with naming all diseases mainly associated with increased secretion of sweat, and only where the nervous influence is indubitable, note it particularly.

Lesions of the cerebrum, on the whole, produce disturbances of the sudoral secretion but rarely; and if so, usually a partial hyperhidrosis. The latter becomes universal occasionally in the course of morbus Basedowii. After an injury penetrating the temporal bone, Bloch observed hemilateral hyperhidrosis and, in another case of epileptiform convulsions, profuse perspirations. Bouveret had a similar experience with a gumma of the cortex cerebri. Adamkievicz reports two cases of ataxia of one arm occurring after a cortical lesion in which profuse perspiration appeared at intervals, and in which the post-mortem showed an abscess of the cerebrum on the side opposite to the affected arm.

Conditions of cerebral depression often cause more or less developed hyperhidroses. All these phenomena point to the fact that a sudoral nervous apparatus must be present in the brain.

Adamkievicz has observed profuse perspirations occurring with a glioma of the medulla oblongata.

Spinal affections rarely present the picture of hyperhidrosis; now and then in tabes dorsalis, also in sclerosis of the posterior horns and of secondary ascending degeneration of the cord if they are associated with increased reflex irritability and eccentric pains. Profuse perspiration is often observed with neuroses of certain nerves; for instance, in trigeminal neuralgia, where it may be present on both sides (rarely), or along one or more branches; also in occipital, intercostal, brachial, lumbar, and sciatic neuralgias.

Injuries, spontaneous or secondary inflammatory processes, morbid alterations and compression of the trunk of the sympathetic or its ganglia give rise most frequently to hyperhidrosis, chiefly unilateral. As a rule, cutaneous hyperæmia, heightened local temperature, turgescence of the skin, and, with lesions of the cervical medulla, oculo-pupillary disturbance are present.

The first acceptable explanations of these processes were furnished by Vulpian and Nitzelnadel. These were amplified since Nicati has shown that paralysis of the cervical portion of the sympathetic produces in the first stage hyperæmia, heightened temperature, and hyperhidrosis of the face, that this is followed by an intermediate stage with cessation of the perspiration, to be succeeded in the second stage by pallor of the skin, lowering of the temperature, and anidrosis.

In hemicrania, according to Du Bois-Reymond, implication of the sympathetic nerve begins with the irritated condition (vascular spasm) on one side of the head, and is followed by paresis—erythema and perspiration—of the affected half of the face. In many cases, however (Möllendorf), the symptoms are said to occur in the reverse order, still the participation of the sympathetic is unquestionable.

But ephidrosis or hyperhidrosis appears with particular distinctness in cases of compression of the sympathetic. Glandular tumors on the neck, aortic aneurisms, neoplasms, carcinomatous degenerations (Ogle), parotid tumors (Verneuil), sometimes even

the infiltration due to inflammation of the cutis (erysipelas) cause, besides hyperhidrosis, the concomitant phenomena (oculo-pupillary) of paralysis of the sympathetic.

Under this head belong all the traumatic influences affecting the sympathetic in consequence of diseases of the tissues or of injuries acting from without. Thus, for instance, unilateral hyperhidrosis has been repeatedly met with after caries of the vertebræ, also after gunshot fractures, compound fractures of the clavicle (together with paralysis of the brachial plexus). Hayem reports a case affecting the lower extremity, where, three years after a gunshot fracture, abnormal pigment and hair formation and continual perspiration were present on the leg. Particularly instructive is Ebstein-G. Fränkel's case in which ephidrosis of the left half of the head and trunk and upper extremity occurred after angina pectoris, without change in the pupil. During the autopsy there were found on the ganglia of the cervical sympathetic (especially the inferior) macroscopically visible, roundish, brownish-black spots the size of a grain of sand which on microscopic examination could be recognized as cavities (varicose dilatations) situated in the continuity of the vessels. Their interior was invested with a distinct endothelium and filled with blood-corpuscles in various states of preservation. A similar case of left hyperhidrosis is reported by P. Guttmann. It is correct to include under this same head the glossy skin (Paget) of traumatic origin, in which hyperhidrosis of the affected part is likewise present in the beginning, and is followed by anidrosis.

The observations of pulmonary and cardiac diseases associated with hyperhidrosis remain obscure. Gubler had long ago noticed the perspiration and the occurrence of deeply flushed cheeks appearing with severe pneumonia. Since then the combination of valvular lesions, cardiac hypertrophy, and often even simple palpitation of the heart with unilateral perspiration has been pointed out by Fleischmann, Seeligmüller, and other observers. From the latter phenomenon, as well as from the frequent presence of the other symptoms peculiar to paralysis of the sympathetic, it may be argued with considerable probability that here the same lesion may exist. The excessive perspiration in phthisis certainly belongs here, only it is specially increased by the general debility, slight nervous excitement, and the depressed state of mind. The fatty degeneration of the glandular epithelium first discovered by Virchow is to be considered as a sequel.

In hysteria and menstrual anomalies, hyperhidrosis or ephidrosis is rather frequently present. It is hardly ever absent at the menopause. I think that here, too, paralysis of fibres of the sympathetic may be assumed.

In connection with the preceding, we shall briefly refer to Aubert's results in some cutaneous diseases. A. has attempted to represent the sudoral secretion plastically in an ingenious manner,[1] and has obtained the following results. In ichthyosis increased perspiration is said to be present when the disease has a superficial seat, otherwise there will be hyphidrosis. Purpura shows on the hemorrhagic spots suppression of the secretion, while in their circumference it is increased. The same is true of cicatricial spots when the sweat glands have perished. In nævus spilus hypersecretion is found because, according to A., hypertrophy of the tissues is associated with that of the sweat glands. On the hyperæmic skin around deep-seated abscesses, wound surfaces, etc., hypersecretion is said to be present, while on parts affected with erythema balsamicum, roseola syphilitica, etc., the secretion is normal. In prurigo, psoriasis vulgaris, eczema, herpes, zoster, pemphigus, the secretion is reduced, in erysipelas completely arrested; it returns on recovery, though only after two weeks in erysipelas.

[1] To this end it is suggested to fasten a piece of thin white paper lightly to the part whose secretion is to be ascertained, for about one or two minutes, and after the drops of sweat—for instance, produced by pilocarpine—show themselves on the paper, the latter is carefully lifted off, drawn through a 0.5 per cent solution of silver nitrate, and then exposed to the light. By the negative impressions obtained in this manner, which are compared with results gained from other healthy parts of the skin, it is possible to get information as to the quality of the secretion.

Symptoms, Course, and Termination.—Perspiration is not an indifferent secretion despite the small quantity of solid constituents (0.5 to 2.5 per cent). When occurring moderately and temporarily, it gives rise to an agreeable sensation by cooling and lubricating the skin.

It is different when the perspiration appears in excess and with but slight intermission; when it annoys anæmic and cachectic persons after slight effort, and if, as in phthisis, it continually bathes the skin. In such cases, not only the causative factor of the perspiration, but also the relation of the sweat to the cutis is of importance.

To be sure, we do not know the relation between the consumption of material and the production of perspiration. But even admitting that the latter is really unimportant on account of its few solid constituents, still the profuse perspiration cannot be unimportant by reason of the great loss of water and the nervous excitement, even if but partial, connected with it. Hence we find, too, that such patients, already debilitated by the primary affection, are weakened still more by the abundant perspiration.

But the hypersecretion manifests itself also by local effects. In such an event the skin, at the uncovered places, is kept clean with difficulty, always feels unpleasantly cool, and, in more delicate persons, the epidermis is macerated. This is true in a still greater degree when the evaporation is hindered by the covering of the part and the various organic and inorganic materials remain there. The latter not only attack the epidermis, but provoke inflammatory symptoms associated with itching, chiefly eczema.

The most striking example of the consequences of general hyperhidrosis is offered by

(*a*) sudamina.

In summer this eruption attacks chiefly fleshy persons who are dressed rather warmly or who are very hirsute and are forced to work in the heat of the day; in the winter it attacks workmen, especially artisans whose shop is very hot, particularly at night. The eruption begins with violent itching, soon extends over the greater part of the trunk, consists of closely aggregated nodules, vesicles, or pustules the size of a pin's head, which appear in fresh crops for several days, and finally the several efflorescences dry into small crusts. This inflammatory cutaneous disease may change into a fully developed moist eczema if the injurious factors persist or if unsuitable irritating appliances are made. But if care be taken that the body do not perspire much, if every other injurious influence be guarded against and the skin kept dry, the efflorescences retrogress, and, the crusts and detached epidermis being cast off, the process is completed within a week.

(*b*) *Miliaria.*—This affection has been regarded as a special form of disease which frequently arises in the train of other, generally febrile, maladies.

As in the acute infectious diseases, writers distinguish three stages of the process. The prodromal stage is characterized by malaise, loss of appetite, depression of mind, suddenly followed by chill or rigor, succeeded by heat. At the same time the patient suffers from severe headache, often lapses into unconsciousness, has syncope, epistaxis, a feeling of oppression on the chest, heaviness of the stomach, nausea, pain in the back and loins, numbness of the finger tips, cramps in the calves, and constant jactitation. This condi-